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1. A Phase III Clinical Trial of Analatro® [Antivenin Latrodectus (Black Widow) Equine Immune F(ab')2] in Patients with Systemic Latrodectism

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Background: There are 5 species of widow spiders (Latrodectus) in North America, with 1692 bites reported to US Poison Centers in 2014. Therapy for latrodectism in the US may include opioid analgesics, benzodiazepines, muscle relaxants or a currently approved equine based antivenom (AV), which carries a perceived increased risk of hypersensitivity due to its whole IgG preparation. The objective of this study is to determine the safety and efficacy of a highly purified equine based F(ab')2 AV (Analatro®) for the treatment of pain associated with latrodectism.
Methods: A Phase III, randomized, double blind, placebo controlled trial was conducted at 16 sites across the US. Patients were randomized to 1 of 2 treatment groups: F(ab')2 AV or saline placebo. Patients with moderate to severe pain intensity measured using the visual analog scale (VAS) were enrolled and treated with up to 2 doses of study drug. Pain intensity was assessed at baseline and every 30 minutes thereafter, for up to 150 minutes. Patients with moderate to severe pain or those who failed to achieve a clinically significant reduction in pain after dose 1 received a 2nd dose. The primary outcome measure was treatment failure, which was defined as failure to achieve and maintain clinically significant reduction in pain for 48 hours post-treatment. The proportion of treatment failures in each group was compared using a 1-sided Chi-squared test. Pain intensity differences (PID) over time were assessed using change in VAS scores relative to baseline at each post baseline time point using a mixed effects model. The summed PID (SPID) was computed for each subject using the area under the curve trapezoidal method. Adverse events (AEs) were recorded through day 17.
Results: 60 patients were treated (29 AV:31 placebo). The mean age was 39 years and 66% were male. There were 15 treatment failures in the AV group and 24 in the placebo group ($p = 0.0185$). The mean difference in PID observed between groups was significant at 60, 90, 120, and 150 minutes post baseline (Table). The mean SPID was significantly greater for the AV group ($p = 0.0135$). The frequency and types of AEs reported were similar between groups. No deaths or serious drug related AEs were reported.

Time (mins)	Mean Difference in PID (AV- Placebo)	p-value
30	10.9	0.1244
60	14.6	0.0401
90	16.6	0.0195
120	20.2	0.0047
150	21.2	0.0029

Conclusions: This equine based F(ab')2 AV was effective at reducing moderate to severe pain caused by latrodectism. No serious safety concerns were identified in this study.

KEYWORDS Black widow; antivenom; latrodectism

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2. A randomised controlled trial of low dose antivenom and fresh frozen plasma versus high dose antivenom for coagulopathy in Russell's viper envenoming

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Objectives: Russell's viper (Daboia russelii) envenoming is a major health issue in South Asia. Russell's viper envenoming causes venom induced consumption coagulopathy (VICC), which takes 24-48h to resolve. We investigated the effect of fresh frozen plasma (FFP) and two different antivenom doses in correcting the coagulopathy from Russell's viper envenoming, post-antivenom.
Methods: We undertook an open-label randomised controlled trial at two Sri Lankan hospitals in patients with VICC, comparing 20 vials of polyvalent antivenom (high dose) to 10 vials of antivenom (low dose) with 4U FFP. Patients (>15yr) were recruited if they had a Russell's viper bite and positive whole blood clotting test. Patients were allocated in a 1:1 randomisation. The primary outcome was the proportion with an INR <2.0, six hours post-antivenom. Secondary outcomes included anaphylaxis, adverse effects, major haemorrhage and death. Analysis was intention to

treat followed by a non-randomised analysis of actual treatment received. In 82 patients (44 with antivenom alone; 38 with antivenom + FFP) with analytically confirmed Russell's viper bites, serial citrated samples were collected. We measured prothrombin time/INR, fibrinogen, activated partial thromboplastin time (aPTT), D-Dimer, factor V, VIII and X and recovery of clotting function was compared using survival analysis.

Results: From 214 eligible patients, 141 consented and were randomised; 71 to antivenom alone, 70 to antivenom and FFP. Eight patients did not have 6h bloods for the primary outcome. Six hours post-antivenom 22/67 (33%) of patients in the antivenom only group had an INR <2 compared to 28/66 (44%) in the antivenom plus FFP group [absolute difference 11%; 95% CI: −5% to 28%; p = 0.21]. In a non-randomised analysis of treatment actually received 23/74 (31%) given antivenom alone had an INR <2 compared to 28/59 (47%) receiving antivenom and FFP [absolute difference 11%; 95% CI: −0.3% to 33%; p = 0.07]; 10 patients allocated no FFP were given FFP, 16 patients allocated FFP didn't get FFP. There was no significant difference in the development of severe anaphylaxis post-antivenom [15/64 (19%) vs. 15/47 (24%); p = 0.54]. One patient given FFP developed clinical features consistent with transfusion related acute lung injury. There was no difference in the time from bite to FFP between patients responding and not responding to FFP [(6.8h; interquartile range [IQR]: 5.1-8.6h) vs (5.8h, IQR: 3.7-7.8h); p = 0.18]. There were no deaths or major haemorrhages. In 82 patients with clotting tests, the median times to recovery of all clotting times and factors were similar between groups, with no difference in the time to recovery of INR or fibrinogen. There appears to be a more rapid recovery of factor V and X in the FFP group (within 24h), which was significant for factor V (Gehan-Breslow-Wilcoxon test; p = 0.04).

Conclusions: FFP post-antivenom in VICC due to Russell's viper bite did not appear to hasten recovery of coagulopathy, but did not worsen VICC, suggesting that the lower dose of antivenom is sufficient. A major adverse effect occurred in one patient administered FFP. Based on the favourable per treatment trend and success of FFP in VICC in other snakes, further work may determine if there is a subgroup of patients with Russell viper bites that respond to FFP.

KEYWORDS Snake; coagulopathy; envenomation

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3. Rattlesnake envenomations treated with and without maintenance antivenom

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Background: Manufacturer instructions for Crofab®, the only commercially available rattlesnake antivenom in the US, recommend maintenance dosing (2 vials every 6 hours × 3 doses) after establishing initial control of the envenomation. Concern over the high cost of antivenom without clear benefit of maintenance dosing has led some toxicologists to dose antivenom on an as-needed basis. Comparative outcomes of these two practices, including hematologic sequelae, antivenom utilization, hospital length of stay, and recurrence, are not well described.

Methods: This is a retrospective study of adult rattlesnake envenomations treated at a regional toxicology center both during and after routine use of antivenom maintenance dosing. Patients admitted to a regional toxicology service for rattlesnake envenomation between 2009 and 2012 were included. In 2011, practice policy at the study institution changed from routinely using maintenance dosing antivenom to a clinical and laboratory triggered (PRN) dosing strategy. Patients were divided into two groups. The first group included patients admitted between 2009-2010 (maintenance dosing), the second group included patients admitted between 2011-2012 (PRN dosing). Patients were excluded if they received an antivenom dosing schedule not consistent with the practice policy that year. Additional exclusion criteria included dry bites, patients not receiving antivenom, or patients receiving any antivenom other than Crofab®. Pearson Chi-Square analysis was performed for categorical data, and independent t-test and Mann-Whitney U Tests were performed for continuous data.

Table 1. Comparison of Hematologic Parameters

	Prothrombin time max sec (median, IQR)	Fibrinogen nadir mg/dL (median, IQR)	Platelet nadir K/mm^3 (median, IQR)
PRN	15.1 (14.1–16.8)	249 (192–282)	173 (129–210)
Maintenance	16.2 (15.0–17.61	219 (168–296)	169 (88–200)
p value	0.003	0.368	0.175

Results: Between 2009 and 2012, 218 rattlesnake envenomations were identified and 134 met inclusion criteria. Ages ranged from 14 to 88 years. Men accounted for 77% of envenomations. Baseline patient characteristics, including age, sex, presence of systemic symptoms of envenomation, bite location, and lab values were similar. Time to antivenom was not significantly different between PRN and maintenance groups (390 minutes and 585 minutes, respectively; p = 0.320). A median of 8 (IQR 6-12) and 16 (IQR 12-18) vials antivenom were used in the PRN and maintenance groups, respectively; p < 0.001. Hospital length of stay was shorter in the PRN group (21.3 (IQR 18.0-38.8) vs. 31.3 hours (IQR 24.0-40.9); p = 0.004). ICU length of stay was also reduced in the PRN group (20.0 (IQR 16.1-24.5) vs. 24.3 (IQR 17.8-35.5) hours; p = 0.004). Except for maximum prothrombin time, there were no differences in hematologic outcomes prior to discharge between groups (Table 1). Limited follow up data were available, however there was no difference in readmissions for recurrence between those receiving PRN (4) and maintenance (6) dosing; p = NS.

Conclusions: Initiation of a PRN strategy for antivenom dosing after rattlesnake envenomations was associated with decreased antivenom use and shorter lengths of stay than routine maintenance dosing. This benefit was not associated with negative effects on hematologic parameters.

KEYWORDS Rattlesnake envenomation; antivenom; maintenance

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4. Incidence of hypersensitivity reactions following copperhead snakebites treated with Fab AV or placebo

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Background: Risk of adverse reactions to antivenom remains a concern for many clinicians. A meta-analysis of patients treated with ovine-derived Crotalidae polyvalent immune Fab AV (CroFab®, BTG International) estimated immediate hypersensitivity

reactions or serum sickness were 8% and 13%, respectively. Because the included studies could not distinguish between venom effects and antivenom effects, the meta-analysis conservatively attributed all reactions to the antivenom. We still do not know the comparative incidence of reactions to snakebites between patients with snakebites of similar severities treated with Fab AV or no antivenom at all. Objective - To compare the incidence of hypersensitivity reactions following copperhead envenomation treated with Fab AV or placebo in a double-blinded, randomized, controlled trial.
Methods: Patients with copperhead snakebites received treatment and follow-up in a prospective, multicenter, randomized, controlled trial of Fab AV or placebo (ClinicalTrials.gov Identifier NCT01864200). The treatment allocation included Fab AV to placebo ratio of 2:1. All patients had 24 h of observation with follow-up at hospital discharge. Scheduled follow-up visits occurred at 3, 7, 14, 21, and 28 d after discharge. We included all patients who received at least one dose of study treatment. We reviewed all treatment-emergent adverse events (TEAEs). We used a previously published scale to classify likely hypersensitivity reactions as MILD (skin and subcutaneous tissues only), MODERATE (respiratory, cardiovascular or gastrointestinal), or SEVERE (hypoxia, hypotension, or neurological compromise).
Results: The trial enrolled 74 patients (39 M, 35 F) at 12 sites with 45 patients (23 M, 22 F) treated with Fab AV and 29 patients (16 M, 13 F) treated with placebo. Five of 45 Fab AV patients and 4 of 29 placebo patients had moderate envenomations; the remaining ones were mild. There were 25 FabAV patients 8 placebo patients who had >/= 1 TEAE. Mild skin reactions occurred in 11/45 FabAV patients (pruritis in 6, urticaria in 5, rash in 4, ecchymosis in 2, erythema in 1) and 1/29 placebo patients (pruritis in 1). Moderate GI TEAEs occurred in 7 (16%) of FabAV patients (nausea in 4, vomiting in 2, constipation in 2, diarrhea in 1, oral paresthesia in 1) and in 2 (7%) of placebo patients (nausea in 2). Respiratory TEAEs occurred in 3 (7%) of FabAV patients (dyspnea in 1, pulmonary embolism in 1, nasal congestion in 1, sneezing in 1) and no placebo patients. Hypotension (severe reaction) occurred in 1 patient in each group.
Conclusions: In a randomized, double-blinded, placebo-controlled trial of Fab antivenom for copperhead bites, the incidence of hypersensitivity reactions is low. The majority of reactions were mild skin reactions.

KEYWORDS Antivenom; adverse events; copperhead

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5. Impact of a Chronic Pain Management Pathway on Opioid Administration and Prescribing to Adult Patients in an Emergency Department.

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Background: Prescription opioid abuse and misuse is a significant public health crisis. Emergency Medicine providers are challenged with managing this health crisis while balancing appropriate and ethical treatment of pain. In 2012, an opioid prescribing pathway for patients with chronic pain presenting to the Emergency Department (ED) was implemented to reduce the inappropriate use of opioids while providing appropriate and compassionate medical care. The objective of this study is to determine the impact of the pathway on administration of opioids in the ED as well as the prescribing of opioids for home use after discharge.
Methods: Retrospective cohort study of consecutive patients presenting to the ED with acute and chronic pain complaints before and after implementation of the pathway. For the purposes of this study, we included patients with chronic abdominal pain and chronic back pain, defined as pain present for greater than three months and acute pain as acute long bone fracture. Medications administered, dosing and demographic data were collected from the electronic medical record. Descriptive statistics were performed.
Results: We identified 266 chronic pain patients before pathway implementation and 263 chronic pain patients after pathway implementation. We identified 163 acute pain patients before pathway implementation and 170 after implementation. Before pathway implementation, there was no statistically significant difference in the mean morphine equivalent dose administered for chronic or acute pain patients. After pathway implementation, there was a decrease in morphine equivalent dose administered to chronic pain patients ($p = 0.0200$) but not to acute pain patients ($p = 0.0820$). However, there was a decrease in fentanyl administration for acute pain patients ($p = 0.0250$). The number of patients with chronic pain who received one or more opioid prescriptions upon discharge from the ED decreased by 13.52% ($p = 0.0017$). In the acute pain cohort, no significant differences were found in the number of patients who received opioid prescriptions upon discharge ($p = 0.7749$). However, the number of pills per prescription decreased by 2.49 ($p = 0.0017$) and the morphine equivalent per prescription decreased by 32.20 ($p = 0.0361$).
Conclusions: After the implementation of an opioid prescribing for chronic pain patients presenting to an Emergency Department, there is a decrease noted in opioids administered to patients with chronic pain both in the ED and prescriptions on discharge. In patients presenting with acutely painful conditions, there was overall no decrease in opioid administration to patients in the ED.

KEYWORDS Opioid; pain; prescribing

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6. Public Health Actions Based on Incidents of Public Health Significance Identified by National Surveillance of Poison Center Data

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Background: The National Poison Data System (NPDS) is the national reporting database and a surveillance system for poison center (PC) data. The Health Studies Branch (HSB) of the Centers for Disease Control and Prevention uses NPDS to identify incidents of potential public health significance such as disease outbreaks. NPDS surveillance algorithms detect anomalies in call and health effect volume and identifies calls about substances of interest such as bioterrorism agents. Staff from HSB and the American Association of Poison Control Centers review these anomalies to determine if the anomaly meets CDC's criteria for potential public health significance and if the PC and state or territorial epidemiologist should be notified. A follow-up SurveyMonkey™ questionnaire is sent to the state epidemiologist three days after notification to assess public health action taken based on the incident. Our objective was to analyze survey responses from January 1, 2015 to January 5, 2016.
Methods: Outcome variables included number of notifications sent, surveys completed, and survey results. The latter included if

action was taken, action type, if the state was aware of the incident prior to the notification, and if the notification contributed to any action. The descriptive analysis was performed using SAS 9.3.
Results: During the study period, HSB sent out 47 notifications and 35 survey requests; some incidents did not have surveys sent for varied reasons such as a second notification about the same incident. The majority of those who received surveys completed them (n = 24; 68.5%). Most respondents (n = 20, 83.3%) reported that action was taken as a result of the incident. The most common action reported was "request for more information from patient, hospital, poison center or other entity" (n = 14, 70.0%) followed by "public health investigation activity" (n = 10, 50.0%). Half of respondents (n = 12, 50.0%) reported awareness of incident prior to HSB notification and half (n = 12, 50.0%) reported no previous awareness. For the majority of respondents (n = 15, 62.5%), the email notification did not contribute to initiating any action. However, seven respondents (29.2%) reported that the email did contribute to the decision to take public health action, such as sending a health alert to providers. Of respondents not previously aware of the incident prior to the notification (n = 12), over half responded the notification had contributed to the decision to take some public health action (n = 7, 58.3%). Of respondents that were previously aware of the incident (n = 12), almost all responded the notification did not contribute to any public health action (n = 11, 91.7%).
Conclusions: National surveillance of poison center data can be used to inform state health departments about incidents of public health significance in their jurisdictions that they might not be aware of and which may require public health action by them.

KEYWORDS NPDS; Public Health; Surveillance

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7. Identifying the holes in our safety net: Socioeconomic disparities in callers to a regional Poison Center

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Background: Poison center penetrance is the annual rate of human poison exposure calls per 1,000 persons. Penetrance has been shown to vary by cultural background and county-level sociodemographic characteristics. However, sociodemographic variability in penetrance and Poison Center (PC) reporting can be assessed with precision at a more granular level through analysis of United States Census tracts. This may facilitate a better understanding of factors contributing to individual poison center penetrance. Objective - To identify predictors of regional Poison Center (PC) use at a level exceeding current knowledge, and to elucidate the geospatial distribution of PC callers.
Methods: Retrospective review of all closed human exposure calls initiated by non-healthcare providers to a regional Poison Center (PC) between 1 January 2010 and 31 December 2014. Exposure substance, gender, age, and zip code were collected and geocoded to 2010 US Census tract data for household income, educational attainment, age, and primary language. The resulting dataset was spatially apportioned to US census tracts based on their quantifiable spatial and population overlaps. Resulting demographic data were analyzed by descriptive statistics and linear regression to define predictors of PC calls.
Results: 168,630 exposure calls from non-healthcare settings were identified. 159,794 records included zip codes. Calls originating outside of the state and calls with zip codes that could not be geocoded were excluded. 156,805 records were geocoded for analysis. Penetrance ranged from 0.081 - 38.47 calls/1,000 population/year (mean 5.84 calls/1,000 population/year). After predictor variable transformation, linear regression revealed positive associations between penetrance and educational attainment of 8th grade or higher ($p = 0.001$) and larger proportions of non-Hispanic blacks ($p = 0.019$) or American Indian residents ($p < 0.001$). Negative associations were evident in populations with higher proportions of Hispanic residents ($p = 0.001$). Geospatial mapping of penetrance demonstrated significant variability in penetrance at a level of granularity previously unexplored (figure provided on acceptance).
Conclusions: PC calls originating from non-healthcare providers vary substantially across sociodemographic strata. In this study, US Census tracts with higher proportions of non-Hispanic black or American Indian residents and those with a higher proportion of residents with educational attainment at or in excess of the 8th grade had higher PC call penetrance, while tracts with high proportions of Hispanic residents had lower PC penetrance. Geospatial mapping, when coupled with sociodemographic correlates, may reveal disparities in PC access and identify communities for which the utilization of PC resources is not yet optimized.

KEYWORDS Poison Center; socioeconomic desparities; penetrance

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8. webPOISONCONTROL®: Can Poison Control be Automated?

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Background: A new, free webPOISONCONTROL® app allows the public to determine the appropriate triage of many poison ingestions without calling Poison Control. The webPOISONCONTROL® user provides substance name, amount swallowed, age and weight in an automated online tool or downloadable app, and is given a specific triage recommendation to stay home, go immediately to the ER, or call Poison Control for further guidance.

Table 1. Linear regression analysis Poison Center Penetrance by U.S. Census Bureau sociodemographic characteristics

Penetrance	Coefficient	Standard Error	t	P>|t|	[95% Confidence Interval]	
>8th grade education	5.57	1.69	3.29	0.001	2.25	8.89
Population <5 years old*	−0.91	0.94	−0.96	0.34	−2.76	0.94
Asian population*	0.22	0.46	0.49	0.63	−0.68	1.12
Non-Hispanic black population*	0.94	0.40	2.34	0.019	0.15	1.73
Non-Hispanic American Indian population*	3.07	0.55	5.54	0.000	1.98	4.15
Hispanic*	−1.62	0.47	−3.46	0.001	−2.54	−0.70
Households below the poverty Line	−0.03	0.46	−0.07	0.95	−0.94	0.88

*Square-root transformation of variables to achieve normality.

If accepted and safe, this alternative expands access to reliable poison control services to those who prefer the internet over the telephone and could eventually lead to dramatic cost-savings. This study was conducted to determine feasibility, safety and user-acceptance of automated online triage of asymptomatic, non-suicidal poison ingestions.
Methods: Consecutive non-duplicated public cases were analyzed. Automated triage safety was determined by assessing outcomes of home-triaged cases with follow-up and the correct application of algorithms through toxicologist review of every case. Case completion times and user perceptions of speed and ease of use were analyzed to assess user-acceptance.
Results: Of 9256 completed cases, 73.3% were triaged to home, 2.1% to an emergency department, and 24.5% directed to call poison control. 86.3% of cases were from the U.S.; 13.7% were from 86 other countries. Children <6 years were involved in 75.2% of cases and 50.7% of all cases were either 1 or 2 years of age. Automated follow-up was completed in 31.2% of home-triaged cases (1 to 6 follow-ups per case), and 82.3% of these had no effect, 10.4% had minor effects, and 1.4% had moderate effects. No major or fatal outcomes were reported. Twenty-six cases initially triaged to home had an automated change in triage recommendation based on data the user entered on follow-up. Pharmaceuticals were implicated in 40.5% of cases; 59.5% involved household products or plants. Cigarettes were the leading substance in children younger than 3 years. User recommendations and algorithm adherence were consistent from case to case and triage calculations were uniformly accurate. More than 91% of survey-respondents found the tool quick and easy to use. The median webPOISONCONTROL® case completion time was 4.1 minutes; 96.1% of users obtained a recommendation in less than 10 minutes.
Conclusions: webPOISONCONTROL® augments traditional poison control services by providing automated, accurate online access to case-specific triage and first aid guidance in a poison ingestion emergency. This study shows this online solution is safe, quick and easy to use. If usage increases, webPOISONCONTROL® could decrease the cost of providing poison control services.

KEYWORDS Automated poison control; online poison control; virtual poison center

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9. Intravenous cobinamide successfully rescues swine (Sus Scrofa) in a model of hydrogen sulfide toxicity and apnea

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Background: Although hydrogen sulfide is considered an uncommon cause of poisoning death, deaths due to H2S appear to be on the rise. Between 2008 and 2010, the reported instances of intentional death by H2S gas inhalation have increased in the U.S. from 2 cases to 18. Furthermore, research suggests that the incidence of H2S suicide is probably underestimated by public health officials. Although the number of suicide reports is relatively small, the mortality rate remains extraordinarily high and poses significant hazards for first responders and bystanders. Objective - To compare survival among groups of swine with acute H2S induced apnea treated with intravenous (IV) cobinamide, IV hydroxocobalamin or saline and to evaluate the model.
Methods: Twenty-four swine (45-55 kg) were anesthetized, intubated, and instrumented with continuous femoral and pulmonary artery pressure monitoring. After stabilization, anesthesia was adjusted such that animals would spontaneous ventilate with an FIO2 of 0.21. Sodium hydrosulfide (NaHS; concentration of 8 mg/ml) was infused at 1mg/kg/min until 1.5 minutes post apnea and then decreased to a final infusion of 0.1 mg.kg/min. One minute post apnea animals were randomly assigned to receive cobinamide (4.2 mg/kg), hydroxocobalamin (4 mg/kg) or saline and monitored for 60 minutes. G* power analysis using the Z test determined that equal group sizes of 8 animals were needed to achieve a power of 80% and α of 0.05.
Results: There were no significant differences in baseline variables nor was there a significant differences in the mg/kg dose of NaHS (5.6 mg/kg; $p = 0.45$) to produce apnea. All of the cobinamide treated animals survived, none of the control or hydroxocobalamin treated animals survived ($p < 0.001$). Urine sodium thiosulfate levels (μg sodium thiosulfate/mg creatinine) demonstrate the initial significant increase (mean baseline 23.0 ± 26.6 μg to mean peak levels ten minutes post apnea 1224 ± 2675 μg; $p < 0.05$) in the biological marker for toxicity with subsequent decrease in those animals that survived (mean 60 min 465 ± 92.6 μg). Moreover, cytokine data supports the model as toxic but survivable. As in severe lung infection/injury, expression of IL-1β and TNF α increased within minutes ($p < 0.05$) in all groups and in the case of the cobinamide treated animals began to decrease. IL-6 also increased ($p < 0.05$), but had a more sustained increase in expression such that there was no difference or interaction among the groups.
Conclusions: Cobinamide successfully rescued the severely NaHS-poisoned swine from apnea in the absence of assisted ventilation. This model appears to be robust and potentially may be used to evaluate other antidotes to H2S.

KEYWORDS Cobinamide; hydrogen sulfide; resuscitation

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10. In-vivo evidence of renal and hepatic dysfunction following the administration of diglycolic acid, the toxic metabolite of diethylene glycol

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Background: Diglycolic acid (DGA) is one of two primary metabolites of diethylene glycol (DEG). Diethylene glycol is an industrial solvent that has been implicated in mass epidemiological poisonings in the United States and countries abroad, with the hallmark sign of toxicity being renal failure. Our lab has strong in-vitro evidence suggesting that DGA is the metabolite responsible for the proximal tubule necrosis observed following ingestion of DEG, leading to decreased kidney function. Furthermore, we've also shown that DGA accumulates remarkably in kidney tissues (100X) following DEG administration. Therefore, we hypothesize that oral administration of DGA will result in renal and hepatic DGA accumulation, as well as proximal tubular necrosis and liver injury.
Methods: To measure the in-vivo effects of direct DGA administration, adult male Wistar rats were divided into three treatment groups (control or DGA at 100 or 300 mg/kg) and dosed via oral gavage. Urine was collected every 6-12 hours and animals were

sacrificed at 48 hours and blood, kidneys, and liver were removed for analysis.
Results: DGA accumulated significantly in both kidney and liver tissue only at the high dose. DGA concentrations in the kidneys correlated significantly with increased markers of renal injury (blood urea nitrogen [BUN], creatinine and kidney injury molecule-1 [KIM-1] urine protein), while DGA concentrations in the liver also correlated significantly with the liver injury marker, aspartate aminotransferase (AST). Moreover in the high dose rats only, histopathological analysis revealed severe vacuolar degeneration of the proximal tubule with necrosis, as well as substantial fatty changes in the liver. Blood pH and blood bicarbonate were slightly decreased in high dose DGA-treated animals, indicating mild acidosis.
Conclusions: These results provide further evidence for DGA as the toxic metabolite of DEG, suggesting that DGA is primarily responsible for the toxicity observed in mass poisonings. DGA exhibited moderate liver damage and marked renal injury, with the latter confirming what previous in-vitro studies have demonstrated.

KEYWORDS Diethylene glycol; nephrotoxicity; hepatic toxicity

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11. Translational research: the effects of pesticide ban policy on poisoning epidemiology and deaths in Sri Lanka

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Background: Deliberate pesticide self-poisoning is a major contributor to suicide deaths in the developing world. In Sri Lanka dimethoate, fenthion and paraquat had been identified as having high relative toxicity in self-poisoning. In 2008 regulatory authorities in Sri Lanka made a policy decision to remove these compounds from the market place over 3 years. It was estimated that these bans could result in a 30% reduction in deaths. This study examines the effectiveness of these bans on deaths and epidemiology of poisoning.
Methods: Prospective data was collected on all self poisonings presenting to 6 Sri Lankan hospitals for the years 2006-2015. Data was analyzed by aggregated types of poisoning for the proportion of total presentations and the case fatality rate for each group. Within the agrochemical group data was dis-aggregated and analyzed for specific agents to all allow the effects of the ban to be estimated and post ban substitutions to be identified. Agrochemicals that had not been banned were examined for evidence of reduction in mortality which could be an improvement in treatment
Results: There were 54,463 admissions with poisoning during the study period. The pesticide restrictions were associated with a rapid decline in presentations of banned pesticides. Overall mortality in the cohort dropped from 5.71 to 2.1% (Risk ratio 0.37, 95% CI:0.29- 0.47). Two major contributors to this change was identified. A reduction in agrochemical mortality from 8.9 to 4.6% which was due both to the pesticide bans and a reduction in mortality of up 50% in non banned agrochemicals that suggested improved treatment responses. The major identified post ban substitution products were chlorpyrifos for organophosphate and glyphosate for paraquat. The epidemiology of poisoning altered with a reduction in the proportion of patients presenting with agrochemicals from 48.6% to 30% and an increase in the the less toxic group of medicines from 18.8% to 40%. Modeling the changes for a representative cohort of 1000 patients was undertaken, observed deaths fell from 61/1000 in 2006 to 19/1000 in 2015. The reduction in deaths could be attributed to a changing epidemiology (15/1000), paraquat bans (12/1000), organophosphate bans (4/1000) and a conservative estimate of improved care (15/1000). The changes were consistent with those seen in the national health statistics.
Conclusions: The translation of research to policy to practice to remove toxic pesticides from the market was associated with a reduction in deaths that was consistent with the pre-restriction estimates. Changes in epidemiology and care had effects of similar size.

KEYWORDS Pesticide; suicide; translational

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12. Impact of Packaging Standardization on Dosing Errors Involving Pediatric Liquid Acetaminophen

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Background: Pediatric medication dosing errors (MDEs) involving liquid single ingredient (SI) acetaminophen medications are commonly reported to US poison centers (PCs). Recent efforts, including guidance from the FDA, have eliminated multiple concentrations of these products to reduce caregiver confusion and have encouraged further standardization of labeled dosing information and in-pack dosing devices. We sought to determine if increased standardization of packaging elements impacted reported amount of drug (dose) involved in MDE cases reported to US PCs.
Methods: All pre-hospital MDEs of SI acetaminophen involving children <12 years old at 6 participating PCs were eligible. Parents were contacted within several days of the event and invited to participate in a phone survey which included product verification. PC narratives were used to extract dose while PC survey data were used to evaluate packaging type by standardized (standardized dosing device and industry-standard acetaminophen concentration of 160 mg/5 mL) vs. non-standardized (dropper dosing device or acetaminophen concentration other than 160 mg/5 mL) packaging. Data were limited to reports between 01Aug13-31Jan15. The Wilcoxon rank sum test was used to compare distributions of the reported max individual dose. The number of cases greater than or equal to the max recommended individual dose (15 mg/kg) was also evaluated.
Results: 618 parents completed a MDE survey. Pediatric SI acetaminophen liquids were the only product ingested in 487 children of which 439 (71%) had evaluable dose, weight and reported packaging type. Of these, 295 (67%) involved a child <2 years, 73 (17%) a child aged 2-3 years, 34 (8%) a child aged 4-5 years, and 37 (8%) a child aged 6-11 years. Male children were reported in 220 (50%) cases. The max individual dose was lower with standardized packaging, as compared to non-standardized packaging. A lower percentage of cases with standardized packaging type had a max individual dose >15 mg/kg compared to non-standardized packaging type ($p = 0.0350$). The odds of a dose >15 mg/kg were 1.8 times larger with non-standardized packaging than standardized (95% CI: 1.04, 3.20).

	Standardized (n = 370)	Non-Standardized (n = 69)
Median (IQR) of Max Individual Dose (mg/kg)	17.7 (12.5, 27.1)	23.5 (14.7, 34.1)
Max Individual Dose =15 mg/kg	212/370 (57%)	49/69 (71%)

Conclusions: MDEs of liquid SI acetaminophen involving standardized packaging were significantly less likely to involve a larger than recommended dose (≥15 mg/kg) compared to non-standardized packaging. Continued encouragement to further standardize labeled dosing information and in-pack devices would likely reduce MDEs resulting in overdose.

KEYWORDS Acetaminophen; medication error; pediatric

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13. Does two-stage intravenous N-acetylcysteine (NAC) regimen in acetaminophen overdose offer similar hepatic protection to the FDA three-stage intravenous regimen in Pediatrics?

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Background: Acetaminophen is the most common cause of acute liver failure in the United State. The FDA-labeled dosing formulation of intravenous NAC is a three-stage infusion regimen: a bolus over 60 minutes, a 4–hour infusion, and a 16-hour infusion. Few studies suggested that this complex formulation has led to unnecessary medication errors; therefore they recommended a two-stage infusion regimen. The Montreal Children's Toxicology Service adopted a two-stage regimen in 2003. The regimen consists of 150mg/kg bolus over 50 minutes, followed by a 20-hour infusion of 150 mg/kg. It infuses the same total dosage suggested by the FDA: 300mg/kg.
We hypothesized that the two-stage NAC regimen is as effective as the FDA-labeled regimen with less complexity and smaller chance of administration errors.
Methods: Retrospective chart review of two cohorts. We compared the three-stage regimen cohort who was prior to May 2003 and the two-stage regimen cohort from May 2003 to May 2013. Inclusion criteria - Children who received intravenous NAC for the treatment of acute acetaminophen overdose. Exclusion criteria: (1) History of chronic liver disease; (2) Received intravenous NAC for any other indication; (3) Received oral NAC therapy or had NAC infusion terminated for a sub toxic level; (4) Presented with hepatitis; (5) History of chronic acetaminophen toxicity; (6) Acetaminophen abused in the extended release form.
Outcome measures - Incidence of hepatotoxicity which was defined as ALT or AST >1000 u/L. Data reflecting medication errors: unnecessary delays, interruptions or incorrect dosage of NAC.
Results: 448 charts reviewed. 126 patients met the inclusion criteria. 65 patients received the two-stage regimen and 61 patients received the FDA labeled regimen. Hepatotoxicity (ALT or AST >1000 u/L) developed in 2 out of 65 of the two-stage regimen and and 2 out of the FDA labeled regimen (Pearson chi2 = 0.004 and P value =0.9). All hepatotoxicity have resolved, no patient had liver transplant or died. There were a total of 27 NAC administrations errors. 4 errors in the two-stage regimen (2 interruption errors, 1 dose related error and 1 missing loading dose) and 23 errors in the FDA labeled regimen (15 interruption errors, 6 dose related errors, 1 missing loading dose and 1 infusion stopped before 21 hours), with statistically significant difference P value <0.001. There was no major adverse reaction in the two-stage regimen.
Conclusions: the two-stage regimen is effective, well tolerated and has less administration errors than the FDA-labeled regimen. The two-stage regimen could be an effective and safe alternative to the FDA labeled regimen.

KEYWORDS Two-stage N-acetylcysteine regimen; simplified N-acetylcysteine regimen; alternative N-acetylcysteine regimen

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14. The impact of one-bag N-acetylcysteine dosing on administration delays: a five-year look

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Background: Previous research has reported that the traditional three-bag IV N-Acetylcysteine (NAC) administration method can bring about significant delays in therapy during the process of ordering, admixing, and delivery of each dose. This prompted the development and implementation of a one-bag IV NAC regimen at our institution. The 300 mg/kg dose received by the patient using the one-bag method is identical to the three-bag method. Rather than hanging three separate medication bags, a new infusion rate is programmed into the smart pump for each consecutive dose. This project sought to compare administration delays during IV NAC regimens for 2.5 years before and after the implementation of this method.
Methods: Our institution implemented the one-bag IV NAC delivery method in March 2013. A retrospective chart review was performed for all IV NAC orders between January 2011 and November 2015. Data collected included time to receive the total dose, delay between the end of dose 1 and start of 2 (delay #1), and delay between end of dose 2 and start of dose 3 (delay #2). An independent samples t-test was used to compare pre-implementation and post-implementation groups.
Results: Chart review revealed 39 patients who received the traditional three-bag regimen and 36 for the one-bag method. Average time to complete a full course with the one-bag regimen compared to a 3-bag regimen was 21:48 (hh:mm) vs. 24:50 (−3:01, p < 0.001, 95% CI −4:40, −1:22). Delay #1 was 0:20 vs. 1:31 (−1:10, p < 0.001, 95% CI -1:44, -0:36), and delay #2 was 0:27 vs. 2:25 (−1:58, p = 0.013, 95% CI -3:30, −0:26).
Case Discussion: There are several limitations to this study. Institutional changes during the study period including introduction of smart pump technology and staff training with the one-bag protocol may have contributed to the reduction in administration delays.
Conclusions: Our data suggests that the implementation of the one-bag NAC regimen has decreased administration delays between doses at our institution. While it can be hypothesized that this decrease may enhance patient care, further research is needed to evaluate clinical outcomes and whether incidence of side effects differs with this version of the one-bag method.

KEYWORDS N-Acetylcysteine; Antidote; Dosing

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15. Treatment of acetaminophen overdose with a 12 h acetylcysteine regimen: The first report of safety and efficacy in routine clinical practice

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Background: Acetylcysteine (NAC) is effective at preventing acute liver injury after acetaminophen overdose. The standard 21 h intravenous regimen is associated with significant adverse effects, particularly anaphylactoid reactions, that cause interruption to treatment and prolong admission to hospital. The SNAP trial demonstrated that a modified 12 h NAC regimen ('SNAP regimen') was associated with substantially fewer adverse effects (Lancet: 383:697-704). However, this trial was not sufficiently powered to robustly determine efficacy with regard to preventing liver injury. The SNAP regimen is now standard clinical practice for delivery of NAC following acetaminophen overdose in all patients at the Royal Infirmary of Edinburgh, UK. This is the first report of the safety and efficacy of using the SNAP regimen in routine clinical practice.

Methods: The SNAP regimen was introduced at the Royal Infirmary of Edinburgh on 28th September 2015 for all patients requiring treatment for an acetaminophen overdose. Briefly, this regimen consisted of intravenous NAC 100mg/kg over 2 h then 200 mg/kg over 10 h. At the end of the second bag, NAC treatment was discontinued if: INR <1.3; AND ALT <100 U/L and not more than doubled from admission; AND acetaminophen concentration <20mg/L. If there was evidence of liver injury then NAC was continued at 200mg/kg over 10 h. Therefore, higher risk patients received more NAC (500mg/kg) compared with the standard 21 h regimen (300mg/kg). All patients, irrespective of whether NAC was continued or discontinued, had further blood sampling 8 h later to determine the need for extended treatment (at the equivalent time point as the standard 21 h regimen). An audit of consecutive patients was conducted for a 5-month period before and after the regimen change. Statistical analysis was performed by a two-tailed Fisher's exact test.

Results: There were 267 admissions in the pre-change period, of which 209 received the standard 21 h NAC regimen and there were 310 admissions post-change, of which 262 received the SNAP regimen. Anaphylactoid reaction or the need for an antihistamine was reported for 26 (12.4%) patients in the 21 h regimen group compared with 4 (1.5%) in the SNAP treatment group ($P < 0.0001$). Extended treatment because of acute liver injury was needed for 27 (12.9%) patients in the 21 h regimen group compared to 28 (10.7%) with SNAP treatment. No patients needed NAC restarted if it had been discontinued immediately after the second infusion.

Conclusions: The SNAP NAC regimen resulted in significantly fewer anaphylactoid reactions and this builds on the SNAP trial by demonstrating enhanced safety in an unselected patient population. Thus far, the SNAP regimen has similar efficacy to conventional therapy with regard to preventing liver injury.

KEYWORDS Acetylcysteine; Acetaminophen; Paracetamol

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16. Acetaminophen Elimination in Overdose – CAOS and US-NMS Experience

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Background: The elimination half-life of acetaminophen (APAP) is generally believed to increase in overdose and to be further increased in hepatic injury. We examined APAP half-life and predictors in all qualifying cases from the Canadian Acetaminophen Overdose Study (CAOS) and the National Multicenter Open Study of Oral N-acetylcysteine for the Treatment of Acetaminophen Overdose (US-NMS).

Methods: We examined 16,314 APAP concentrations from 4,301 patients admitted to hospital between the years 1980-2005 (CAOS) and 1976-1985 (US NMS). We selected those APAP concentrations >10 and <1000 mcg/ml that were obtained >3.5 and <48 hours post-ingestion. Cases where the first APAP concentration was obtained <20 h post-ingestion and where >2 concentrations were drawn, were selected. We calculated the half-life via linear regression of log[APAP] and extracted highest observed [APAP] (max[APAP]), time of first [APAP], age, sex, pre-existing liver disease, alcoholism and concomitant medications in 8 categories including ethanol. Outcome measures included max aminotransferases (AST, ALT) max INR, max BUN, and max creatinine. Data which were log-normally distributed were log transformed. Predictors of half-life were selected by stepwise multiple regression with p-value thresholds of 0.1 to enter and 0.05 to leave. R-squared, the fraction of the half-life variability described by the model (rsqr) was reported. LogWorth and Pvalue were used to assess the statistical contribution of each predictor. All analyses were via SAS JMP 12.0.1.

Results: The analysis dataset included 3012 APAP levels from 756 patients (450 from CAOS and 306 from US-MCS). Median [min, max] half-life was 3.68 [1.60, 23.3] hours, 537 (71%) were female, and there were 2 deaths. The best multivariate model of half-life (excluding outcome measures) included max[APAP], time of first [APAP], sex, and pre-existing liver disease ($n = 756$, $rsqr = 0.146$, $p < 0.0001$). The table shows the best multivariate model of half-life including outcome measures ($n = 539$, $rsqr = 0.380$, $p < 0.0001$). There were only 539 patients because these outcome measures were not available for all patients. The last column shows the half-lives predicted by the model for the extremes (min, max) of each predictor with the other predictors set to their median values. Thus an INR of 0.8 gave a half-life of 3.12 hours and an INR of 11.4 gave a half-life of 7.93 hours. The predicted half-life for a patient with the max value of all 7 predictors would be 25.1 hours.

Conclusions: These large datasets permit both a statistical test of our assumptions and a sound predictor of APAP half-life (and its variability). Results confirm the increase in half-life with larger dose (max[APAP]), alcoholic status, and liver disease, though these affects were relatively modest. Other predictors (age, sex, concomitant medications, and creatinine) did not contribute statistically to the half-life model. The results suggest a greater relative importance of increased INR and transaminases as indicators of a reduced rate of APAP elimination. The half-life model emphasizes the importance of the outcome measures in explaining the elimination of APAP and suggests their relative contribution to hepatic injury.

Acetaminophen Half-life Model including Outcome Measures

Source (Predictor)	Log Worth	P value	Median [min, max] untransformed	Half-lives associated with [min, max]
Max(INR)*	9.47	<0.0001	1.17 [0.8, 11.4]	3.12, 7.92
Max(AST, ALT)* (U/L)	7.44	<0.0001	33.1 [5.19, 350]	3.22, 5.04
Max[APAP]* (mog/mL)	3.45	0.0004	149 [24,828]	3.07, 4.11
Alcoholic	2.19	0.0065	No/Unknown, Yes	3.57, 4.07
Liver disease	1.37	0.0427	No, Yes	3.30, 4.09
Max(BUN)	1.35	0.0446	11 [1.3, 195]	3.49, 5.34

*Indicates log transformed data were used

KEYWORDS Acetaminophen overdose; Elimination half-life; Statistical models

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17. I dream of rain: Desert Rose and hidden glycosides causing detectable digoxin concentrations

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Background: Cardiac glycosides inhibit the sodium/potassium ATPase pump, indirectly increasing intracellular calcium and, thus, cardiac contractility. Naturally occurring cardiac glycosides are numerous; some, such as thevetin A & B (Thevetia peruviana), are responsible for a significant global burden of morbidity and mortality attributable to self-harm. Intentional injury with these plants in the US is less common. Adenium obesum (Desert Rose) contains cardiac glycoside alkaloids throughout the plant. We report the case of a 19-year-old woman became symptomatic following intentional ingestion of Adenium obesum leaves.

Case Report: A healthy 19-year old woman presented to the urgent care of a large, public university complaining of abdominal pain, nausea, vomiting, and dizziness. Examination revealed a nontoxic-appearing woman with equal, round, reactive pupils, a benign abdomen with normal bowel sounds. Initially recorded vital signs included a pulse of 78 beats per minute (BPM), blood pressure of 127/88 mmHg, and a respiratory rate (RR) of 18 on room air. The patient abruptly excused herself to the restroom, subsequently returning with a hand-written note reading "I am sorry I deceived you. I ate Adenium obesum." She admitted to consuming many leaves to harm herself, vomiting once, 3 hours prior to arrival. The treating physician consulted the regional Poison Center (PC), and transfer to the emergency department (ED) for activated charcoal (AC), basic labs and electrocardiogram, and extended observation was collaboratively agreed upon. In the ED she developed fatigue, nausea, and an episode of emesis. Following a dose of AC, pulse was 69 BPM, blood pressure 101/63 mmHg, and RR 20. EKG demonstrated terminal inversion of the anterior precordial t-waves with normal PR (140 msec), QRS (72 msec), and QTc (412 msec) intervals. PC recommended overnight observation and a digoxin assay for qualitative evidence of cardiac glycosides, and dixogin Fab fragments only if severe toxicity developed. Laboratory results 7 hours after ingestion included a digoxin concentration of 1.5 μg/L, undetectable serum acetaminophen concentration, and potassium of 3.6 mEq/L. During the evening, the patient's pulse dipped to the 50's and she experienced a moderate headache, orthostasis, and several additional bouts of emesis. The next morning she was asymptomatic with normal vital signs. She was assessed by consulting psychiatrists and transferred to inpatient mental health. Administration of digoxin Fab fragments was not required.

Case Discussion: Intentional ingestion of Adenium obesum is not previously reported in the English-language literature. In vitro assessments of Adenium obesum elucidate at least 30 cardiac glycosides, including 11 unique to this species (Yamauche & Abe 1990). The symptoms with which this patient presented were typical of mild cardiac glycoside toxicity. To our knowledge, this report is the first to demonstrate the qualitative presence of cardiac glycosides following Adenium obesum ingestion via quantitative total digoxin assay.

Conclusions: Though rarely reported, Adenium obesum (Desert Rose) ingestion may result in symptomatic cardiac glycoside toxicity. Total digoxin concentration assay was positive in this case, supporting the use of digoxin concentration assays to provide qualitative evidence of exposure to Adenium obesum.

KEYWORDS Adenium obesum; digoxin; cardiac glycoside

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18. Fatality Following Cantharidin Ingestion As Treatment For Gastric Cancer

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Background: Cantharidin (CAN) is a blistering agent found in "Spanish Fly," that is produced by blister beetles of the family Meloidae. Its use in Traditional Medicine dates back 2000 years, with recently renewed interest as a chemotherapeutic. Severe toxicity from CAN is reported, but death is uncommon. We report a patient who died after ingesting CAN as a treatment for gastric cancer.

Case Report: A 42 year old woman with advanced gastric cancer presented to the emergency department (ED) 3 hours after ingesting CAN imported from China. Within 30 minutes she developed dysphagia, dyspepsia, and oropharyngeal burning, followed by hematemesis and hematochezia. Vital signs were: BP, 132/87mmHg; HR, 90/min; RR, 20/min; Temp, 98.4 °F; O2 Sat, 97% on room air. Examination showed dried blood in her oropharynx but was otherwise unremarkable. Initial laboratories were: HCO3-15 mEq/L, K+ 2.9 mEq/L, Glucose 230 mg/dL, BUN 35 mg/dL, Cr 1.7 mg/dL, Hb 12.7 g/dL, WBC 11×10^3/mm3, Platelets 327×10^3/mm3, pH 7.32, PC02 29 mmHg, lactate 4.5 mmol/L. An ECG showed sinus tachycardia. Urinalysis was grossly bloody. A non-contrast abdominal CT scan was normal. 9 hours after ingestion she had multiple episodes of hematemesis and hematochezia. Repeat vital signs were: BP, 88/64 mmHg; HR, 114/min. She

received 2 units packed red blood cells. Her mental status deteriorated 15 hours after ingestion, and she was intubated for airway protection and massively transfused. Endoscopy was not performed due to concern for perforation. 18 hours after ingestion, she developed severe hemoptysis. A chest x-ray showed complete left lung opacification. While preparing for bronchoscopy, she developed bradycardia unresponsive to atropine and deteriorated into unrecoverable PEA arrest. Autopsy was declined due to religious beliefs. A quantitative assay for CAN is pending.
Case Discussion: Most reports of CAN toxicity involve its use as an aphrodisiac. Toxicity is dose dependent, occurring with local application or ingestion, and results in corrosive damage and systemic toxicity. CAN damages tissues directly through acantholysis. The most severe effects are GI and pulmonary hemorrhage, and acute tubular necrosis. CAN is also a potent inhibitor of protein phosphatase 1 and 2A, which prompted research into its chemotherapeutic use . Currently there are no approved cancer therapies based on CAN. Individuals seeking CAN as a cancer therapy must obtain it from non-regulated sources, raising the risk of toxicity from inappropriate use or dosing.
Conclusions: To our knowledge, this is the first reported fatality of CAN use as a chemotherapeutic. The potential public health risk of CAN as an alternative cancer treatment may increase as research into this use progresses.

KEYWORDS Cantharidin; alternative medicine; natural toxin

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19. Anticholinergic Toxicity from the Dermal Application of Topiricin®, A Homeopathic Remedy for Plantar Fasciitis

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Background: Homeopathy is an alternative medical practice where highly dilute preparations are administered to treat an ailment. The dilutions are of substances that, if administered to a healthy subject would correspond to the manifestations of the disease. The practice was founded under the assumption that "like should be cured with like". Despite the lack of scientific explanation behind the use of the dilutions, homeopathy is popular in the UK and US. Similar to other alternative therapies, there is very minimal regulation by the government, and none by the FDA.
Case Report: A 40-year-old man with a past medical history of hypertension presented to the emergency department (ED) with a change in mental status. According to his wife, he was not acting like himself that evening. He would repeat questions and did not recognize his family. Initial evaluation by neurology felt that he was not a candidate for TPA and likely diagnosis was transient global amnesia. He had a blood pressure of 180/90 mm Hg and heart rate of 86 bpm. His pupils were dilated and skin was dry and flushed. He became more confused and agitated and required Lorazepam for sedation. A bladder catheterization was performed and over 1000ml was collected. Upon further questioning, his wife recalled that he has been using a topical cream for foot pain called Topiricin multiple times a day for the past few days. Upon review of the ingredients, the mostly likely etiology of this anticholinergic delirium was Belladonna 6X. Physostigmine not given. He was admitted to the hospital and returned to his baseline. An elevated creatine kinase was treated with intravenous fluids and he was advised to stop the homeopathic cream.
Case Discussion: Adverse reactions (AR) to homeopathic treatments are rare, but do exist. Often they occur due to lack of proper diagnosis and therapy provided by conventional medicine. A systematic review done in 2012 in the UK showed the most common AR was allergic reactions, followed by ingestion of toxic substances. ARs to homeopathic therapies are likely to be under reported and a true incidence is unknown.
Conclusions: Many trust that homeopathic treatments are effective and safe, without sufficient evidence to support either belief. Theoretically, the amount of substances in homeopathic medications are intended to contain minimal concentrations. However, the lack of regulation makes this impossible to determine. It is important to keep these over the counter therapies in the differential diagnosis of any patient who presents to the ED with delirium.

KEYWORDS Anticholinergic; homeopathic; topical

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20. Metabolic alkalosis after "healthy" high pH water consumption

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Background: Alkaline water is a growing health fad in the United States. Numerous types of bottled alkaline water are available in grocery stores for consumption. Now commercial high pH water coolers are available as well.
Case Report: We report a case of an 83 year old male who presented to our emergency department after a syncopal event. He noted a prodrome of lightheadedness, but the event occurred without chest pain, palpitations, or shortness of breath. The patient had a previous history of coronary artery disease treated with stents. Upon arrival to the emergency department, his vital signs were a temperature of 36.8, heart rate of 62, blood pressure of 185/78, oxygen saturation of 100% on room air. EKG upon arrival revealed a normal sinus rhythm, normal intervals, no ischemic changes and frequent premature ventricular contractions. His CBC showed white blood cell count of 5.7, hemoglobin of 10.9, hematocrit of 32.4, and platelets of 132. A metabolic panel demonstrated a sodium of 140, potassium of 2.6, chloride of 94, bicarbonate of 38, BUN of 19, and creatinine of 1.4. The patient reported no history of tobacco use or COPD. Venous blood gas revealed a pH of 7.48, pco2 of 44, and a calculated bicarbonate of 32.5. The patient had previously been taking metoprolol, though discontinued it several weeks earlier after an alternative health practitioner informed the patient he could drink alkaline water instead. The patient consumed 6-8 glasses of high pH water per day for two months. He was drinking water from a Pure Water 3i + Alkaline. Per the sales representative the water distributed is at a pH of approximately 10. The patient was hospitalized for two days. He had a stress test which was negative for any inducible ischemia. After two days and a normal diet his repeat metabolic panel showed a sodium of 147, K of 3.3, chloride of 105, bicarb of 32, BUN of 17, and a creatinine of 1.08
Case Discussion: This case demonstrates the risks of alkaline water. The patient reported over two months of daily consumption of 6-8 glasses of high pH water. Likely his metabolic alkalosis from exogenous bicarbonate intake is what led to his hypokalemia. With the increase in health fads, physicians should be aware of excessive alkaline water as a potential cause of metabolic alkalosis.
Conclusions: While it is unclear if the metabolic alkalosis contributed to the patient's syncope, ongoing consumption of

commercially available alkaline water can cause metabolic alkalosis and resulting hypokalemia.

KEYWORDS Alternative health; alkalosis; health supplements

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21. Food supplement inquiries to a Poisons Center in 2013–2015

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Objectives: Food supplements (FS) are a growing group of consumer products, readily available in stores and via the internet. Poisons Centers (PCs) receive inquiries about these products and good quality product information is usually not available for assessing the toxicological risk. Our objective is to identify risky product groups within the FS.
Methods: Telephone inquiries to our PC are recorded in the PC database, including patient, exposure and symptom information. In case of food supplement inquiries, extra information is requested on the intended use and details on the origin of the product. In case of moderate or severe poisoning symptoms the call is followed up for outcome and samples of the supplement are requested for chemical analysis. Products causing serious health effects and/or containing regulated substances are reported to the National Health Care Authorities. The FS were classified into 4 different categories of intended use; Relaxing, Energizing (energy drinks, weight loss ("stackers") and pre-workout), Aphrodisiacs (mainly erectogens) and Other. Characteristics of exposures in these categories in 2013, 2014 and 2015 are described.
Results: A total of 1941 exposures to FS were registered (see table 1). Most FS calls concerned Relaxing FS, of which 73% contained melatonin and 25% contained valerian. Mono-ingestions with FS generally caused none or minor symptoms. In the Energizer category however, moderate to serious toxicity occurred in 9% of the mono-ingestions. Typically sympathomimetic overstimulation with gastrointestinal disturbances, restlessness, tachycardia, palpitations and sometimes hypertension, occurred within minutes to hours after taking an overdose of an Energizer. In some cases overstimulation was seen even after using the dose indicated on the package. In 10 cases (all Energizers), samples were retrieved and analyzed by the laboratory of the National Institute for Public Health and the Environment. The pharmaceuticals detected were mainly amphetamine-derivatives but also sibutramine and fluoxetine. Several FS were taken off the market, but sales from abroad via the internet were difficult to regulate.
Conclusins: Our PC received an increasing number of inquiries about FS. Most exposures to FS caused none or mild clinical symptoms. Products from the Energizer category seem to pose most health risks. Recording intended use and identifying products with a high health risk, helps to improve information supply to future callers. Collaboration with laboratories for chemical analysis and with public health authorities for regulation helps to protect unwitting consumers from harmful FS.

	2015	2014	2013
Relaxing	474	406	422
Energizing	147	129	138
Aphrodisiacs	24	23	20
Other	77	48	33
Total	722	606	613

KEYWORDS Dietary/Food supplements; health risks; poisons information

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22. Cordyceps poisoning due to consumption of "cicada flower" in Vietnam: Case-series report

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Background: "Cordyceps" in Chinese traditional medicine means "winter bug summer herb" or "cicada flower". It is an antlered fungus that grows in insect larvae, usually before the insect's cocoon is formed. It is gathered in the early summer. This fungus is found in some countries in Asia, especially in China, and it has been used in cancer treatment, to strengthen the immune system and improve renal function. Cicada flowers have been found in the Vietnam delta, and we report cordyceps poisoning after consumption of cicada flowers.
Case Reports: Between May 2008 and March 2015, 60 cases of cordyceps poisoning were admitted to 7 hospitals in the South Vietnam. Signs and symptoms occurred within 30-60 minutes of consuming" cicada flower", and included dizziness, vomiting, salivation, sweating, miosis, delirium, somnolence, stiff jaw, constipation, urine retention, seizures and coma. The severity of poisoning depended on the number of "cicada flowers" consumed. All routine laboratory investigations were normal. All the patients were required only supportive management during their hospitalization, and their signs and symptoms gradually resolved over two weeks.
Case Discussion: Poisoning occurs because live cicada nymphs cannot be distinguished from "cicada flower" (i.e., cicada nymphs' bodies infected by cordyceps fungus). The spores of the fungus attached themselves to the external surface of the cicada nymph bodies where they germinate in the anaerobic underground environment for many years during the development of the cicada nymph. The fungus directly invades and penetrates the exoskeleton of the cicada nymph. By the time the insect is dead, its entire body is full of fungal mycelium. The fruiting bodies of the fungus sprout from the cicada nymph's head in the aerobic environment. When people consume fungus infected cicada nymphs, they are consuming mostly fungus. The species of Cordyceps is a parasite on cicada nymphs in Vietnam, it is similar to the Ophiocordyceps heteropoda and its toxin is confirmed that ibotenic acid was positive, muscarine and muscimol were negative in the sample of cicada flower.
Conclusions: The presenting signs and symptoms of our patients, the first case series of human cordyceps poisoning, appeared similar to ibotenic or muscarine syndrome.

KEYWORDS Cordyceps fungi poisoning; Cicada flower; ibotenic acid

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23. Venom Dose Response in Genetically Related Multicasualty Massive Bee Envenomation

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Background: The ubiquitous Western (European) honey bee (Apis mellifera) and its many subspecies have been partners in food production for thousands of years. While agriculturally used subspecies are docile, the breeding of a hybrid bee crossed with A.p. scutellata produced the reviled "Africanized" bee prevalent in the wilds of the Southern United States and throughout Mexico and Latin America. This hybrid is known to swarm more frequently and to respond to threats more quickly with greater numbers. The result is more frequent massive envenomation than with more docile subspecies.
Case Report: We present the results of massive envenomation in which two human adult sisters (P1 and P2) and one of their daughters (P3) were stung by a single swarm. As a multi-victim massive envenomation with a high degree of relatedness and 40 years of age difference, this case provides insight into dose response and age response relationships of apitoxin with minimum genetic confounders.
Case Discussion: Apitoxin is a complex mixture of proteins with various effects. Melittin, a protein kinase C inhibitor, and phospholipase A2 are the main constituents and are thought to result in cell lysis leading to rhabdomyolysis, transaminitis, cardiac myocyte injury, and renal dysfunction among others. Since all stings were delivered from a single swarm, between 25–75% relatedness in the bees is expected so large variations in venom characteristics are not anticipated.
Conclusions: The characteristics of P1 and P2 allow us to correlate dose response while those of P1 and P3 allow us to correlate age response. As shown in the tables, creatinine kinase (CK) was elevated in a dose-dependent manner. Troponin was elevated in a non-dose dependent manner but did appear to have an age correspondence. Transaminitis was markedly elevated in P1 compared to P3 and likely reflects both age and dose correlation. Significant hepatic synthetic dysfunction was not seen despite transaminitis. No renal dysfunction was demonstrated. The patient's genetic similarity (mother-daughter-aunt) and age range allow us to correlate results to stings/kg and thus venom load. CK elevation was closely associated with apitoxin dosing. Transaminitis and cardiac injury were less related to apitoxin dosing and may reflect either anaphylaxis hypotension (in P1) or age factors in addition to apitoxin's direct effects.

Patient Characteristics

Patient	Age (y)	Weight (kg)	Stings(counted)	Stings/Kg
1	51	81	750*	10.4
2 (sister of 1)	47	72	561	6.4
3 (daughter of 2)	11	39	364	9.4

*500 stings counted excluding back and buttocks. Estimated at 750 including uncounted area

Note: P1'scare complicated by anaphylaxis, intubation, subsequent bacterial pneumonia. P2's and P3's care was supportive with IV hydration only.

Peak labs and timing

Patient	CK (IU/L)	Trop (ng/mL)	AST (1 U/L)	INK
1	4913@39h	0.77@15h	1288@11h	1.3@21h
2	1153@20h	0.08@7h	65@14h	1.2@20h
3	7660@47h	0.04@11h	241@47h	1.3@12h

SCr ware not elevated. ALT ~1/3 AST. Patient 3 INR was a single value.

KEYWORDS Bee; Massive; Envenomation

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24. Homemade Play-Dough: Money Saving and Potentially Life Threatening?

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Background/Objective: Recipes for homemade play-doughs can be found online. The majority of homemade play-dough recipes require varying amounts of table salt. Approximately 0.5 to 1 mg/kg of table salt (sodium chloride) is considered potentially toxic in a child. One tablespoon of sodium chloride contains 305 mEq of sodium, and can raise a 15 kg child's serum sodium by 30.5 mEq/L. The objective of this study is to compare the sodium content per tablespoon of dough for three homemade play-dough recipes.
Methods: We identified three different recipes for homemade play-dough online. The recipes were prepared on the same day using the same standard kitchen measuring cups. Recipe A required 1/3 cup of table salt; recipe B required 1/2 cup of table salt; recipe C required 1 and 1/2 cup of table salt. Each dough was weighed on a digital laboratory balance. The amount of table salt per tablespoon of dough was calculated using the following formula:

$$(\text{weight of tablet salt added})/(\text{total weight of the dough preparation}) = \%\ \text{of NaCl}$$

$$(17.85\ \text{g NaCl})/(1\ \text{tablespoon}) \times \%\ \text{NaCl} = (\text{grams of salt})/(1\ \text{tablespoon of dough})$$

Results: One tablespoon of dough from recipe B and C would put a 20 pound child in the toxic range for sodium chloride ingestion (4.54 to 9.1 grams) and potentially raise the child's serum sodium by 10 to 20 mEq.
Conclusions: One tablespoon of homemade play-dough may contain a potentially toxic amount of sodium chloride. Caution should be exercised when letting small children play with homemade play-dough.

KEYWORDS Sodium Chloride; Table Salt; Play-dough

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Table 1. Calculations of table salt per tablespoon of dough

	Amount of Table Salt Required (Cups)	Weight of Table Salt (grams)	Weight of the Final Product (grams)	Percentage of Dough Estimated to be Table Salt	Table Salt per Tablespoon oF Dough (grams)
Recipe A	1/3 Cup	101.5 g	471 g	22%	3.93 g
Recipe B	1/2 Cup	163.3 g	512.5 g	32%	5.71 g
Recipe C	1 and 1/2 Cup	474.1 g	1589.1 g	30%	5.36 g

25. Clinical effects of kratom use: a poison center observational study

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Background: Kratom, Mitragyna speciosa, has been used for centuries by the indigenous people of Southeast Asia. The active ingredient mitragynine, is an alkaloid which reportedly has stimulant and opioid properties. Given the current opioid epidemic recreational use may be increasing. We performed a review of cases at a single poison control center to summarize the clinical effects of kratom.
Methods: A retrospective chart review of poison center cases between January 2002 and January 2016 coded as kratom was performed. Each chart was abstracted for clinical data including: reason for use, vital signs, documented laboratory abnormalities, signs and symptoms, co-ingestants, co-morbidities, daily medications and final disposition.
Results: There were ten cases reported during the study period, none prior to 2010. The most common clinical findings included confusion (n = 5), sedation (n = 4), altered mental status (n = 4), seizures (n = 4), nausea (n = 3), and agitation (n = 3). Intended use of kratom was unclear in most cases and included recreational use (n = 7), self-treatment for opioid withdrawal (n = 1), and suicidality (n = 2). One patient consuming kratom for five weeks had elevated transaminases with a total bilirubin of 4.2, a rarely reported effect. One patient with concomitant cocaine exposure presented with seizures, sympathomimetic vital signs and was intubated. Seven patients were hospitalized: 4 to a general medical floor, 1 to an intensive care unit, and 2 to a psychiatry floor. There were no deaths reported.
Conclusions: Clinical effects from kratom exposure are diverse. Central nervous system effects were common. Seizures have been reported previously, although this series had a high prevalence. Limitations include missing data, co-morbidities, and potential co-ingestants.

KEYWORDS Kratom; seizures; central nervous system

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26. Use of Polyvalent Equine Anti-Viper Serum to Treat Delayed Coagulopathy in Two Children

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Background: The Western Pygmy Rattlesnake (Sistrurus miliarius streckeri) is commonly encountered in eastern Oklahoma, and reportedly has refractory delayed coagulopathy when treated with CroFab® (Crotalidae Polyvalent Immune Fab). We report two children where polyvalent equine anti-viper serum (AntivipmynTRI®) was administered to two children to treat recurrent coagulopathy after a Western Pigmy (WPR) envenomation.
Case Reports: Case 1 - A 16 month old male was bitten by a confirmed WPR. The patient received a total of 18 vials of CroFab®. His labs normalized, swelling gradually improved, and the child was discharged to home. On day 5, the child still had a great deal of inguinal tenderness. Labs were obtained and the child's INR was >13.1, while the fibrinogen was <60 mg/dL and the d-dimer was 11.72 mg/L. A decision was made to administer Antivipmyn TRI®, and the child received a total of 10 vials. Lab values significantly improved: INR 1.2, fibrinogen 93 mg/dL, and d-dimer 4.21 mg/L. Five days after the final dose of Antivipmyn TRI® the parent called with complaints of an urticarial rash with pruritis, which was treated with oral prednisolone and diphenhydramine over three days without recurrence. Case 2 - A 20 month old male presented following a suspected snake envenomation. Based on the clinical scenario, WPR was strongly suspected. Moderate envenomation protocol was initiated and the child was administered a total of 22 vials of CroFab® over approximately 70 hours following envenomation. Physical exam continued to improve, however lab results showed an increasing INR 1.98, decreasing platelet count 124 × 103 per μL, fibrinogen <60 mg/dL, and D-dimer >20 ug/mL. A total of fifteen vials of Antivipmyn TRI® were given to this patient. Following this administration, labs and clinical exam both significantly improved. Labs revealed INR 1.16, fibrinogen 110 mg/dL, d-dimer 3.2 ug/mL and platelet count 215 × 103 per μL.
Case Discussion: CroFab® is still the first line treatment for children bitten by a WPR but in some cases recurrent coagulopathy that is refractory to further AV administration has been frustrating for clinicians. To date, no specific approach to the problem has been proposed. Based on in vitro neutralization studies, Antivipmyn TRI® may be effective against WPR venom. This information led us to try this AV for these recurrences. The rapid response, which is atypical in recurrences to WPR's treated with CroFab®, lead us to conclude that this is a potential therapy for this situation.
Conclusions: Recurrent coagulopathy and thrombocytopenia may be problematic in children treated with CroFab® after a WPR bite. In these two cases, recurrent defibrination promptly reversed when subsequently treated with Antivipmyn TRI®.

KEYWORDS Western pigmy rattlesnake envenomation; delayed coagulopathy; polyvalent equine anti-viper serum

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27. Shiitake dermatitis – a case of food poisoning from a common edible mushroom

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Background: Shiitake dermatitis, or flagellate dermatitis, was first described in Asia in 1977. More cases of this toxicoderma have been reported in the United States in recent years. Shiitake dermatitis is an acute toxic response to lentinan, a polysaccharide found in raw or lightly cooked Shiitake mushrooms. The reaction appears to affect only a small percentage (about 2%) of those exposed to lentinan, and it is not considered an allergy. We document the first reported case of Shiitake dermatitis in our state.
Case Report: On December 23, 2015, a man in his 40s called the state health department and reported that he suspected he had Shiitake dermatitis. The man had been seen by medical professionals in urgent care and dermatology for his symptoms, which included a feverish sensation and a blistery rash on his upper body extending to his face, back, arms, and hands (photos provided). The rash was particularly itchy and painful on his hands and face, and had a streaky appearance in some areas. Both healthcare providers suspected a poison ivy rash; however, he had not had contact with woody or brushy areas and had not traveled. The patient was prescribed prednisone (which he did not take) and topical triamcinolone which helped relieve itching.

About 40% of the rash remained 10 days later, and fully resolved in 4 weeks. After researching his symptoms online, the patient recalled eating a stir fry dish with Shiitake mushrooms at an Asian chain restaurant 51 hours prior to his symptom onset. Environmental health specialists visited the restaurant and reviewed stir fry cooking practices. Stir fry dishes usually reached 200° F, but temperatures were not routinely taken.
Case Discussion: Shiitake dermatitis is rare and not well-known to the medical, public health, or poison center communities. This patient was able to identify the cause of his symptoms through astute online research, and not through visits to multiple medical professionals. The reaction causes a characteristic flagellate dermatitis, but could be misdiagnosed or considered idiopathic because of the rarity of Shiitake dermatitis. Lentinan is thermolabile and inactivated by thorough cooking. However, there are no specific recommendations for cooking of edible mushrooms, and the "safe" cook temperature is not known.
Conclusions: With the popularity of Asian cuisine, which commonly includes Shiitakes, and the growing raw and natural food movements, physicians, poison centers, and public health professionals should be aware of Shiitake dermatitis. Those who react to lentinan should avoid Shiitakes or at least ensure that they are thoroughly cooked before consumption. Others wishing to reduce the risk of Shiitake dermatitis can thoroughly cook these mushrooms.

KEYWORDS Shiitake mushrooms; Flagellate dermatitis; Lentinan

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28. Spontaneous intraparenchymal cerebral hemorrhage in a patient taking Mirtragyna speciosa (Kratom)

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Background: Mirtragyna speciosa is a pharmacologically active plant native to Southeast Asia. Recently, its use spread throughout the world. Preparations of concentrated extracts of the plant are sold as dietary supplements, incense, or legal "opioid" alternatives. They are marketed as "Kratom", "Krypton", and others. M. speciosa contains at least 20 alkaloids with mitragynine being primarily responsible for its effects. Over the last five years, case reports have been published describing adverse effects associated with these products. Reported adverse effects include seizures, intrahepatic cholestasis, nausea, vomiting, diarrhea, and even death. There are no prior reports associating its use with intracranial hemorrhage.
Case Report: A 26-year-old man presented to an emergency department (ED) after being found unconscious by his mother. There were no signs of trauma but he did vomit. He was in remission from intravenous heroin abuse. His mother reported that he received a package from another country containing "Kratom" three days before. On examination, there was a left facial droop and left-sided hemiparesis. A CT scan of the head showed a "large right frontal lobe, parietal lobe, and basal ganglia intraparenchymal hemorrhage associated with intraventricular hemorrhage in the right lateral ventricle, foramen of Monroe, third ventricle, and fourth ventricle." He was transferred to an academic hospital for further management. A thorough evaluation of the patient did not find another cause for his hemorrhage. His evaluation included a normal CT angiogram of the head and neck. A small arteriovenous malformation was found on an interventional carotid and vertebral angiogram. However, it did not abut the area of hemorrhage and there was no associated aneurysm. An echocardiogram demonstrated a small patent foramen ovale. A urine drug screen was negative for drugs of abuse. A urine sample was sent to Medtox Laboratories, Inc. and liquid chromatography/tandem mass spectrometry revealed a positive qualitative test for 7-hydroxymirtragynine and a confirmatory quantitative mitragynine level of >500 ng/mL.
Case Discussion: While this patient did have a history of IV heroin abuse, his urine drug screen sent over 6 hours after his presentation to the first ED was negative. His workup for a cause of the bleed was thorough and did not demonstrate the etiology of his hemorrhagic stroke.
Conclusions: This case describes a patient with M.speciosa (Kratom) associated cerebral hemorrhage.

KEYWORDS Adverse effects; kratom; cerebral hemorrhage

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29. Loxoscelophobia: Characteristics of Brown Recluse Spider Bites Called To A Regional Poison Control Center

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Background: Bites from the brown recluse spider (BRS), Loxosceles reclusa, can cause a wide variety of symptoms, from local pain and swelling to life-threatening systemic effects. Though case reports attribute significant morbidity and occasional mortality to BRS bites, there is a paucity of other literature detailing the characteristics and outcomes of BRS bites. We sought to analyze a regional poison control center's database of reported BRS bites to better characterize this exposure and compare reported BRS bites to other spider bites reported to the poison control center (PCC).
Methods: A retrospective review of the database of a regional PCC located in a BRS endemic area was performed from 1/1/2011 to 12/31/2015 for all spider bite cases. From these cases the following data was collected: age, sex, type of spider coded by PCC, month of the year, location of the bite, caller indentification of spider, whether the alleged spider was seen, antibiotic treatment, emergency department (ED) visits, admission to hospital, and ICU admission. Those with BRS spider bite were compared to those with other or unknown spider bites. Statistical significance was calculated using Fisher exact test and t-test where appropriate.
Results: Five hundred sixty-four cases were identified. Of these cases, 185 (33%) were coded as brown recluse bites (BRS group). In 127 (69%) of the BRS cases the callers volunteered that they had been bitten by a BRS but only reported seeing the spider in 57 of these cases. Overall, 60 (32%) of BRS cases reported actually seeing the spider that bit them. There were 379 cases coded as other species of spider or as an unknown spider bite (Other group). The spider was seen in 143 (38%) of the these cases. Table 1 lists and compares the characteristics of the BRS group to the Other group.
Conclusions: In this retrospective PCC study, reported BRS bites were most common in June and were more likely to result in antibiotic use, ED visits, and hospitalization than other reported spider bites. However, the alleged spider was seen in only 32% of reported BRS bites. This study highlights the significant morbidity associated with reported BRS bites but also demonstrates the frequent lack of identification of the offending spider which makes interpretation of PCC spider bite data challenging.

KEYWORDS Spider Bite; Loxosceles; Poison Control Center

Table 1. Comparison of BRS bites vs Other Spider Bites

	BRS [11 = 185]	Other [11 = 379]
Mean Age (years) [range]	33 [0. 75-91]	31 [0. 33-94]
Male/Female	88/97	183/196
Month with most calls [n]	June [38)]	June[63] July [631
Most Common Location of Bite[n]	Lower extremity [59]'	Upper extremity [129]
%Antibiotics given Till	43% [79]*	12% [461*
% ED visit [n]	38.4% [71]*	19% [72]*
% Admitted [n]	13-5% [> 5]*	3.4% [l3f
Days admitted [range]	5 [1-21]*	2 [1-71*
%ICU admit [11]	3-8% [7]*	0.003% [1]*

*= statistically significant, p<0.05

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30. Myocarditis associated with enzyme-linked immunosorbent assay-confirmed Loxoceles reclusa envenomation

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Background: Envenomations by the brown recluse spider (BRS), Loxoceles reclusa, have been reported to cause toxicity ranging from local symptoms to severe systemic effects. Myocarditis has not previously been attributed to BRS envenomation. We present a case of myocarditis associated with an enzyme-linked immunosorbent assay (ELISA) confirmed BRS envenomation.
Case Report: A 16-year-old male presented to the emergency department with fatigue, myalgia and chest pain. Initial vital signs were: temperature 39.1 °C, pulse 143 bpm, blood pressure 136/77 mm Hg, and respiratory rate 18 with a 100% SpO2 on room air. Labs were significant for a hemoglobin of 14.9 g/dL and WBC count of 2.8 x 10 3/mm 3. Over the next 8 hours, his temperature rose to 39.5° C, tachycardia persisted and he became hypotensive despite 2 L of intravenous fluids. He was admitted and started on antibiotics for an infection of unknown etiology. On hospital day (HD) 1 he developed an erythrodermal rash and a swollen, tender left forearm. A BRS bite causing systemic loxoscelism was suspected. He was started on vasopressors for refractory hypotension. He developed hemolysis and was transfused to maintain his hemoglobin. He was started on methylprednisolone 1 mg/kg IV every 6 hours. Over HD 3-6 he remained hypotensive despite norepinephrine, dopamine and milrinone infusions. On HD 3 an echocardiogram demonstrated an ejection fraction (EF) of 55%. On HD 5, an EKG showed diffuse T-wave changes, his troponin peaked at 0.29 ng/ml (normal <0.05), and he developed evidence of pulmonary edema on chest radiograph. A cardiac MRI performed on the same day demonstrated myocarditis with an EF of 45%. IVIG therapy was initiated. Over HD 7-10 his hemolysis worsened with a hemoglobin nadir of 5.9 g/dL. Plasmapheresis was initiated for the refractory hemolysis. By HD 14 his hemoglobin improved to 9.5 g/dL as the hemolysis abated. A repeat echocardiogram showed an EF of 80%. His wound developed a necrotic center which healed by secondary intention. He was discharged on HD 20. A polyclonal ELISA for BRS venom on a swab sample from the wound taken on HD 4 signaled an estimated 0.093 nanograms, with control sample signal at background.
Case Discussion: Myocarditis has been reported with other spider envenomations, particularly from the Latrodectus species. To our knowledge, it has not previously been reported to occur with BRS envenomations. A previous animal study found BRS venom could cause myocardial damage but human reports are lacking. In this case, the myocarditis may have caused his initial tachycardia and hypotension and exacerbated the hemodynamic instability caused by the acute hemolytic anemia. Though the spider was not seen and a bite was not initially reported, the physical findings were typical for a BRS envenomation, it occurred in an endemic region and ELISA confirmation of venom at the wound site add credible evidence for a BRS bite.
Conclusions: This case demonstrates that myocarditis may be associated with BRS envenomations and lead to refractory tachycardia and hypotension. Medical toxicologists and other health care providers should be aware of this possible complication.

KEYWORDS Loxosceles; Myocarditis; Spider

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31. A Toxic Alcohol By Any Name?: Comparison of Laboratory Confirmed Ethylene Glycol and Methanol Exposures Reported To A Regional Poison Control Center

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Background: Ethylene glycol (EG) and methanol (MET) exposures are rare but can cause significant morbidity and mortality. Though frequently treated as similar, there is limited literature comparing the clinical characteristics of these two exposures, especially in cases confirmed with quantitative serum levels. We sought to describe and compare the clinical characteristics of EG and MET exposures confirmed with detectable serums levels reported to a statewide poison control center (PCC).
Methods: A retrospective review of a statewide PCC's database from 7/2005 to 7/2015 identified all EG/MET exposures coded as greater than a lick/taste with a detectable serum level who were evaluated at a health care facility. The following data was obtained: age, sex, month of exposure, exposed substance (EG, MET or both), reason for exposure, admission rate, duration of PCC follow up, initial EG/MET levels, serum pH, serum creatinine, anion gap, serum ethanol level, max anion gap, max osmolar gap, therapy performed (hemodialysis, fomepizole, ethanol), and death. Those with a positive EG serum level were compared to those with a positive MET level using Fisher's exact test and Student's t-test where appropriate.
Results: The search identified 75 cases, with 59 cases (79%) having only detectable EG levels and 15 cases (20%) having only detectable MET levels. There was one case with simultaneously positive EG and MET level; a reported methanol exposure found to have an EG level of 5 mg/dl and a MET level of 109 mg/dL. The average EG level was 126 mg/dl (range 5-834). The average detectable methanol level was 78 mg/dl (range 5–396). Table 1 compares the characteristics of the EG positive and MET positive groups. The only statistically significant difference was in the maximum anion gap reported. One death was reported; in the EG positive group who had an initial level of 266 mg/dL.
Conclusions: In this study of EG/MET exposures, EG exposures were more common than MET exposures but had similar demographics, laboratory findings and interventions. Continued studies are warranted to further characterize these uncommon exposures.

Table 1. Comparison of Group with Positive EG serum levels vs Positive MET serum levels

	EG positive (n = 59)	MET positive (n = 15)
Age (years) [range]	33 [1.6-71]	31 [1.2-66]
Sex (M/F)	37/22	11/4
Most Common Month of	December [8]	January[3]
Exposure [n]	April [8]	March [3]
% Intentional [n]	85% [50]	87% [13]
% Admitted [n]	97% [57]	87% [13]
Mean Duration of PCC follow-up (hrs) [range]	104 [2-396]	65 [6-206]
Mean initial pH [range]	7.28 [6.6-7.52]	7. 31 (7. 09-7. 52)
Mean initial creatinine (mg/dL)[range]	1.24 [0.3-4.9]	0. 98 [0. 62-1. 9]
Mean Max Anion gap [range]	20* [8-35]	14* [6-34]
Mean Max Osmolar gap [range]	38 [(-) 10-129]	38 [3-142]
% Fomepizole administered [n]	88% [52]	85% [11]
% Ethanol administered [n]	5% [3]	24% [3]
% Hemodialysis performed [n]	42% [25]	24% [3]

*=statistically significant, $p<0.05$

KEYWORDS Methanol; Ethylene Glycol; Poison Control Center

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32. A fatal case of mistaken identity: cardioactive steroid poisoning from herbal tea

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Background: Cardioactive steroid (CAS) poisoning in the United States is largely confined to overdose of the medication digoxin. Though uncommon, several native plants contain cardiac glycosides capable of causing severe toxicity following ingestion. We report a case of fatal poisoning following ingestion of a CAS-containing plant.
Case Report: A healthy 69 year-old woman presented to the emergency department (ED) with complaints of nausea, vomiting, and weakness. These symptoms began after drinking a homemade tea composed of leaves believed to be comfrey (Symphytum spp.). Her vital signs on arrival were: blood pressure, 153/76 mmHg; pulse rate, 30 BPM; and respiratory rate, 20 breaths/min. She appeared pale, with diaphoresis. An initial EKG showed junctional bradycardia (rate 34 BPM), and the patient's monitor showed occasional sinus pauses of up to 6 seconds. Laboratory studies were notable for hyperkalemia (serum potassium 6.6 mmol/L).
Shortly after arrival to the ED, the patient's blood pressure dropped to 75/40 mmHg with a pulse of 30-40 BPM. Intravenous fluids and atropine were administered with minimal response. Hyperkalemia was treated with insulin, glucose, and sodium bicarbonate, though repeat potassium level after these interventions was 8.2 mmol/L. Clinical deterioration progressed to cardiac arrest. The patient was intubated and cardiopulmonary resuscitation (CPR) initiated. Unintentional foxglove (Digitalis purpurea) poisoning was suspected after consultation with poison control, and the hospital's entire supply of digoxin-specific antibody (6 vials) was administered. Despite ongoing CPR and administration of atropine, epinephrine, magnesium, calcium, isoproterenol, dopamine, and transvenous pacing, return of spontaneous circulation was not achieved, and the patient expired. A serum digoxin level ultimately returned at 55 ng/mL (therapeutic range 0.5-2.0).
Case Discussion: Several plants, including foxglove, are known to possess cardiac glycosides. Foxglove leaves resemble comfrey leaves when the flowers are not in bloom. Several cases of unintentional foxglove poisoning are present in the literature, though only one death has been reported. This patient's serum digoxin level was markedly elevated, demonstrating the assay's ability to qualitatively assist in making the diagnosis of non-digoxin CAS exposure. Large doses of digoxin-specific antibody may be required for patients poisoned by plant-derived CAS. Unfortunately, the hospital did not have a sufficient supply of antibody.
Conclusions: Foraging for edible plants can lead to unintentional poisoning due to misidentification. This patient, seeking comfrey leaves for an herbal tea, brewed and ingested foxglove, with fatal results.

KEYWORDS Cardioactive steroid; Naturally occurring toxin; Accidental ingestion

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33. Severe Methemoglobinemia In An Infant Following The Use Of An Herbal Remedy

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Background: Tabebuia impetiginosa, commonly called pau d'arco, is a genus of tropical plants native to the rain forests of Central and South America. Pau d'arco has been touted as a treat option for bacterial infections, cancer, inflammatory diseases, and peptic ulcers.
Case Report: A 5-month-old infant was noted by his parents to be pale, and lethargic with cyanotic lips and rapid respirations. His primary care physician called 911 and transported the baby to the emergency department where he was emergently intubated. A POC lactate level was 4.0 and blood work sent. The baby was rapidly transferred to the PICU. Endotracheal tube position was corrected and mechanical ventilation initiated. The Phillips pulse oximeter read 89% with minimal variation and a good waveform. A left subclavian vein catheter was placed and the blood return was notably black. The parents were approached and revealed a normal birth and first 5 months of life. No vaccinations had been given. The baby was breast fed with some supplemental water feedings from city water that was charcoal filtered. They had given the baby some homogenized yellow squash. With direct questioning they admitted to giving the child a small amount of an herbal supplement capsule that they opened into the water bottle. On the night prior to admission they had given an entire capsule. The product was Yeast/Fungal Detox capsules from Nature's Sunshine and contained a variety of ingredients including pau d'arco (Tabebuia impetiginosa). The lab reported that the methemoglobin level was 58%. A single dose of 1 mg/kg of methylene blue was administered over 1 minute via the central line. Within 15 seconds the pulse oximeter reading plunged from 89% to 28% and then slowly rose to 100%. At 1 hour the methgb level was 11% and without further treatment was 1% at 4 hours post treatment. The infant was rapidly weaned from mechanical ventilation and was discharged in apparent normal condition.
Case Discussion: Pau d' Arco is a quinone compound. Similar compounds have been reported to cause methgb. We are unaware of previous reports of commercially available herbal supplements causing methgb. In this case, the methgb was sufficient to produce a lactic acidosis and tachypnea.
Conclusions: The lack of variation in pulse oximetry readings and abnormal appearance of venous blood should suggest methgb.

Practitioners should be cautioned regarding the dramatic effect of methylene blue on pulse oximetry prior to administration.

KEYWORDS Methemoglobin; natural products; methylene blue

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34. Case Report of a Child Requiring Extracorporeal Membrane Oxygenation after a Brown Recluse Bite

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Background: Systemic loxoscelism can lead to multiple complications including hemolysis, disseminated intravascular coagulation, and acute renal failure. We present a case of a pediatric patient having multiple cardiac arrests due to profound hyperkalemia secondary to massive hemolysis from envenomation by a brown recluse spider-Loxosceles reclusa. The patient was placed on extracorporeal membrane oxygenation cardiopulmonary resuscitation after his third cardiac arrest and continuous renal replacement therapy (CRRT) for hyperkalemia and acute renal failure.
Case Report: A 9-year-old male presented with fever, a diffuse rash, hematuria and a lesion consistent with a brown recluse spider bite on his left chest wall. Systemic loxocelism with hemolysis was suspected and intravascular methylprednisolone was initiated. The patient deteriorated and began to have hemodynamic instability, worsening lactic acidosis, and renal dysfunction. He demonstrated signs of disseminated intravascular coagulopathy and had four cardiac arrest events associated with severe hyperkalemia prompting the placement of ECMO. His clinical and laboratory status improved on 2 days of ECMO. He remained anuric for 2 weeks and he was placed on continuous renal replacement (CRRT) therapy until his acute renal failure improved. He was discharged on amlodipine for renin induced hypertension with a normal physical examination and normal renal function.
Case Discussion: Treatment for systemic loxoscelism is mainly supportive and includes aggressive hydration, serial complete cell blood counts, and urinalysis in severe cases. Many treatments have been proposed for systemic loxoscelism, including steroids, dapsone, and antibiotics; however, no treatment has been clinically proven in randomized trials to demonstrate efficacy.
Conclusions: Despite the initiation of corticosteroids and supportive care, we report the case of a patient who had four cardiac arrest events secondary to brown recluse envenomation prompting the placement of ECMO to protect his end organ perfusion.

KEYWORDS brown recluse; extracorporeal membrane oxygenation (ECMO); hemolysis

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35. "Current events": Hepatotoxicity Related to Ingestion of Silver Isolated from Homemade Electrolysis

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Background: Complementary and alternative medicine treatments are often used by patients in an attempt to treat a wide variety of medical conditions. Patients will often go to great lengths to find a "natural" cure for their ailments. Significant adverse effects may occur related to the use of these products.
Case Report: A 54-year-old man presented to the emergency department (ED) with complaints of cough, shortness of breath, and nausea and vomiting over the previous week. Initial vital signs included: BP, 106/79 mm Hg; HR, 147/min; RR, 32/min; T, 36.4 C (97 F); SpO2, 95% on 2L O2. The patient admitted to ingesting a homemade silver preparation for the preceding 5 weeks for purported general health benefits which he produced via a homemade electrolysis setup. He produced the solution by heating a mixture of sodium bicarbonate and distilled water then partially submerging 99% silver coins that were connected to external battery sources into the solution. He states he would ingest 1-2 ounces of the solution 3-times per week. He denied other medication use, including prescription and over-the-counter products. Initial serum silver concentration was 210 nmol/L (normal range 0-3 nmol/L). Initial laboratory investigations revealed: creatinine, 419 μmol/L (4.7 mg/dL); urea, 35.3 mmol/L (98.8 mg/dL); calcium, 1.30 mmol/L (5.2 mg/dL); ALT, 4459 U/L; AST, > 2600 U/L; ALP, 184 U/L; total bilirubin, 81 μmol/L (4.7 mg/dL); INR, 4.1. Hepatitis screen, autoimmune hepatitis markers, and serum acetaminophen concentrations were negative. Liver ultrasound was grossly normal showing mild increased hepatic hyperechogenicity. He was initiated on N-acetylcysteine for presumed non-APAP related hepatotoxicity, received vitamin K, intravenous (IV) fluid rehydration, and calcium replacement. Complicating the patient's presentation was an incidental finding of a large pericardial effusion on day 3 post-admission of which the fluid aspirate was positive for Streptococcus anginosus. He received adequate treatment with broad-spectrum IV antibiotics. He also had atrial flutter with rapid ventricular response that was refractory to IV β-blockers and calcium channel blockers and required delayed electrical cardioversion. Over the course of the patient's hospitalization, serial liver function tests slowly decreased and his hepatic and renal dysfunction corrected. At the time of discharge, he was asymptomatic with normal laboratory values.
Case Discussion: This patient experienced significant hepatotoxicity and hepatic dysfunction following ingestion of a homemade silver solution extracted through electrolysis. The absence of overt argyria in this patient may relate to the relatively short period of time over which the silver solution was consumed. The clinical scenario was complicated by the presence of atrial flutter and positive cultures from his pericardial effusion. However, both of these factors were felt to be unrelated to the patient's hepatotoxicity and hepatic failure.
Conclusions: Silver has a long history of use in healthcare and the food industry for its strong antimicrobial properties. Its use, typically as colloidal silver, has been touted by fringe groups to be beneficial for a number of different medical conditions. Health care practitioners must be vigilant to inquire about the use of alternative medicine in patients presenting with complex presentations.

KEYWORDS Silver; Hepatotoxicity; Alternative medicine

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36. Two Deaths from Ingestion of Pong-Pong Tree (Cerbera odollam) Seeds

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Background: Suicides after ingestion of seeds from the Cerebera odollam tree are common in Eastern Asian countries where this plant is indigenous. We present two deaths in the US from ingestion of Pong-Pong Tree seeds purchased via the Internet.
Case Reports: Case 1 - A 30-yr old schizophrenic male arrived 12 hours after intentional ingestion of an unknown amount of Pong-Pong Tree seeds. Presenting symptoms included vomiting, blurry vision, headache, and chest heaviness. Initial vitals were normal except a RR of 27. EKG showed atrial fibrillation with a rate of 60 bpm. Labs were remarkable for K + of 10.1 mmol/l, creatinine (Cr) of 2.8 mg/dl, and a serum digoxin concentration of 1.6 ng/ml. Initial therapy included hyperkalemia treatment and 2 vials of digoxin immune FAB fragments (DIF). Repeat labs in the ICU showed a K + of 7.4 mmol/l and Cr of 1.98 mg/dl. Further therapy included bicarbonate infusion and 3 hours of hemodialysis. At approximately 33 hours post ingestion, he became markedly bradycardic and hypotensive. He was treated with ACLS including transcutaneous pacing, isoproterenol infusion, and 5 vials of DIF. He progressed to asystole and died. Case 2 - A 22-yr old transgender depressed female arrived 7 hours after an intentional ingestion of one Pong-Pong Tree seed. Presenting symptoms included vomiting, diarrhea, chest pain and palpations. Initial vitals were normal except for BP 42/32. EKG showed an ectopic atrial tachycardia with 2:1 conduction block with a rate of 51 bpm. Labs were remarkable for K + of 5.2 meq/l, Cr of 1.0 mg/dl, and serum digoxin concentration of 1.3 ng/ml. Therapy included 10 vials of DIF. Approximately 1.5 hours after arrival (8.5 hours post ingestion), her HR dropped into the 30's and she became pulseless. Further therapy included ACLS, an additional 10 vials of DIF, and 20% lipid emulsion therapy. Despite ongoing aggressive resuscitative efforts for over 2 hours, she died.
Case Discussion: Cerbera odollam belongs to the Apocynacceae tree family. Indigenous to certain areas of India and Southeast Asia, the plant is commonly referred to as the Pong-Pong Tree or the Suicide Tree. The fruit kernels contain the cardiac glycoside cerberin. They are used for suicide in regions where this plant is indigenous, but only one other case in the US has been reported. Pong-Pong Tree seeds are easily obtained over the Internet in the US. Both of our patients presented with abnormalities associated with cardiac glycoside toxicity, and had detectable serum digoxin concentrations. Despite aggressive treatment, including DIF, both patients died.
Conclusions: Western practitioners should be aware of the availability and toxicity of the cardiac glycoside containing Pong-Pong Tree seeds, which can be easily obtained via the Internet, and may not respond to DIF.

KEYWORDS Pong Pong Tree; cardiac glycosides; cerberin

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37. Cardiac glycoside toxicity from ingestion of commercially available tincture of Convallaria majalis

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Background: Convallaria majalis (Lily of the valley) is a flowering woodland plant native to Asia and Europe. C. majalis contains over 30 cardiac glycosides, including convallatoxin, a potent inhibitor of Na-K-ATPase. We present a case of cardiac glycoside toxicity from chronic ingestion of a commercially available product containing tincture of C. majalis.
Case Report: An 86-year-old woman with a history of CAD, hypertension, and hyperlipidemia presented to the ED with one week of nausea, vomiting, abdominal pain and fatigue. Vital signs were blood pressure (BP) 106/53 mmHg, heart rate (HR) 54 beats per minute (bpm), respiratory rate 16 breaths/minute, SpO2 97%, temperature 98.4oF, blood glucose 166 mg/dL. Physical examination revealed normal mental status, bradycardia, and mild bilateral pitting edema of her lower extremities. Electrocardiogram showed a ventricular rhythm at a rate of 44 bpm and left bundle branch block. Laboratory results were significant for leukocytosis 17.6 x10^3/uL, hyperkalemia 7.2 mmol/L, BUN 53 mg/dL, Cr 3.8 mg/dL, lactate 4.5 mmol/L, and troponin $<$0.03 ng/mL. Despite fluid resuscitation, the cardiac monitor showed HR 42-75 bpm and BP 88-106/27-53 mmHg. Atropine 0.5mg was given without a clinical response. Review of her medications revealed the patient was taking an over-the-counter Polish herbal supplement Cardiol-C containing tinctures of C. majalis, valerian root, caffeine, and extract of hawthorn and cola nut. She was not prescribed digoxin. The patient reported taking approximately 25 drops of the solution twice a day for the past several months. Ten vials of digoxin immune FAB (DigiFab^{TM}) were given for suspected cardiac glycoside toxicity, which did not result in clinical improvement. A serum digoxin level drawn shortly after DigiFab^{TM} administration was 4.9 ng/mL. The next day a repeat digoxin level was 2.6 ng/mL and BUN and Cr improved to 40 mg/dL and 2.04 mg/dL. The patient was discharged in stable condition two days after presentation.
Case Discussion: Extracts of C. majalis are marketed as herbal remedies for "cardiac insufficiency." Convallatoxin is renally excreted and may accumulate in renal insufficiency resulting in cardiac glycoside toxicity. Laboratory tests specific for convallatoxin are not readily available, however convallatoxin cross-reacts with commercially available digoxin immunoassays, which may be used to confirm exposure. Convallatoxin is poorly bound by DigiFab^{TM} at normal therapeutic dosing. Our patient did not significantly improve after DigiFab^{TM} administration.
Conclusions: Overuse of tincture of C. majalis may result in cardiac glycoside toxicity in the setting of renal insufficiency. Digoxin immunoassay may be used to confirm exposure.

KEYWORDS Convallatoxin; lily of the valley; digoxin

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38. Ayahuasca Exposure: Descriptive Analysis of Calls to United States Poison Control Centers from 2005–2015

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Background: Ayahuasca is a hallucinogenic plant preparation which usually contains the vine Banisteriopsis caapi and the shrub Psychotria viridis. This tea originates from the Amazon Basin where it is used in religious ceremonies. Due to interest in these religious groups spreading as well as awareness of use of ayahuasca for therapeutic and recreational purposes, its use is increasing. Banisteriopsis caapi is rich in β-carbolines, especially harmine, tetrahydroharmine and harmaline, which have monoamine oxidase inhibiting (MAOI) activity. Psychotria viridis contains the 5HT 2A/2C/1A receptor agonist hallucinogen N,N-dimethyltryptamine (DMT). Usual desired effects include hallucination, dissociation, mood alteration and perception change. Undesired effects previously reported are nausea, vomiting, hypertension, and tachycardia.
Methods: All human exposure calls to the American Association of Poison Controls Centers' (AAPCC) National Poison Data System (NPDS) between September 1, 2005 and September 1, 2015 were collected. Cases were selected with the product codes 2330118, 3633355, 4798273, and 7238375. All exposure reasons and all subject ages including unknown were included. Data requested comprised exposure reason, exposure route, 3 digit ZIP code, clinical effects, treatments given, medical outcomes and fatality abstracts. Calls for information without a human exposure were excluded.
Results: Five hundred and thirty-eight exposures to ayahuasca botanical products were reported. The majority of the calls to poison control centers came from health care facilities (83%). The most common route of exposure was ingestion. Most subjects were men (81%). The mean age was 23. Almost all exposures were acute. Three hundred thirty-seven (63%) were reported to have a major or moderate clinical effect. The majority of effects resolved by 8 hours (78%). The most common clinical effects reported were hallucinations (35%), tachycardia (33%), agitation (33%), hypertension (16%), mydriasis (13%) and vomiting (6%). Benzodiazepines were commonly given (30%). There were 29 subjects in the series who required endotracheal intubation. Four subjects were reported to have had a cardiac arrest and 7 a respiratory arrest. Twelve subjects had a seizure. Reports of exposures called to poison centers increased during this period (Table 1). No fatalities were reported for the product codes above during the study time frame.
Conclusions: Ayahuasca exposures reported to poison centers increased during the time frame of this study. Most of the exposures reported to poison control centers were young people, more likely to be men and already in a health care facility. Life threatening cardiovascular and pulmonary complications can result from ingestion of these botanical products.

KEYWORDS Ayahuasca; poison control center; dimethyltryptamine

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Year	2006	2007	2008	2009	2010	2011	2012	2013	2014
Exposures Reported	8	31	16	50	50	74	101	86	69

39. Epidemiology of herb induced acute liver injury in Taiwan: a poison center-based study

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Background: Traditional Chinese medicines (TCM) and/or other herbs are frequently used worldwide. Their potential adverse effects however are frequently overlooked. Among the adverse effects of TCM and/or other herbs, herb induced liver injury (HILI) is of major concern because it can occasionally lead to hepatic failure. However, the epidemiology and diagnostic criteria of HILI remain uncertain. We herein studied the severity and risk factors of HILI in Taiwan.
Methods: From the database of the Taiwan National Poison Control Center (PCC-Taiwan), we identified potential cases of HILI between 1986 and 2014. The relevant medical records were then reviewed by two independent investigators. Acute liver injury was defined as ALT >2N (upper normal limit) or ALP >2N. Cases with liver injury caused by substances other than herbs were excluded. Logistic regression analysis was applied to compare the characteristics between severe and non-severe cases.
Results: A total of 105 HILI cases were eligible for analysis. Among them, 15 (14.3%) had mild effects, 53 (50.5%) had moderate effects, 20 (19.1%) were of severe effects and 17 (16.2%) died. Six (5.7%) cases presented with multi-organ injury on admission. In simple logistic regression analysis, age, abdominal fullness, acute kidney injury, coagulation disorder, hypoalbuminemia, serum creatinine and total bilirubin were significantly associated with the severity of HILI. Nevertheless, only age (OR 1.09, 95% CI 1.01-1.17) and acute kidney injury (OR 21.6, 95% CI 3.8-123.1) remained statistically significant in multivariate logistic regression analysis. Cases manifesting hepatic failure or multi-organ failure were exposed to the following herbs: Taxus Chinensis, C. ducloux-ian, Taraxacum, TCM containing arsenic, snake gallbladder, Caulis Tinosporae Sinensis, Melastoma candidum D., Caulis Polygoni Multiflori, Cassia occidentalis, Xanthium sibiricum and Java tea.
Conclusions: Herb induced liver injury is not common among cases reported to PCC-Taiwan. However, the incidence of HILI is likely to have been underestimated given that more than one-third of the reported cases had severe/fatal HILI. Prospective studies that employ better data collection system and diagnostic criteria are warranted to delineate the magnitude of HILI.

KEYWORDS Traditional Chinese medicine; herb induced liver injury; Poison Control Center

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40. Topical Magnesium Gel: It will take your breath away

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Background: Hypermagnesemia can cause paralysis, arrhythmias, and death. Magnesium toxicity is seen almost exclusively in iatrogenic cases or in patients with renal failure. We present a case of symptomatic hypermagnesemia from accidental ingestion of an

over-the-counter topical magnesium preparation in a patient with normal renal function.

Case Report: A 48-year-old female presented to the emergency department with profound weakness which developed 5 minutes after an accidental oral ingestion of magnesium gel. The gel was contained in a cup that was confused for water and consumed. Similar commercial products contain 225 milligrams of magnesium per milliliter. The amount ingested was estimated to be 12 grams of magnesium. Initial vitals signs were significant for a heart rate of 68 beats per minute, blood pressure of 130/77 mm Hg, respirations of 14 breaths per minute, and an oxygen saturation of 88%. Physical exam revealed an alert patient with profound generalized weakness. She exhibited little motor function beyond finger wiggling and responded to questions with single words. Deep tendon reflexes were absent. Labs revealed magnesium of 10.3 mg/dL, creatinine of 0.61 mg/dL, and an ionized calcium of 1.21 mmol/L. An electrocardiogram demonstrated normal sinus rhythm with QRS duration of 0.112 seconds and QTc of 0.466 seconds. The patient was given 3 grams of intravenous calcium gluconate and subsequently showed a dramatic improvement in respiratory effort and ability to speak. After 6 additional grams of calcium gluconate, the patient had a normal physical exam. Serum magnesium concentrations 5 hours and 7 hours after arrival were measured to be 5.1 mg/dL and 3.7 mg/dL, respectively. The patient was discharged the next day in her usual state of health.

Case Discussion: Magnesium is an essential ion in the body with a tightly controlled serum concentration. There have previously been case reports of symptomatic hypermagnesemia following oral ingestion of over-the-counter magnesium-containing products, but this is the first case describing a symptomatic ingestion of a highly concentrated topical magnesium product. These topical products are readily available online and marketed for the treatment of fibromyalgia. Treatment of hypermagnesemia includes intravenous hydration, calcium for muscular weakness, respiratory support, and dialysis (in the setting of renal failure or severe, refractory symptoms).

Conclusions: We report a case of profound weakness and hypoxemia from an accidental ingestion of a topical magnesium-containing gel product in an individual with normal renal function. This case demonstrates the danger of readily available, highly concentrated topical magnesium products.

KEYWORDS Magnesium; supplement; overdose

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41. Systemic loxoscelism with delayed onset of near compartment syndrome of the forearm

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Background: Brown recluse spider bites (BRS) have a variable presentation and severity of associated symptoms.

Case Report: A 10 y.o. male presented with fevers and forearm swelling 3 days after an unwitnessed spider bite. Exam revealed a painful, edematous left dorsal forearm with a 2mm violaceous lesion surrounded with blanched skin and erythema extending up the arm. The patient had decreased ROM without open wounds, drainage or fluctuance. He had fever, chills, malaise, decreased appetite and a diffuse, exanthematous rash consistent with Acute Generalized Exanthematous Pustulosis (AGEP). Pediatrician started cephalexin and prednisone. The patient was later seen in the ER for progression of symptoms and admitted for elevation, IV fluids and clindamycin. He was afebrile but tachycardic with a WBC of 22,800 and AST of 74; ALT of 213 U/L. Hgb, creatinine, and UA were normal. Toxicology was consulted. Fever developed (102.4) and a Coombs + hemolytic anemia; Hgb fell to 5.5 gm/dL on hospital day 3 requiring transfusion of 3 units pRBCs. High dose IV methylprednisolone (4mg/kg/day) was added. Within 2 days the Hgb stabilized at 8.9 gm/dL. His WBC spiked to 74,100 and peripheral smear was evaluated by Hematology who diagnosed leukemoid reaction from steroids. The left forearm edema was nearly resolved with elevation and he was discharged on hospital day 5. On post bite day 6, forearm pain and swelling significantly worsened and lesional blisters appeared. He was afebrile, with WBC trending down to 58,400 and stable Hgb 8.9 gm/dL. Patient was readmitted for IV vancomycin. Significant swelling, pain and erythema of the dorsal forearm and hand persisted with decreased ROM. MRI suggested diffuse cellulitis, myositis and fasciitis. Hand team was consulted and compartment pressures were measured under anesthesia as elevated but below fasciotomy threshold. The arm was strictly elevated and observed closely. Slowly, the swelling and pain resolved and the patient was discharged on post bite day 10 on linezolid. After 4 weeks, he has recovered full ROM and strength with a 2×3 cm dry eschar at the initial wound site that may require future repair.

Case Discussion: BRS envenomation can present with a dermonecrotic lesion, delayed hemolytic anemia, AGEP and skin desquamation. There is no currently approved antivenom in the USA. Leukemoid reactions to this degree are rare in loxoscelism. Delayed compartment syndrome after initial cutaneous symptom resolution also has not been described, and in this case nearly required surgical management.

Conclusions: BRS bites have a variable presentation and severity of associated symptoms. Knowledge of all possible systems affected allows thorough evaluation to optimize outcomes. We report a rare case of BRS bite associated with severe systemic loxoscelism, hemolytic anemia and delayed onset of near compartment syndrome of the forearm in a pediatric patient.

KEYWORDS Loxoscelism; Hemolysis; compartment syndrome

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42. Novel Sources of Lead Exposure: A Case of Severe Lead Poisoning from an Ingested Foreign Body

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Background: Childhood lead poisoning continues to be a major public health dilemma in the United States, with vulnerable groups of children being disproportionately affected.

Case Report: 1 year old male of Indian descent was identified on routine lead screening by his primary care physician to have CDC class V lead poisoning with a venous lead level of 97 mcg/dL. Further laboratory testing revealed a microcytic anemia with hemoglobin of 9.5 g/dL, MCV 68.2 fL in the setting of normal iron studies and an elevated Zinc Protoporphyrin. Abdominal radiographic findings revealed an irregularly shaped radiopaque foreign body in the pylorus. The patient was taken to the operating room for extraction of the foreign body, which revealed an $1.8\,cm \times 2.1\,cm \times 0.4\,cm$ metallic foreign body. The family identified this medallion as his 4-year-old sister's jewelry pendant depicting a Hindu deity that was purchased abroad in Northern

India. The pendant had been misplaced for approximately one month and the family recounted that during this time period, this patient had repeated non-bilious emesis after most meals with associated weight loss. The patient was started on parenteral chelation therapy with British Anti-Lewisite (BAL) and Calcium-Disodium Edetate. Approximately six months after serial cycles of oral chelation therapy with Dimercaptosuccinic Acid (DMSA) and D-Penicillamine, this patient's blood lead level is persistently elevated at 39 mcg/dL. Bioaccessibility analysis of the medallion was conducted by placing medallion in 50 mL of artificial gastric juice (0.2% NaCl 0.8% HCl) with serial rotation of sample. This analysis revealed 1.09 mg/mL lead in the aliquot containing the medallion with undetectable levels in the untreated gastric fluid.

Qualitative X-Ray Fluorescence of the medallion are tabulated (Heavy Metal Qualitative XRF Analysis)

Lead	155,000 ppm (15%),
Copper	530,000 ppm (53%)
Nickel	49,000 ppm (4.9%)
Arsenic	22,000 ppm (2.2%)
Antimony	12,000 ppm (1.2%)
Tin	3,000 ppm (0.3%)
Silver	1300 ppm (0.13%)

with the following elements below the detection level: cadmium, zinc, mercury, cobalt, molybdenum, uranium, thorium, iron, manganese.

Case Discussion: This patient was exposed to potentially fatal levels of lead poisoning had his elevated blood lead level and retained foreign body not been identified by routine lead screening by his primary care physician. Despite this recognition, this child is at significant risk for the deleterious neurologic health effects of lead poisoning. This ingested metallic jewelry contained approximately 250 times the allowable content of lead in children's metallic jewelry as per the U.S. Consumer Product Safety Commission enforcement guidelines and other heavy metals with potential deleterious health effects. This case also highlights the ubiquitous nature of lead in our global environment and the increased risk of lead exposure to novel sources of lead for vulnerable populations such as recent immigrants.

Conclusions: A 1-year-old was identified by routine lead screening to have CDC class V lead poisoning in the setting of swallowing imported children's metallic jewelry who despite treatment with chelation therapy and removal of the foreign body has had persistently elevated blood lead levels and is at risk of deleterious health effects.

KEYWORDS Lead Poisoning; Vulnerable Populations; Pediatrics

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43. Neonatal lead poisoning treated with chelation

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Background: In utero lead exposure may result in long-term neurologic disability. Screening during pregnancy identifies women with elevated blood lead levels (BLLs), which results in subsequent neonatal testing. Experience managing severe neonatal lead poisoning is limited and consensus guidelines for this age group do not exist. We present the case of a newborn with a markedly elevated BLL.

Case Report: A boy was born at 38.5 weeks gestation by vaginal delivery to a 23-year-old G3P2002 woman who admitted to consuming soil during pregnancy. The newborn exam was normal with APGAR scores of 9/9 and birth weight 3.57kg, length 50 cm (BSA 0.21 m2). On day of life (DOL) 1 his venous BLL was 78 μg/dL. Other infant laboratory studies: hemoglobin 17g/dL, MCV 90 fL, free erythrocyte protoporphyrin 197 ug/dL, transferrin saturation 15%. Routine chemistry was unremarkable. A maternal BLL obtained five months earlier was 10 ug/dL. Repeat maternal BLL on post-partum day 2 was 49 μg/dL. Repeat BLLs in the newborn were 78 μg/dL and 76 μg/dL on DOL 2 and 3. On DOL 3 the newborn was transferred to a level IV NICU for chelation therapy with CaNa2EDTA and succimer. Because CaNa2EDTA was not available initially, he received oral succimer 350mg/m2 via orogastric tube every 8 hours as monotherapy. Succimer was continued through DOL 6, during which time the BLL decreased from 76 μg/dL to 48 μg/dL (37% reduction). On DOL 6, CaNa2EDTA was added to the regimen at 1000mg/m2/day as a continuous IV infusion. Dual chelation therapy continued for five days at which time CaNa2EDTA was discontinued and succimer was continued to complete a 19-day course. BLL was 17 μg/dL on DOL 12 and 28 μg/dL on DOL 22. Chelation was well tolerated by the neonate.

Case Discussion: Current CDC and NYC DOH guidelines recommend use of 2 drug therapy for management of children with a BLL >70 μg/dL. The patient was initially treated with succimer alone because CaNa2EDTA was unavailable. Succimer monotherapy has been used to manage children with moderate to severe lead poisoning in resource poor nations with promising results and provided some benefit in this case. CaNa2EDTA has recently undergone a marked cost increase. As a result, the treating institution and 14 surrounding hospitals discontinued stocking it, causing a delay in its use, and placing the infant at risk for poor outcome.

Conclusions: Succimer monotherapy safely reduced the BLL in a newborn with severe intrauterine lead poisoning. When combined with CaNa2EDTA an improved reduction in BLL was achieved. Succimer alone may provide effective initial chelation therapy for neonatal plumbism if CaNa2EDTA is not available.

KEYWORDS neonate; lead; chelation

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44. Failure of chelation therapy in metallic mercury poisoning: a case report

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Background: Poisoning with intravenous (IV) metallic mercury is uncommon and the benefit of chelation is unclear. We describe a carefully phenotyped case of metallic mercury poisoning secondary to deliberate IV injection.

Case Report: A 54-year old man presented to hospital following self-injection of 4mL (54g) metallic mercury into his left femoral vein. His blood mercury concentration was 7980 nmol/L (reference range <50 nmol/L). Diffuse radio-dense mercury particles were present in his lungs and vasculature on chest and abdominal radiographs. A CT head scan and echocardiogram revealed mercury deposits in his brain and heart, respectively. The patient underwent two five-day courses of IV 2,3-dimercapto-1-

propanesulfonic acid (DMPS) chelation therapy (30 mg/kg/day). This was followed by a further five-day course of oral 2,3-dimercaptosuccinic acid (DMSA) (30 mg/kg/day). Chelation resulted in significant urinary mercury excretion (see below). He was discharged on day 26 post-exposure. The patient was re-admitted to hospital two months later with malaise, weight loss and anorexia. Blood tests now demonstrated acute kidney injury and a renal biopsy demonstrated membranous nephropathy with focal cortical acute tubular necrosis. Repeat blood mercury concentration was 13,854 nmol/L. Despite best supportive care he died on day 159.

Case Discussion: The use of chelating agents has been shown to increase urinary mercury excretion up to fivefold. In our case, urinary mercury excretion peaked at 128,596 nmol/day during the first course of DMPS chelation. By contrast, in chelation-free periods, the average urinary mercury excretion was 2500 nmol/day. A smaller increase in urinary mercury excretion was observed during the second DMPS chelation period. No significant increase in urinary mercury excretion was demonstrated following oral DMSA. Cumulative renal elimination during chelation therapy, however, resulted in removal of only 0.16% of the total body load of mercury (88mg versus total injected dose 54g) and the blood mercury concentration remained unchanged.

Conclusions: This case highlights that urinary mercury excretion may be increased by IV chelation therapy but the total amount removed may not affect the eventual outcome following large mercury exposures.

KEYWORDS Mercury; Heavy metal; Poisoning

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45. Life on A Farm: Accidental Ingestion of 35% Hydrogen Peroxide by a 15-year-old Boy

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Background: Hydrogen peroxide is a clear, colorless, oxidizing agent that is widely used in a number of household products, natural health applications, agricultural and industrial products. Most exposures involve common household-strength (3%) hydrogen peroxide with little to no morbidity. We describe a case of a 15-year-old male who presented to the emergency department (ED) after accidentally ingesting 35% hydrogen peroxide.

Case Report: A healthy 15-year-old boy presented to the ED following the accidental ingestion of 35% hydrogen peroxide used on a farm. He took a water bottle out of the refrigerator that was labeled "poison" mistaking it for water and took 1-2 swallows (30 cc). Immediately after ingestion he complained of abdominal bloating and had "white foam" in his mouth and sialorrea, followed by hyperemesis. On arrival to the emergency department, he was hemodynamically stable and in no respiratory distress. The oral cavity and oropharynx were slightly erythematous. Intravenous (IV) fluids and IV pantoprazole were initiated. Initial laboratory tests were normal. Chest and abdominal x-rays showed increased lung markings within the left lung consistent with atelectasis and intrahepatic biliary air with air within the region of the stomach wall. A computed tomography scan of the abdomen revealed diffuse wall edema/liquefactive necrosis and intramural air involving the stomach and duodenum, extensive portal venous gas within the stomach and duodenum and airspace disease within the left lower lobe. The patient was admitted to pediatric intensive care and had gradual improvement of symptoms over the next 24 hours. The patient was discharged from hospital on the third day without complications.

Case Discussion: Concentrated hydrogen peroxide causes toxicity via three main mechanisms: corrosive damage, oxygen gas formation and lipid peroxidation. Management of exposures depend on the severity of ingestion and include airway management, frequent monitoring, and diagnosis and therapy of associated complications. Both x-ray and computed tomography are useful for ruling out viscous perforation and air embolism. Endoscopy was not felt necessary in our patient because he had no evidence of gastric perforation and his clinical condition as well as serial x-ray imaging showed improvement.

Conclusions: The storage and use of 35% hydrogen peroxide for agricultural, industrial and natural health benefits results in an emerging source for more serious ingestions. Concentrated hydrogen peroxide can cause significant morbidity when accidently ingested, and needs to be treated with caution and stored appropriately. Extension of poison prevention to include the caustic alkalis used on farms should be considered.

KEYWORDS Hydrogen Peroxide; Farm; Caustic

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46. Get Ahead of Lead: A Lead Exposure in Pregnancy Outreach Project

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Background: Lead is a neurotoxin and is associated with lowered IQ in children. Children are commonly exposed to lead-based paints in older homes and may be exposed in utero as lead readily crosses the placenta. Philadelphia residents remain an at-risk population for lead toxicity. A high incidence of elevated whole-blood lead levels (WBLL), defined as $\geq$5 mcg/dL, were observed in 9.4% of children screened in 2014 (national average 4.2%). 9.1% of children screened at our teriary-care center had a WBLL $\geq$5 mcg/dL in a recent analysis. Secondary prevention and treatment are hindered when an elevated WBLL is discovered at the typical 9-12-month screening window. Maternal screening for lead exposure could give early warning to an unsafe home environment. However, routine WBLL screening is not recommended for pregnant women in the United States. We proposed an innovative outreach project aimed at screening pregnant women in a high-risk population for lead exposure, early identification of children at risk for lead toxicity, and guidance for primary prevention of early childhood lead exposure.

Methods: This outreach project was a three-phase lead education, screening and reduction program. Phase I included free WBLL screening, a home lead test kit, and educational material to all pregnant women residing in Philadelphia presenting to our emergency department and prenatal clinic. All pregnant women with WBLL $\geq$5 mcg/dL were enrolled into phase II which included monitoring, clinical guidance, and instruction for lead abatement. Children born to mothers in phase II were enrolled into phase III which included monitoring and clinical guidance.

Results: 495 patients were screened. The mean age was 25.3 years. 71.1% were Black, 18.6% were Hispanic, and 3.4% Asian. 39.4% reported a household income below the poverty line, 22.6% above and 38.0% were unable to answer. 70.3% noted receiving public assistance. 67.9% rented their home, 18.0% owned while 4.2% were either homeless or lived in a shelter. 34.1% stated they lived in a home built before 1979 while 37.2% after and 28.7% were unable to answer. No patient screened had a WBLL $\geq$5 mcg/dL. 2 patients had a detectable WBLL but were

<5 mcg/dL; one of which had two children currently in treatment for lead toxicity. No patient met criteria for enrollment into Phase II or III.
Conclusions: Screening pregnant women for lead exposure was not an effective strategy to prevent early childhood lead exposure. Pregnant women in this population had a low incidence of elevated WBLL despite living in a community with a known high incidence of childhood lead exposure.

KEYWORDS Lead Poisoning; Environmental exposure; Prenatal Screening

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47. Childhood Lead Screening in Tbilisi, Republic of Georgia

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Introduction: Lead exposure is an important public health problem that causes cumulative toxicity in multiple body systems. Children are particularly vulnerable to its neurotoxic effects and therapy consists of removal of the exposure source and good nutrition. Georgia is a country with a population of 3.7 million, a third of which lives in Tbilisi that contains new and old dwellings relatively concentrated in separate areas. There is no data on lead exposure or levels in children in Georgia. The aim of the study was to describe the blood lead level (BLL) in children living in Tbilisi.
Methods: Georgian National Center for Disease Control and Public Health (NCDC) conducted a cross-sectional pilot study between November 22 and December 31, 2015 using a convenience sample of children aged 2-5 years visiting the private, tertiary Iashvili Children Hospital in Tbilisi. Technical support was provided by Emory University and the American Clinic in Tbilisi. Institutional review board approval was obtained from the NCDC. Venous BLLs were measured using a LeadCare II analyzer provided by Magellan Inc. The blood draw was already being performed on the child during the visit. The home address was classified as belonging to the old or new part of the city based on a predesigned list. Descriptive statistics were calculated to determine the geometric mean, median, percentile and quartile values. All results were generated using SAS. BLLs that were below the detection limit of 3.3 mcg/dl were converted to 3.3 mcg/dl.
Results: 254 children were included. 58.7% were male. 70.5% of children were aged between 2 and 4, 24.4% were 4 years old and 5.1% were 5 years old. 24% were living in the old part of the city. The BLL geometric mean was 5.7 mcg/dl and the median was 3.9 mcg/dl. The range was 3.3 to 57.0 mcg/dl with a standard deviation of 5.3. 33% of BLLs were ≥5 mcg/dl, 9.5% ≥10 mcg/dl, 2.8% ≥20 mcg/dl and 0.4% ≥45 mcg/dl. The mean BLL was 7.6 mcg/dl in children living in the old part of Tbilisi. Log-rank test showed a statistically significant difference ($p = 0.0136$) in BLL for new and old regions. There was no statistically significant difference for gender or age group.
Conclusions: Based on this sample, the BLL in children is higher than the data from the USA. Although only 2.8% had a BLL ≥20 mcg/dl and 0.4% ≥45 mcg/dl, there is a need for public health action to eliminate any lead exposure in children and decrease the percentage with BLL ≥5 or 10 mcg/dl. The sample size is small and not random, the age of the dwelling is unknown and there was no subject data collected to inform about possible sources of exposure. Therefore our results are not generalizable. Further research should be performed at a population level with a focus on old dwellings and include an environmental component.

KEYWORDS Lead; children; Tbilisi

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48. Hot and Cold? - Yes or No?

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Background: There are thousands of temperature-related injuries (TRI) in the United States every year. The association between extremes of weather and toxicological exposures, however, are unknown. Certain associations, such as alcohol and hypothermia, are well described. But the burden of disease is poorly understood. The aim of this study is to describe the association between toxicological exposure and TRIs injuries treated in hospitals.
Methods: The Illinois Outpatient and Inpatient Hospital Databases (based on billing records) were queried for TRIs treated in emergency rooms and hospitals, respectively, in Illinois from 2011 to 2013. Patients who were Illinois residents and diagnosed with ICD-9 codes for cold related injuries (991.0-991.9) or heat related injuries (992.0 -992.9) were included. The cause of injury was collected for all cases.
Results: From 2011 to 2013, in Illinois, there were a total of 18,303 TRIs accounted for in the database. Of these, 3303 were cold-related injuries treated as inpatient, and 3426 treated in the emergency department; there were 1324 heat-related injuries treated inpatient and 10250 treated in the emergency department. The most common identified cause of TRI was environmental, and out of the 23 known causes listed, two were toxicological in nature, as listed in Table 1. Also shown in Table 1 are pertinent associated comorbidities. These results suggest alcohol abuse is associated with TRI and toxicological causes are more frequently associated with cold-related injuries requiring admission.
Conclusions: Toxicological causes of TRI are most commonly associated with cold-related injuries. Alcohol abuse was associated with the highest percentage of inpatient cold-related injuries. Drug abuse was associated with the next highest number of TRIs, cold-related injuries being the most common. This study illustrates high clinical association between cold-related injuries and drug and alcohol abuse.

	Cold-related injuries		Heat-related injuries	
Causes	Inpatient	Outpatient	Inpatient	Outpatient
Adverse drug effects	214 (6%)	34 (1%)	33 (2%)	54 (1%)
Poisoning	49 (1%)	13 (0%)	2 (0%)	7 (0%)
Total drug related causes	263 (8%)	47 (1%)	35 (3%)	61 (1%)
Drug abuse*	355 (11%)	122 (4%)	92 (7%)	121 (1%)
Alcohol abuse*	650 (20%)	501 (15%)	112 (8%)	213 (2%)

*Comorbidities, NOT causes

KEYWORDS temperature-related injury; poisoning; hospital

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49. Animosity in Bed – Olfactory and Neurologic Dysfunction and Antimony Exposure

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Background: Antimony (Sb) has been used cosmetically and medicinally for thousands of years, and is still used to treat leishmaniasis and schistosomiasis. Its toxic effects are similar to those of arsenic – local irritation and rashes, various gastrointestinal effects, cardiovascular dysfunction, as well as neuropathy. Industrially, it is used as a flame retardant, and can be found in air samples of shooting ranges and electric waste sites. Here we present a case of olfactory and neurological dysfunction associated with antimony exposure.
Case Report: A 42 year old right-handed female presented with myalgias, muscle stiffness and weakness, blurred vision, memory loss, daily headaches, neck pain, burning in her forearms, anxiety, metallic taste, and odor sensitivity. The odor sensitivity was such that within 30 minutes of exposure to intense aromas, she would develop headaches and visual obscuration with fortification spectra, generalized muscle tightness, leg weakness, and impaired memory. These symptoms began after sleeping on a new mattress for six months. These symptoms markedly improved after the mattress was removed. Testing of the mattress revealed elevated levels of antimony and arsenic. Mattress antimony level was 4.9 ppm while mattress cover was 1060 ppm (normal <1.0 ppm). Mattress arsenic level was less than 1.0 ppm while mattress cover was 3.0 ppm (normal <0.2 ppm). Biological samples from the patient before exposure demonstrated no antimony and after exposure confirmed elevated antimony levels (Table 1). Two months after the mattress was removed, physical exam showed palmar erythema and multiple neurological abnormalities. These included mental status exam, left ptosis, bilateral abductor digiti minimi signs, bilateral Hoffman reflexes, and absent smell (anosmia) by Quick Smell Identification Test.
Case Discussion: Exposure to antimony, while rare, can cause myriad toxic effects. Neurologic and chemosensory manifestations may be explained by antimony exposure.
Conclusions: Olfactory dysfunction has never been reported in association to antimony exposure, but these results suggest antimony may be a pathologic agent in chemosensory dysfunction. It also highlights the need to assess for this toxin in cases where the origin of complaints is unclear.

KEYWORDS Antimony; anosmia; mattress

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Sample Type	Patient Value	Normal Range
PRE-EXPOSURE LEVEL		
Hair	0.018 mcg/g	Less than 0. 050 mcg/g
POST-EXPOSURE LEVELS		
Hair	0.37 mcg/g	Less than 0. 050 mcg/g
Blood	3.2 mcg/L	Less than 5 mcg/L
Urine	1.6 mcg/L	Less than 1 mcg/L

50. Trends in Liquid Laundry Detergent Packet Exposures in Children Reported to US Poison Centers from 2012–2015

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Background: The safety of LDPs has received considerable attention, including a 2012 CDC report describing the health hazards associated with exposure and other reports describing >17,000 exposures in children aged <6 years reported to US poison centers from 2012-2013. The concerns highlighted by this attention led to a host of industry changes beginning in 2013 (e.g. opaque packaging, double-latching lids) aimed at reducing unintended LDP exposures. The objective of this abstract is to evaluate increases in pediatric LDP exposures in relation to LDP sales to understand the impact of these industry changes.
Methods: The National Poison Data System (NPDS) was searched for LDP exposures in children aged <6 years that occurred between 01 January 2012 and 31 December 2015. Sales data for LDP sales from 2012-2015 were obtained from the Nielsen Company (Nielsen Holding NV). Annual exposure rates were calculated for number of exposures per 1 million LDP capsules sold.
Results: 39,575 LDP exposures in children <6 years of age were reported from 2012 and 2015, with an overall increase in reported exposures of 50.3% during this time. 12.5 billion capsules were sold from 2012 to 2015, which increased 60.5% during this period. Rates of LDP exposure decreased annually each year from 2012 to 2015, with a 21.5% overall decrease in rate of exposure. In 2015, the rate of exposure was 2.96 per million capsules sold or 1 exposure per 337,837 million capsules sold.
Conclusions: While the overall number of exposures to LDPs in children aged <6 years increased annually, the overall rate of exposures adjusted for LDP market penetration decreased over the surveillance period. Though the timeline for implementation of safety measures varied by manufacturer, the data suggest that the safety activities implemented throughout 2013 may have reduced the risk of unintentional pediatric exposures. Continued surveillance is warranted to gauge the impact of ongoing industry safety interventions and implementation of new standards.

KEYWORDS Liquid laundry detergent packets; surveillance; national poison data system

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	2012	2013	2014	2019
NPDS Exposures	6,345	8,907	11,718	12,605
Sales (million capsules per year)	1,683.49	2,826.18	3,723.83	4,262.00
Exposures per Million Capsules Sold	3.77	3.19	3.15	2.96

51. Elevated Lead Levels after Chronic Ingestion of Pomegranate Powder for Teething

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Background: Traditional medicines have been identified as a culprit for lead poisoning by the Centers for Disease Control. The practice of rasa shastra in Ayurvedic medicine, a form of Indian alternative medicine, is particularly noteworthy as it involves combining metals such as lead with other herbs to treat various maladies. Routine lead screening provides the opportunity for intervention before sequelae from chronic exposure becomes apparent. We present a case of chronic oral exposure to pomegranate powder from India resulting in elevated lead levels necessitating chelation.
Case Report: A 12-month-old boy was found to have an elevated lead level after routine capillary screening. This was confirmed with a venous blood lead level of 49 mcg/dL, prompting chelation with succimer and home evaluation. He was asymptomatic at the time. Environmental investigation revealed the use of oral pomegranate powder purchased from India as a teething agent intermittently over the previous 10 months. The tested pomegranate powder was evaluated by the local health department laboratory and results confirmed it contained 7.9% lead. The child's lead decreased to 37.7 mcg/dL approximately one month post-chelation. Follow-up levels at 60 and 90 days post-chelation remained elevated at 35.5 mcg/dL and 36.5 mcg/dL, respectively, despite discontinuing use of the powder. The child has remained asymptomatic and was meeting developmental milestones at 15 month follow-up.
Case Discussion: Pomegranate containing products have been touted as treatment and prevention for many conditions, ranging from osteoarthritis to several types of cancer. Health claims emphasize pomegranate's anti-inflammatory, antioxidant and anti-carcinogenic properties. In this case, a traditional Indian medicine was used as a teething remedy. With the lack of regulatory oversight there is the potential for these medicines to contain harmful metals including lead but also mercury and arsenic. Chronic exposures to lead in children can result in impaired neurobehavioral development and decreased intelligence. Levels in this child have remained elevated several months after chelation. Given the child's age, long term developmental and neurological effects may not yet be evident.
Conclusions: Traditional medicines, particularly Ayurvedic medicines, may contain significantly high lead concentrations, and may be overlooked in evaluation for common environmental sources of lead. This includes pomegranate powder which may be used to treat teething. Testing may be required to confirm the exposure source. Child health practitioners should be aware of folk remedies for common childhood ailments that may result in inadvertent lead poisoning.

KEYWORDS Lead; Pediatric; Traditional Medicine

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52. Sedation after Suspected Hydrogen Sulfide Exposure in a Pediatric Patient Supported by Elevated Urine Thiosulfate Concentration

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Background: Reported cases of hydrogen sulfide exposure in the pediatric population are rare. Data regarding urine thiosulfate concentrations in the pediatric population after hydrogen sulfide exposure are limited.
Case Report: A 3 y.o. male presents to an outside hospital after being found down. A plumber was using sulfuric acid to unclog pipes in the patient's basement. After the plumber started working, the patient was found by his parents unconscious and cyanotic in an upstairs bathroom that smelled of "rotten eggs". EMS was called and described the patient as posturing and unresponsive, vital signs: HR-167, RR-22, 97% on 100% FIO2 non-rebreather mask, glucose of 256 mg/dL. The patient was stabilized at an outside hospital and transferred to a tertiary care facility. He received 2, 0.5 mg IV lorazepam during transport. The patient had no history of medication or substance ingestion. He had no past medical history. There was no history of trauma or seizure activity. No one else in the house was sick. No darkening of metal items in the house. Physical exam - T. 99 F, P-114, BP-122/80, RR-30, 100% on 100% FIO2 NRB. He was sedated and minimally responsive, miosis bilaterally, otherwise unremarkable exam. Initial data: Head CT-normal, EKG-normal sinus rhythm QRS-68 msec QTc 440 msec, CXR-normal, CMP-AST-66 U/L, dex-117 mg/dL otherwise normal, CBC-normal, VBG-pH-7.31, pCO2-48 mmgHG, pO2-59 mmHg, ASA-negative, ethanol-negative, methanol-negative, isopropanol-negative, acetone-negative, UDS-negative (amphetamine, barbiturates, benzodiazepines, cannabinoids, cocaine, opiates, PCP). The patient arrived to the tertiary care hospital 1.25 hours after presentation and was admitted to the ICU. He was weaned off of supplemental oxygen and remained hemodynamically stable and stable from a respiratory status. His mental status improved and he was back to his neurologic baseline within 12 hours. He was discharged within 24 hours of presentation. After the patient was taken to the hospital the windows in the house were opened and the "rotten egg" smell resolved. The county health department evaluated the house the day after the incident and did not detect hydrogen sulfide gas.
Send out labs on urine at presentation: Drug screen expanded (NMS labs, test code 1876U): lorazepam 650 ng/mL, otherwise negative. Thiosulfate urine (NMS labs, test code 4472U): thiosulfate 28 mcg/mL, thiosulfate 61 mg/g creatinine.
Case Discussion: Hydrogen sulfide can cause knockdown effects and a rotten egg smell that were both documented with this case. This patient had no other medical explanation for his presentation, thorough toxicology testing did not show an alternate etiology for his presentation. Whole blood sulfide was not an available test so urine thiosulfate was sent to determine etiology. The patient's thiosulfate concentrations were consistent with hydrogen sulfide exposure. Immediate environmental testing for hydrogen sulfide gas was not done. This patient improved rapidly with supportive care.
Conclusions: This case represents a suspected hydrogen sulfide exposure in a symptomatic patient with elevated urine thiosulfate concentration.

KEYWORDS Hydrogen Sulfide; Thiosulfate; Pediatric

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53. Once in a Prussian Blue Moon: Death from Thallotoxicosis

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Background: Thallium is an exceptionally toxic metal with myriad historical uses; it is rarely implicated in human poisonings in the 21st century. The exact mechanism of toxicity is unknown, but its similarity in size to potassium may contribute to its impact on multiple enzymatic processes. Symptoms typically begin with vague gastrointestinal symptoms followed by ascending painful, peripheral neuropathy, delirium, and ataxia. In severe cases, patients develop seizures, coma, and death. Alopecia is the classic manifestation of thallotoxicosis, but typically presents 10 days to 1 month after poisoning. Delays in diagnosis are common and exacerbate toxicity. We present a fatal case of thallotoxicosis where diagnosis was delayed.
Case Report: A 31-year-old female was admitted to the hospital with presumed hypertensive encephalopathy. Encephalopathy worsened over 7 days, and she was transferred to a large quaternary hospital. Upon arrival she was comatose. On hospital day (HD) 17, treating physicians discovered a urine thallium level of >800 mcg/L. The first blood level obtained was 268 mcg/L. The Poison Center recommended Prussian blue administration. Procurement of the antidote was difficult as neither the state's Department of Health, the Radiation Emergency Assistance Center/Training Site (REAC/TS), nor the Centers for Disease Control (CDC) were able to provide Prussian blue due to its status as an investigational new drug. CDC eventually released a limited supply while awaiting the sole manufacturer's response. On HD 19, a dose of seven grams of Prussian blue four times daily was initiated. Multi-dose activated charcoal (MDAC) and dialysis had been previously initiated. Over the next 12 days, Prussian blue was continued and the thallium concentration gradually decreased to 18 mcg/L. On HD 31, she showed no clinical improvement and care was withdrawn.
Case Discussion: Despite a characteristic set of clinical manifestations, diagnosis of thallotoxicosis is frequently delayed. Both MDAC and Prussian blue promote elimination by interrupting the enterohepatic circulation of thallium. Orally administered Prussian blue incorporates thallium into its chemical matrix in place of potassium. Case reports, animal studies, and expert opinion have suggested Prussian blue is an effective antidote – alone or in combination with MDAC. Dialysis for thallium elimination has not been studied. Emergently obtaining Prussian blue is currently difficult, potentially delaying management.
Conclusions: This case of severe thallium toxicity was managed with dialysis, MDAC, and Prussian blue. As a result of a delay to diagnosis and definitive treatment, the patient had no clinical improvement and expired. Clinicians should have a high clinical suspicion for the poison and undertake early, aggressive treatment when the diagnosis is confirmed.

KEYWORDS Thallium; Prussian blue; thallotoxicosis

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54. Occult occupational dermal exposure to methanol requiring dialysis

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Background: Methanol poisoning mostly occurs by ingestion, whereas inhalation or dermal exposures have rarely been published. We report a case of occult occupational methanol poisoning by dermal exposure.
Case Report: A 30-year-old male presented to the emergency department on a Friday at 9:30 PM with a cutaneous rash on his forearms, ataxia and mild confusion. He worked in a chemical plant using acetone and other hydrocarbons. There was no suicidal ideation or other ingestion history. His medication included clobazam, levatiracetam and phenytoin for a seizure disorder. Initial electrolytes, lactate and renal function were normal. His first blood gas was pH 7.32, PCO2 34 and HCO3 17. Anion gap was 16 and osmol gap 44. Salicylates, acetaminophen, ethanol and acetone were negative. Phenytoin concentration was therapeutic. Initial methanol concentration at 10:15 pm, at least 6h after work, returned at 41 mmol/L (131 mg/dL) and later reported at 47 mmol/L (150 mg/dL), both by mass spectroscopy in different laboratories. The patient repeatedly denied ingesting ethanol or any methanol-containing product in the previous 24h. The composition of the solvent was unavailable at the time. He received fomepizole, folic acid and dialysis was requested with a consultation to the nephrology team. The patient was dialyzed for 5 hours. Methanol concentrations were 31 mmol/L (99mg/dL) 13h before dialysis, 26 mmol/L pre-dialysis and 3.8mmol/L (12md/dL) post dialysis. The patient was discharged home without any sequelae. Information available when the plant reopened revealed that he was the only worker to dip electrical circuit pieces in a polyurethane solvent, using a solution of methanol 100% to clean up streaks. The room was poorly ventilated and the worker didn't always use protective equipment during his 8h workday and may have attempted removal of polyurethane with methanol on his arms prior to consulting. Public health was contacted to review workplace safety procedures.
Case Discussion: This case is the highest concentration of methanol published from non-ingestion exposure. Methanol false positives were considered and discussed with the biochemist on duty but deemed unlikely given the reproducibility with mass spectroscopy and chromatographic assays. Prolonged dermal absorption may have blunted the occurrence of metabolic acidosis by preventing rapid systemic absorption with saturation of methanol liver metabolism and respiratory elimination.
Conclusions: Significant systemic absorption of methanol can occur with dermal exposure in unsuspecting individuals. No laboratory interferences are known with current methanol assays. Worker education on the dermal toxicity of products used in their workplace might have prevented this exposure.

KEYWORDS Methanol; Dialysis; Dermal

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55. Too Hip to Quit: Explantation on ECMO to Treat Arthroprosthetic Cobaltism

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Background: An estimated 4.5 million people in the US have arthroprosthetic hips. Ceramics, polyethylene compounds, and metals are used in the manufacture of these implants. Metal-on-metal implants (MOM) are often chosen for durability and may incorporate chromium, cobalt, titanium, or other metals. MOM surfaces may erode over time and lead to systemic cobalt toxicity. This is especially true following MOM replacement of ceramic implants, which may leave debris behind that can accelerate metal wear. Removal of the MOM implants and metal chelators have been used successfully in previous cases of cobalt toxicity. We present a case of severe cobalt toxicity treated with surgical explantation during extracorporeal membranous oxygenation (ECMO) support.

Case Report: A 58-year-old female developed myxedema coma five months after surgery to replace a fractured ceramic implant with a cobalt-containing MOM implant. Following admission, she developed rapidly progressive cardiac and renal failure. On hospital day 4, she was found to have a serum cobalt concentration of 185 ng/mL. She was initially treated conservatively with thiamine and n-acetylcysteine with plans to explant the cobalt hip as soon as she was stabilized. However, her status deteriorated to the point of requiring continuous renal replacement therapy (CRRT) for worsening acidosis and anuria. Severe cardiogenic shock refractory to multiple vasopressors concurrently developed. She was then placed on veno-arterial (VA) ECMO. Hip explantation was encouraged by the regional poison center, as the patient was unlikely to benefit from chelation without removal of the source. Orthopedic surgery explanted the culprit prosthesis while the patient was supported on ECMO. Following explantation, her cardiovascular status improved dramatically and she was removed from ECMO on post-operative day 4. She was discharged to a long-term care facility with an ejection fraction of 55%. Although she remains ventilator and dialysis dependent, her mental status has also improved to the extent that she can make her needs known and follow commands.

Case Discussion: Several case series of arthroprosthetic cobaltism have demonstrated patient improvement after prosthesis explantation and chelation with unithiol. However, unithiol is currently unavailable in the United States. In this case, chelation with calcium disodium EDTA was thought to be of limited benefit while the implant remained. The treating team also deferred post-operative chelation, citing concerns that chelation may not be effective or could possibly interfere with ongoing dialysis. Despite her tenuous status at the time of surgery, dramatic improvement followed explantation.

Conclusions: Explantation of a cobalt-containing hip is feasible even when cobalt-induced heart failure is severe enough to necessitate ECMO. Such patients in extremis should not be considered beyond salvage.

KEYWORDS ECMO; Chelation; Cobalt

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56. Successful N-Acetylcysteine treatment in a severe Dimethylformamide intoxication

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Background: Dimethylformamide (DMF) (CAS #68-12-2) is a colourless solvent used in the production of films and textile fibers. Absorption via inhalation, dermal and oral exposure is significant. The human lethal dose is supposed to be 10 g. Systemic toxic effects include renal, respiratory, neurologic, cardiovascular, dermatologic and hematologic damages. Liver is the main target in both acute and chronic exposures. The damage is dose dependent and reversible. The main elimination route is through the urine.

Case Report: A 34 years old man was victim of an industrial accident. A DMF barrel spilled over causing 1st and 2nd second degree burns on his face, arms and legs, spread irritation at the trunk and important oedema around the oral and ocular regions. Ocular irritation, conjunctival hyperemia and photophobia were observed. Inhalation caused dyspnea, tachypnea, cough and bronchospasm that led to a venous pO2 drop to 23mmHg. Electrocardiogram, transthoracic and abdominal ultrasound, thorax and abdomen x-ray, an encephalic nuclear magnetic resonance and a gastroscopy were all negative except for a mild erythema of the gastric mucosa. The Poison Center suggested a prompt decontamination, forced diuresis, β2 agonists, steroids and hydration, and dermatologists instituted a topical therapy for the skin lesions. Urinal metabolite of DMF was 18 mcg/mL, opioids test was positive due to the use of levofloxacin and tramadol. Five days later he developed an acute hepatitis: blood tests revealed an increase of AST and ALT aminotransferases, bilirubin (7 mg/dL, then 9.7 mg/dL) and iron (250 mcg/dL). Ammonia levels, INR and alkaline phosphatase were still in range. The urinal metabolites quadrupled reaching 88 mcg/mL. During the next few days, the patient felt a foreign body feeling in his throat and his skin began peeling. Meanwhile, ammonia reached 116 mmol/L, pseudocholinesterasis and albumin dropped and awareness decreased. Considering the oxidative action of DMF, 900mg/kg of N-Acetylcysteine were administered in 72 hours to support liver function (Amanita Phalloides intoxication protocol) and procedures for a possible liver transplant were activated. Gradually the liver function improved, aminotranseferases lowered, bilirubin dropped to 5 mg/dl and ammonia to 90 mcg/dL, INR and platelets normalized. Renal function was preserved.

After 14 days since admission, the patient was dismissed in good general conditions.

Conclusions: DMF can provoke serious damage to multiple organs, especially the liver.

Although the causes are yet to be completely understood, an oxidative mechanism seems implicated. In the reported case, N-Acetylcysteine has probably contributed to a positive resolution.

KEYWORDS N-Acetylcysteine; Dimethylformamide; Industrial accident

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57. Four Corners Coordinated Poison Center Role in Animas River Contamination

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Background: On August 5, 2015, waste from the Gold King mine was spilled into Cement Creek in Colorado, which is a tributary creek for the Animas River. The Animas River flows into the San Juan River, with the contaminated water potentially impacting residents in four states (CO, NM, AZ, UT).
Methods: A coordinated effort was initiated to ensure that the public in all affected areas would receive timely and accurate consistent messaging and that all exposed individuals would receive appropriate diagnostic testing if determined necessary. Those involved in this partnership included the five poison centers (PC) serving the four states, the state health departments (HD) in each state, several local HDs, the Environmental Protection Agency, the Department of Environmental Quality, and ATSDR.
Results: The New Mexico HD was notified by the PC that two individuals had ingested water from the San Juan River three days after the spill, at the time when heavy metal contamination was possible. Metals that exceeded levels recommended for oral consumption were provided by ATSDR (Mn, Cd, Co, Pb, Al), and used to guide diagnostic recommendations. On August 12, the poison centers began creating daily combined situation reports containing total number of calls, type of calls, and public health updates for the next week, which were provided to all agencies. The final report contained an accumulated 21 calls from 3 states, 13 of them human exposures, with reported symptoms of skin irritation and nausea. One PC medical director attended two days of community town hall meetings to answer resident's concerns. Other PC activities included daily phone calls with lead agencies and participation in creation of FAQ releases.
Conclusions: Five poison centers found value in combining efforts to develop a consistent accurate message to residents affected by an environmental spill that impacted four states. The constant inter-agency cooperation enhanced the ability of all involved agencies to respond to this event, and to plan future monitoring.

KEYWORDS Heavy metals; mining waste; river contamination

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58. Survival After Self-Poisoning with Hydrogen Sulfide: Implications for Scene Safety

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Background: Hydrogen sulfide (H2S) is a toxic gas that is used in chemical suicide by mixing a strong acid and sulfur donor, such as calcium polysulfide (lime sulfur). Survival is rare. We present a case of a woman who attempted suicide via this method and survived, and the implications for her caregivers.
Case Report: An otherwise healthy 47-year-old woman attempted suicide by mixing 1 liter each of 9.5% hydrogen chloride and 28% calcium polysulfide in a bucket inside her car while parked in a public ramp. She was found outside her car by a passerby who placed her back inside the vehicle, closed the door, then activated 911. The outdoor temperature (temp) was -12oC; she was wearing only pajamas and a jacket. Rescuers without any respiratory protection removed her and noted a strong smell of rotten eggs. The patient arrived to the ED confused (GCS 14), incontinent of stool and urine with bite marks on her tongue. Her lungs were clear. Her skin was notable for cool extremities with ecchymosis in various stages of healing. Conjunctivitis was noted in both eyes with visual acuity later documented as 20/200. Vital signs were as follows: temp 35.5oC, pulse 132 bpm, blood pressure 94/76 mmHg. A head CT was unremarkable. Labs were as follows: serum lactate: 5.3 mmol/L, troponin 1.37 ng/mL, serum bicarbonate 22 mmol/L, anion gap 11, creatinine 0.8 mg/dL, total CK 12,924 IU/L, and venous blood gas: pH 7.35, pCO2 42 mmHg, pO2 28 mmHg. A urine drug screen (LC/GCMS) was negative for any drugs. The patient was treated with supportive care only. Twelve hours later mental status cleared; mini-mental status exam was normal. She had no sequelae other than a peripheral sciatic nerve lesion. Both paramedics who transported the patient had nasal irritation; their exams and vital signs were normal. H2S concentrations [H2S] at the scene were not taken until 1 hour after EMS arrival and were negative.
Case Discussion: H2S is a nasal irritant at 0.025 parts per million (ppm). Eye irritation occurs at 50-100 ppm; the IDLH is 100 ppm. [H2S] above 800 ppm are rapidly fatal. Experimental data suggests using household products [H2S] inside a car can reach >8,000 ppm, but fall to <5 ppm 3 minutes after the doors are opened. Our patient's duration of exposure was never clarified; she was amnestic to all events prior to arrival. Her trivial hypothermia suggests the time her car doors were open was short. The lack of injury to EMS in this case is also consistent with low [H2S] inside the vehicle.
Conclusions: This case of survival after self-poisoning with H2S in a personal vehicle with no meaningful injury to rescue workers suggests that risk to EMS is minimal if the vehicle doors are opened even for a short time. Though this is consistent with experimental data, more studies are needed to validate such an approach.

KEYWORDS Hydrogen Sulfide; Poisoning; Gas

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59. Polyacrylate/nanosilica causes pleural and pericardial effusion, and pulmonary fibrosis and granuloma in rats similar to those observed in exposed workers

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Background: Nanomaterials offer great benefit as well as potential damage to humans. Workers exposed to polyacrylate coatings

have pleural effusion, pericardial effusion, and pulmonary fibrosis and granuloma, which are thought to be related to the heavy exposure of nanomaterials in the coatings. The study aimed to determine if polyacrylate/silica nanoparticles cause similar toxicity in rats as observed in exposed workers.
Methods: Ninety male Wistar rats were randomly divided into five groups with 18 rats in each group. The groups included the saline control group, another control group of polyacrylate only, and low-, intermediate-, and high- dose groups of polyacrylate/nanosilica with concentrations of 3.125, 6.25, and 12.5mg/kg. Seventy-five rats for the 1- week study were terminated for scheduled necropsy at 24h, 3 days, and 7 days post- intratracheal instillation. The remaining 15 rats (3 males/group) had repeated ultrasound and chest computed tomography examinations in a 2-week study to observe the pleural and pericardial effusion and pulmonary toxicity.
Results: We found that polyacrylate/nanosilica resulted in pleural and pericardial effusions, where nanosilica was isolated and detected. Effusion occurred on days 3 and day 5 post-administration of nanocomposites in the 6.25, and 12.5mg/kg groups, gradually rose to its greatest extend on days 7-10, and then slowly decreased and disappeared on day 14. With an increase in polyacrylate/nanosilica concentrations, pleural effusion increased, as shown by ultrasonographic qualitative observations. Pulmonary fibrosis and granuloma were also observed in the high- dose polyacrylate/nanosilica group.
Conclusions: Our study shows that polyacrylate/nanosilica results in specific toxicity of pleural and pericardial effusion, as well as pulmonary fibrosis and granuloma, which are almost identical to results in reported patients. These results indicate that the nanocomposite may cause unusual toxicity compared to its micrometer-sized and bulk counterparts, which should be taken seriously in the development of nanoscience and nanotechnology.
The present study was funded by the National Natural Science Foundation of China (Grant No. 81172614 and Grant No. 81441089).

KEYWORDS Polyacrylate/nanosilica; pleural effusion; pulmonary fibrosis

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60. All that shines is not gold

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Background: Home use of mercury (Hg) can lead to toxicity by the unsuspecting public. Hg inhalation causes severe poisoning after large exposures. We describe a case of systemic poisoning via a massive inhalational exposure resulting in respiratory failure and ARDS secondary to chemical pneumonitis.
Case Report: A 56 year-old male presented with complaints of dyspnea, pain on inspiration, and shortness of breath. He admitted to using Hg to amalgamate gold and silver from computer parts. He was discharged home several days later after treatment with steroids, but returned the same day in acute respiratory failure with bilateral pulmonary infiltrates on CXR. He was intubated and therapy with dimercaptosuccinic acid (DMSA) was initiated. Initial spot serum Hg level sent from first admission was 380 ng/mL. After one day of chelation, serum Hg level was 152 ng/mL. He received DMSA for 5 days. 8 days after admission, he developed increasing oxygen requirements, a right tension pneumothorax, and was restarted on DMSA. Twelve days following readmission, he was transferred for specialized pulmonary care. Repeat serum Hg level was 73 ng/mL and 24-hour urine was >800 μg. His subsequent hospital course was complicated by recurrent pneumonia, multiple pneumothoraces, fungemia and intermittent ventilatory support. He received one week of high dose IV NAC. Chelation was continued for 7 weeks until serum Hg levels remained <25 ng/mL. He was discharged to rehabilitation 15 weeks after initial presentation in stable condition and without neurological deficits.
Case Discussion: This patient had severe pulmonary toxicity with prolonged acute lung injury and secondary sequelae with evidence of a large Hg body burden after inhalational Hg exposure. He had been heating Hg in an indoor potter's kiln for over 1 year. EPA evaluation of the home showed Hg air levels >50,000 ng/m3 and clean-up required destruction of the site and soil removal. After prolonged high dose chelation and antioxidant therapy, he was discharged to rehabilitation without gross evidence of neurologic injury.
Conclusions: Home Hg use to reclaim precious metals is a lucrative way to make money and may be more pervasive than we realize. We describe a case of a massive inhalational mercury exposure from precious metal reclamation, causing systemic mercury poisoning, precipitating ARDS and respiratory failure, and necessitating prolonged ventilator support with eventual recovery.

KEYWORDS Mercury; Pneumonitis; Chelation

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61. Severe alprazolam withdrawal treated with GABA agents and ketamine infusions

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Background: Benzodiazepine withdrawal is a life-threatening condition that manifests with hyperadrenergic, neurologic and psychiatric effects. The use of pure GABA agonists has been linked to incomplete treatment responses. Ketamine, a dissociative NMDA receptor antagonist agent, has been used successfully to treat severe alcohol withdrawal. Parenteral ketamine is becoming more common for treatment of agitated delirium. The use of Ketamine as an adjunctive agent in the pharmacologic management of agitation secondary to severe benzodiazepine withdrawal has not been previously reported.
Case Report: A 26-year-old man presented from jail for evaluation of both auditory and visual hallucinations, insomnia, diaphoresis, and chest pain that started the day prior to evaluation. His clinical history included alprazolam (reportedly 20-40 mg/day) and quetiapine abuse. The timing of his incarceration prior to ED arrival was unclear. He was initially evaluated by psychiatry and was cleared for discharge. While awaiting disposition, he became progressively more agitated and delirious despite receiving a total of 6 mg lorazepam within the preceeding 24 hours. He was placed in mechanical restraints and over the next 4.5 hours, was administered a total of 490 mg of diazepam and 8 mg alprazolam. He was then placed on a propofol infusion at 20 mcg/kg/min but remained agitated and verbal. 1.5 hours later, a ketamine infusion at 0.1 mg/kg/h was initiated and his agitation quickly subsided. He received a total of 23 hours of propofol and 45 hours of ketamine. He received 300 mg IV diazepam while on ketamine and propofol. Both physical restraints and propofol infusion were discontinued 21 hours after initiation of ketamine. He never required intubation. Ketamine was continued for an additional 24 hours

after restraints were removed and he was subsequently discharged on an oral taper of diazepam.
Case Discussion: Alprazolam is a commonly-abused triazolobenzodiazepine whose withdrawal can be difficult to treat with other benzodiazepines such as lorazepam and diazepam. We report the successful use of a combination of propofol and ketamine infusions to control severe agitation from alprazolam withdrawal while avoiding over-sedation and the need for mechanical ventilation.
Conclusions: Propofol infusion in combination with low-dose ketamine should be considered as adjunctive therapy in the management of severe alprazolam withdrawal.

KEYWORDS Alprazolam; Ketamine; Withdrawal

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62. Beer Potomania: Atypical Cause of Severe Hyponatremia in Older Alcoholics

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Objectives: Although hyponatremia is commonly reported in chronic alcoholic patients, the prevalence of severe hyponatremia and its underlying pathophysiologic mechanisms in elderly patients are not well delineated. The syndrome of "beer potomania" is used to describe a patient who presents with hyponatremia in conjunction with low daily solute intake and excessive beer drinking. The aim of this current study was to describe the epidemiology and clinical features of beer potomania in older alcoholics presenting to the emergency department (ED) with severe hyponatremia (<125 mEq/L).
Methods: This was a retrospective, cohort analysis of consecutive older adults (>64 years of age) presenting to the emergency departments (ED) of two university-affiliated hospitals with severe hyponatremia (<125 mEq/L). All eligible patients had a history of chronic alcohol abuse and presented over a six-year study period. Demographics, co-morbidity, clinical features, diagnostic testing, complications, and final disposition were obtained from ED and hospital records using standardized abstraction forms. Key outcome measures were the prevalence and reasons for hyponatremia, associated signs and symptoms, and treatment provided (particularly correction of sodium deficit).
Results: During the study period, 2983 elderly patients were admitted for causes related to chronic alcohol misuse. A total of 135 of the patients (4.5%) had hyponatremia with a range of serum sodium between 104 and 134 mEq/L. Thirty-eight (1.3%) had severe hyponatremia (serum sodium <125 mEq/L); 15 were diagnosed with beer potomania. Clinical features associated with potomania included weakness (11 patients), altered mental status (8), gait disturbance (7), headache (6), peripheral edema (6), and seizures (3). A consistent finding was a recent personal history of binge drinking and/or history of illness (vomiting, diarrhea) that predisposed the patient to a rapid drop in serum sodium levels. The average serum sodium concentration was 112 + 5 mEq/L; abnormal lab results consistent with diagnosis include hypokalemia (mean potassium, 3.1 mEq/L), low blood urea nitrogen (13 mg/dL), plasma osmolality (235 mOsm/kg) and decreased urine sodium levels (33 mmol/L). Patients were treated with varying concentrations of saline, diuretics, fluid restriction, and desmopressin for overdiuresis. The serum sodium increase averaged 8.1 mEq/L (mmol/L) within the first 24 hours and 13.3 mEq/L (mmol/L) by 48 hours. There were no cases of osmotic demyelination syndrome (ODS).
Conclusions: Beer potomania is a rare cause of severe hyponatremia that was previously associated with a high mortality rate. With close monitoring and judicious sodium replacement, ideally osmotic demyelination syndrome (ODS) can be avoided.

KEYWORDS Alcohol; hyponatremia; elderly

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63. Rapid Complete Cardiovascular Collapse in Less Than One Hour from an Intravenous Crotalus horridus Envenomation

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Background: Approximately 8,000 venomous snakebites are reported to US Poison Control Centers on a yearly basis but only a small number of these lead to death. Of published fatalities, the mean time to death is measured in hours to days, with very few published cases of death in less than one hour.
Case Report: We hypothesized that cardiovascular collapse within thirty minutes of snakebite occurred secondary to intravascular envenomation. This is a single patient case report. A 39-year-old healthy man was bit by a rattlesnake on the right ankle. He became unresponsive within several minutes and cardiopulmonary resuscitation was started immediately. He was transported to a community hospital where he was intubated and standard resuscitation measures were employed. After initial return of pulses he was placed on vasopressors and sodium bicarbonate infusions. He was given crotalidae polyvalent immune fab (entire hospital supply of two vials) and fresh frozen plasma. He was found to be coagulopathic and thrombocytopenic with severe metabolic acidosis. EKG showed anteroseptal ST elevations with ST depressions in lateral and inferior leads. Patient transfer to tertiary facility was attempted but he continued to intermittently lose pulses en route and died. Post-mortem examination by the county coroner confirmed two fang marks near the right medial malleolus, overlying the saphenous vein. Given the location of the bite together with rapid cardiovascular collapse in an otherwise healthy 39-year-old male, cardiac arrest was most likely secondary to intravascular envenomation. While the snake itself was not captured, Crotalus horridus is the only endemic venomous snake in this particular region of Western Pennsylvania.
Case Discussion: Several mechanisms of intravascular envenomation leading to cardiac arrest are plausible. Envenomation can lead to disseminated intravascular coagulation, which could have also caused coronary artery thrombus formation. Similarly, envenomation could cause severe hypotension due to direct vasodilation, which, combined with a preexisting unknown coronary lesion, could have led to infarction. The patient had no history of significant snake exposure, making anaphylaxis unlikely. Review of the literature reveals very few published cases of death from snakebite in such a rapid time course.
Conclusions: This case describes a unique case of rapid cardiovascular collapse secondary to intravascular envenomation by Crotalus horridus with ST elevation myocardial infarction.

KEYWORDS Crotalus horridus; STEMI; intravascular envenomation

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64. Lower Maximum Daily Dosages of Acetaminophen, as Recommended by the FDA, Have Not Made a Significant Impact on the Number of Deaths Associated with APAP Poisoning

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Background: The Food and Drug Administration (FDA) made the recommendation to McNeil, the manufacture of acetaminophen (APAP) to decrease the daily-recommended dose of APAP to 3000 mg from 4000 mg. The reason for the recommendation was in an attempt to decrease the number of potentially fatal hepatic injury from APAP poisoning. Acetaminophen (N-acetyl-p-aminophenol or APAP) is used worldwide as an antipyretic and pain reliever. Acetaminophen is an active ingredient in over 600 prescribed and over the counter (OTC) medications. The manufacture, McNeil, claims APAP is safe when taken as recommended and yet thousands of APAP toxic exposures occur each year. Acetaminophen poisonings results in thousands of people requiring inpatient admission, treatment, liver transplants, and deaths.
Methods: review the annual report published by the American Association of Poison Control Centers' National Poison Data System (NPDS) on APAP related deaths. The retrospective review will compare the difference between the yearly deaths outcomes from all formulations of only APAP recorded by AAPCC/NPDS from 2005-2014.
Results: according to the AAPCC, NPDS's annual report, the recommendation from the FDA to decrease the daily dose of APAP to 3000 mg has not made a significant Impact on the death associated with APAP poisoning. The following table will depict the number of deaths from APAP poisoning recorded by AAPCC/NPDS in sequential order from 2007 to 2014; 2007- 59, 2008-54, 2009-63, 2010-60, 2011-80, 2012-80, 2013-69, and 2014-65
Conclusions: The FDA's strategy to decrease hepatotoxicity and death by decreasing the daily dose of APAP to 3000mg is in its 4th year. The AAPCC documented death toll from APAP poisoning has risen since 2011. In the four years before 2011 death toll was 236 and since then it has risen to 294. The FDA's prudent strategies and efforts to prevent APAP toxic death conforms to their mission of keeping the public safe from medications, biological products, medical equipment, food, cosmetic and radiation. A study published in the American Journal of Preventive Medicine by Jennifer King has demonstrated that consumers have a very poor understanding of active ingredients. Taking the findings of this study into consideration, stakeholder should consider being very thorough and improving their patient teaching component in their treatment plan. Improving the patient's understanding of medication may prevent poisonings from prescribed or OTC medication or a combination of both.

KEYWORDS Hepatotoxicity; acetaminophen; death

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65. Dewshine-Associated Death

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Background: In January 2016, we identified four cases of Dewshine ingestion, which are the first reported in the state. Dewshine is a mixture of racing fuel (~100% methanol) and Mountain Dew® (PEPSICO, Inc., Purchase, New York). Two adolescents died following ingestion, and two additional adolescents were medically evaluated and released. Here we report a case of fatal Dewshine ingestion.
Case Report: The evening prior to presentation, the patient, a 16 year old male, was reportedly socializing with friends and drinking. Approximately one-half gallon of racing fuel was mixed with an unknown quantity of Mountain Dew® in a 2L bottle and ingested. The exact amount consumed is unknown. The patient allegedly appeared intoxicated and vomited all night. The next morning, he was notified that another teen that had ingested the mixture was found dead at home, and he admitted to drinking racing fuel to his father. He then had a seizure and was rushed to the emergency room. On presentation, he was intubated, and found to be acidotic with a pH of 6.7 and bicarbonate of 2 mmol/L. Several amps of bicarbonate were administered and the pH improved to 7.1. Initial lactic acid was 12.8 mg/dL and anion gap was 42. Temperature was 35 °C. On exam, pupils were fixed and he was unresponsive. The methanol level was 175 mg/dL, and due to the initiation of continuous renal replacement therapy (CRRT), the patient was started on fomepizole every four hours. The methanol level decreased to 58 mg/dL and lactic acid improved to 1.1 mg/dL. CRRT was discontinued, and fomepizole dosing was decreased to every 12 hours. Follow-up neurologic exam demonstrated that the patient had severe neurologic injury consistent with brain death. He died on hospital day 5.
Case Discussion: The life-threatening component of Dewshine is racing fuel, which is ~100% methanol. Methanol is metabolized within the body to formic acid, which is highly cytotoxic. It is likely in this case that Dewshine was consumed as a substitute for ethyl alcohol. Alcohol use and abuse is common in teenagers, as demonstrated by results of the 2013 Youth Risk Behavior Survey, which found that in the preceding 30 days, 35% of high school students admitted to drinking alcohol and 21% admitted to binge drinking.
Conclusions: The ingestion of Dewshine, a mixture of racing fuel and Mountain Dew®, caused two adolescent deaths. Community health educators should consider distributing information aimed at the teenage population regarding the risks of drinking. In addition, parents, community educators, and the health community should stress that the consumption of homemade forms of alcohol, such as Dewshine, can be deadly. Education efforts should also encourage open communication between parents and adolescents in order to identify potentially unsafe behaviors promptly.

KEYWORDS Dewshine; methanol; death

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66. Employment survey of recent medical toxicology fellowship graduates

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Background: There is limited published information describing the experience of US Medical Toxicology (TOX) fellowship graduates. We sought to describe post fellowship activities and patterns of compensation.
Methods: Contact information was solicited from the directors of the 26 ACGME-accredited TOX Fellowships for graduates 2011-

2015. Graduates were asked to complete an online survey describing pre-fellowship experience (primary medical specialty, whether fellowship was completed immediately following residency graduation), reasons for fellowship training, whether board certification was completed, nature of and percent of time engaged in TOX activities, nature of compensation for TOX activities, job satisfaction, and satisfaction with completion of fellowship.

Results: There were 62 respondents out of 122 graduates (51%). Primary medical specialties included: Emergency Medicine (n = 56), Internal Medicine (n = 2), and Family Medicine (n = 2). Twelve respondents did not enter fellowship immediately with an average delay of 4.42 yrs post residency (range: 1-12). "General interest in the subject" (n = 60); "learn something not taught in residency" (n = 32); "felt that it would help with obtaining employment" (n = 27), "hoped to get full-time job in geographic area of fellowship" (n = 13), "didn't know what else to do after residency" (n = 4) were reasons for fellowship. Forty-three out of 50 eligible graduates completed certification. TOX activities included: research (n = 35); inpt consult (n = 47); outpt consult (n = 15); poison control center (n = 31); and education (n = 51). Forty-nine respondents were employed by an academic institution, 12 by a community group, and 3 full-time government service. Completion of fellowship was not a factor in negotiated salary for community physicians. Three graduates reported no current involvement with TOX, 51 reported < = 50% of time, and 8 > 50%. Forty-eight reported compensation for TOX activities. The most common forms were: "shift buydown" (n = 22); "billed consults" (n = 19); and "PCC on-call or perdiem pay" (n = 12). Eleven reported some form of stipend and 2 reported receiving hospital-based on-call pay. Job satisfaction was reported as: very satisfied (n = 49); slightly satisfied (n = 5); satisfied (n = 5); slightly unsatisfied (n = 2); very unsatisfied (n = 1). Fellowship completion satisfaction was reported as: very satisfied (n = 52); slightly satisfied (n = 1), satisfied (n = 8), slightly unsatisfied (n = 1).

Conclusions: The majority of graduates are engaged in TOX activities, are affiliated with an academic institution, and receive some form of compensation for TOX activities. Job satisfaction and satisfaction with completion of fellowship is generally positive.

KEYWORDS Toxicology; Fellowship; financial compensation

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67. Bothrops moojeni Envenomation: A Rare Situation Further Complicated by an Uncommon Horse Allergy

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Background: Bothrops moojeni is a South American pit viper. Literature reports of clinical experience with Bothrops moojeni evenomation are extremely limited and based on experience with management outside the United States using South American equine derived antivenoms. Local effects of Bothrops moojeni envenomation include pain, inflammation, hemorrhage, and necrosis which may not resolve with antivenom; systemic effects include consumptive coagulopathy, acute renal failure, and hypotension.

Case Report: A 40 year old right hand dominant male with reported sensitization (anaphylaxis) to horses was bitten on his left 4th proximal interphalangeal region by a Bothrops moojeni and immediately noted local symptoms. Prior to arrival in the emergency department, he removed his ring and kept his hand below heart level. Subsequent management included wound care and serial exams, intravenous fluids, opioid pain medication, and serial laboratory assessments. Less than one hour post bite, local findings were limited (puncture sites, edema of digit, pain) and laboratory assessment was normal (INR 1.0, fibrinogen 384 mg/dL, platelet count 226 K/UL, creatinine 0.9 mg/dL.) By five hours post bite, local findings had progressed (edema of the hand, small hemorrhagic lesions) and laboratory assessment identified development of a coagulopathy (INR 1.4, fibrinogen <35 mg/dL.) Following guidelines typical for North American pit viper bites, 6 vials of Crotalidae Polyvalent Immune Fab, Crofab®, were administered. Local symptom progression stopped but initial control of the hematologic effects was not achieved (INR 1.4 to 2.0, fibrinogen <35 to 37 mg/dL, platelet count subsequent low of 136 K/UL) even after additional antivenom. Creatinine remained normal but urinalysis identified proteinuria. Serial doses of Crofab® (total dose 30 vials) were administered until 2.5 days post bite at which time the local wound was improved (minimal edema, resolving lesions, no pain,) the hematologic effects were partially improved (INR 1.7, fibrinogen <35 mg/dL, platelets 160 K/UL,) and proteinuria was resolved. The patient remained hospitalized until 6 days post bite and further improvement in hematologic status (INR 1.3, fibrinogen 47 mg/dL, platelets 211 K/UL.) Serial outpatient laboratory assessments continued. At 13 and 17 days post bite he had no local symptoms and no ongoing hematologic abnormalities (INR 1.0/1.0, fibrinogen 213/247 mg/dL, platelets 223/225 K/UL.)

Case Discussion: Both Antivipmyn Tri® (Instituto Bioclon, South American, equine) and CroFab® (North American, ovine) were available for use in this patient with Bothrops moojeni envenomation and a known sensitization to horses. Discussions with other experts and the patient/family on the issues as well as the lack of applicable management guidelines took place. Due to risk of anaphylaxis with Antivipmyn Tri® and the progressing but moderate envenomation, the patient was treated with Crofab®. In contrast to reports of local effects being refractory to other antivenom therapy, they appeared controlled with Crofab® treatment. Systemic effects were not fully controlled but progression may have been prevented. The patient did not have a hypersensitivity reaction to Crofab®. There was no apparent recurrence of coagulopathy. No apparent serum reaction developed.

Conclusions: Further investigation on antivenom treatment for Bothrops moojeni envenomation local and systemic effects is needed.

KEYWORDS Bothrops moojeni; Envenomation; Antivenom

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68. Development and implementation of patient care tools for the toxicology patient: review of poison center calls for common inquiries from a community health system

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Background: In 2008 and 2014 at NACCT, we presented details on the development of electronic order sets for management of acetaminophen (APAP) toxicity and exposure to radiation and nerve agents. Most recently we collaborated with our Regional Poison Center (RPC) to review the most commonly called exposures from this community health system (CHS) involving 4 hospitals (126,000 patient visits to the emergency department [ED] in 2015). Once this information was identified, electronic order sets were created for use by ED providers to effectively care for the

poisoned patient. The goals of these order sets are to streamline documentation of communication with the RPC, increase efficiency of the ED staff, and decrease potential errors that may arise from transcribing management protocols given to medical staff over the phone.
Methods: A task force was created including representatives from the Pharmacy department, Medical Toxicology, the ED, and the RPC. To determine the most common exposures between October 1st, 2013 and September 30th, 2015, data was collected from the RPC using case data, and from the electronic health records (EHR) of the CHS using ICD-9 codes. Using the data collected, order sets were created for the identified top toxins and implemented into the EHR for use by ED providers.
Results: Between October 1st, 2013 and September 30th, 2015, there were 520 and 1195 different patient encounters from the RPC and CHS records, respectively. The top identified medication exposures were similar amongst both data sets. The top 10 identified medication exposures in the RPC data were benzodiazepines (22%), opioids (11%), APAP (8%), ethanol (11%), anti-convulsants (7%), atypical antipsychotics (7%), hypnotics (4%), non-steroidal anti-inflammatory drugs (NSAIDs) (6%), selective serotonin reuptake inhibitors (SSRIs) (7%) and anti-histamines (5%). The top 10 identified medication exposures in the CHS data were benzodiazepines (22%), opioids (15%), APAP (13%), ethanol (10%), anti-convulsants (9%), recreational drugs (8%), hypnotics (7%), SSRIs (7%), and NSAIDs (6%). From these top agents, 9 different order sets were created. Components of the order sets include medication dosing for adult and pediatric patients, nursing orders, imaging orders, laboratory orders, diet orders, potential consults for other medical services, and any special instructions needed to aid in the immediate care of the poisoned patient.
Conclusions: Well crafted order sets for the acutely poisoned patient provide standardized guidance for basic management, especially for ordering items that are used less frequently or highly error prone.

KEYWORDS Order sets; Electronic health record (EHR); Emergency department (ED)

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69. Mass Casualty Phenol Exposure

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Background: Phenol is a carbolic acid that can be absorbed dermally, via inhalation, or ingestion. Toxicity manifests in caustic injury following ingestion and dermal exposure, respiratory tract irritation after inhalation, and QT prolongation, altered mental status or seizures with systemic toxicity. Phenol in household products has decreased however, can be highly concentrated for use in industrial and medical settings. We present a case of a mass exposure to concentrated phenol.
Case Report: A 43 year old man with history of severe intellectual disability presented to the Emergency Department from a podiatry clinic after ingesting a liquid solution containing 99% phenol. Prior to arrival, he had 2 episodes of non-bloody vomiting and was reporting abdominal pain. In the Emergency Department, he was immediately decontaminated with soap, water, and polyethylene glycol. His vitals were: pulse 89 bpm, blood pressure 143/82 mmHg, respirations 20 breaths/min, and Oxygen saturation 96%. On exam his clothes were saturated with phenol and had a characteristic odor. He had blistering and injury to his lips and chin with mild erythema of the oropharynx but no ulceration. He had no drooling, or stridor. His abdomen was soft and nontender. He was at his baseline neurologic status with minimal verbal communication. His CXR, EKG and laboratory analysis were unremarkable. He was admitted to the ICU for observation and endoscopy which showed moderate-severe gastritis and esophagitis. He did well and was discharged after advancement of his diet. In addition to the primary patient, 10 females and 1 additional male ranging in age from 37-53 years old presented to the ED reporting symptoms related to dermal contact with vomitus or aerosolized particles from the primary exposure. Everyone was triaged and decontaminated if necessary in a decontamination tent outside the Emergency Department. One patient had cutaneous exposure after the primary patient vomited on him. The remaining patients complained of a nasopharyngeal burning, dyspnea, wheezing, or even nausea and vomiting after the inhalational exposure. All patients had normal vital signs, EKG and physical exams. They were all discharged home asymptomatic after mild interventions.
Case Discussion: Mass casualty events provide a unique challenge for all emergency staff and can overwhelm the resources available. Our case illustrates how inhalational exposure can affect a large group of individuals working in a confined space. Phenol can be extremely toxic after ingestion, dermal absorption or via inhalation. We present the first documented cases of a phenol mass casualty event.
Conclusions: Phenol is directly caustic but a concentrated solution may also pose a risk for toxicity to those in close proximity, resulting in a unique mass casualty event.

KEYWORDS Mass Casualty; Phenol; Caustic

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70. The rise and rise of intentional pregabalin poisoning in Australia

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Background: Pregabalin is a novel gamma-aminobutyric acid (GABA) analogue used for neuropathic pain, epilepsy and anxiety. Pre-marketing trials indicated that it has a low abuse potential, and thus in Australia it is a Schedule 4 drug (not subject to additional controls/restrictions). Despite this, there have been several reports of pregabalin abuse. In addition, Pregabalin-induced suicidal ideation has been reported. Given its extensive use (ranked within the 30 most prescribed medications in the US in 2011). It is important to gather further data relating to these risks. This study describes patterns of its use and intentional poisonings in Australia.
Methods: A retrospective study of intentional poisonings with pregabalin and other anti-epileptic medications reported to an Australian poisons centre 2004-2015. Analysis of dispensing data from the Australian Pharmaceutical Benefits Scheme (PBS).
Results: There were 785 intentional poisonings with pregabalin over the study period. Exposures have increased rapidly, especially since 2011, with a 12-fold increase in exposures from 2011 to 2015 (24 and 312 poisonings, respectively). Intentional exposures to other anti-epileptic medications remained stable, or declined (valproic acid and carbamazepine) over the same period. Pregabalin exposures were more likely to involve co-ingestion of opioids, benzodiazepines, ethanol and illicit drugs when compared to gabapentin and carbamazepine (other anticonvulsants also used for neuropathic pain), Table 1. PBS data shows a ten-fold increase in pregabalin scripts dispensed from 2011 to 2014.
Conclusions: A dramatic increase in intentional pregabalin exposures was observed from 2004 - 2015. This is in line with increasing community prescribing rates. Intentional poisonings with other anti-epileptic drugs, including gabapentin, have remained

Table 1. Comparison of co-ingested drugs in intentional anti-convulsant poisonings

	Pregabalin (n = 785)	Gabapentin (n = 321)	Carbamazepine (n = 1537)
Pharmaceutical opioids	31% (244)	18% (59)	5% (72)
Be nzodiazepines/'Z-d rugs'	25% (196)	20% (64)	12% (176)
Ethanol	14% (110)	8% (25)	10% (146)
Illicit drugs	2.5% (20)	0% (0)	<1% (14)

stable or decreased over the same period. Increased pregabalin poisonings may be due to a combination of factors, including increased availability, increased use for pain, and increasing rates of abuse, addiction and suicidal ideation. Physicians prescribing pregabalin should complete a risk assessment for misuse, including an addiction history, and be vigilant for signs of misuse or suicidal thoughts. Regulatory bodies could consider scheduling changes (e.g. in Australia pregabalin could be assigned the same status as benzodiazepines).

KEYWORDS Pregabalin; Prescription drug abuse; Intentional overdose

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71. Distinguishing transaminase elevation in rhabdomyolysis compared to acetaminophen toxicity

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Background: Transaminase (LFT) elevations can occur from direct liver injury or in the setting of rhabdomyolysis. Undifferentiated overdose patients may receive n-acetyl cysteine due to elevated LFT's of unknown etiology out of concern for occult APAP toxicity. The goal of this study is to evaluate indices that can distinguish elevated LFTs due to APAP toxicity from those due to skeletal muscle injury.
Methods: This was a retrospective chart review of consecutive cases reported to our regional poison center. Two groups were compared: 1) those with elevated LFTs after APAP ingestion and 2) those with elevated LFTs and CPK with no evidence of APAP exposure or other known causes of liver injury. Patients with a CPK >1000 were excluded from group 1 and patients with an APAP concentration >10 mcg/mL were excluded from group 2. Data collected include demographics, LFTs and CPK at initial labs reported and first abnormal labs. Results reported as "greater than" a value were treated as equal to that value.
Results: After exclusions, 189 patients were included in group 1, of which 179 had complete initial laboratory data. 182 patients were included in group 2, of which 171 had complete initial laboratory data. The mean ± standard deviation of the ratio AST/ALT at time of arrival for group 1 was 1.3 ± 0.9 and for group 2 was 2.4 ± 1.5 ($p < 0.001$). Using a cutoff of AST/ALT <3.1 (group 1 mean +2 standard deviations) to select cases as more likely APAP related correctly identified 94.4% of APAP cases. Similar results were seen at time of first abnormal laboratory values. Seventeen group 1 patients and 133 group 2 patients had an initial CPK documented. For these cases, the CPK/AST ratios were 2.8 ± 3.8 and 35.2 ± 55.6 respectively ($p = 0.016$). AST/ALT ratios for this subset were not significantly different than the overall groups (1.5 and 2.5 respectively).
Conclusions: In this series, elevated transaminases after APAP ingestion had a different pattern than cases with elevated CPK. A statistically and clinically significant difference was noted in the CPK/AST ratios for the two groups, though larger prospective studies are needed to further support this finding. This study is limited by its retrospective design and use of poison center charts which frequently had missing data.

KEYWORDS Rhabdomyolysis; Acetaminophen; Transaminitis

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72. High flux dialysis with endoscopic removal of carbamazepine bezoar in the treatment of massive ingestion of immediate release carbamazepine

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Background: Carbamazepine is an antiepileptic that is also used to treat trigeminal neuralgia and bipolar disorder. Though rarely associated with fatalities, a massive overdose can be life threatening and challenging to manage. Pharmacologic bezoars are rare, and most reports are based on post mortem findings. Drugs commonly associated with pharmacobezoar formation includes iron, aspirin, venlafaxine, quetiapine, verapamil, aluminium hydroxide, activated charcoal, potassium chloride and theophylline. We report a case of carbamazepine pharmacobezoar from an ingestion of an immediate-release carbamazepine preparation.
Case Report: A 23 year old, 95 kg man was admitted to the emergency department after ingesting an unknown amount of an immediate-release preparation of carbamazepine at an unknown time. He has history of worsening paranoia, suicidal ideation and agitation. On arrival to the Emergency Department, he was agitated and vomiting. Half an hour later, he became obtunded and required intubation for airway protection. His initial serum carbamazepine concentration was 59ug/mL and peaked at 120ug/mL 16 hours later. He was started on intermittent high flux hemodialysis. EEG showed a severely abnormal, generalized cerebral dysfunction. However, no seizures were noted. On the second day of admission, he developed hypotension that responded to vasopressors. Following that episode, his dialysis was changed to continuous veno-venous hemofiltration. He received a total of 7 days of dialysis. Despite ongoing dialysis, his serum carbamazepine concentration showed fluctuations, with the highest recorded at 61.1ug/mL and lowest at 21.7ug/mL. On day 8 of admission, an endoscopy was performed for his persistently elevated serum carbamazepine concentration and a 5cm carbamazepine bezoar weighing 9.9g was removed from his stomach. The patient was transferred out of ICU within 2 days and was discharged to MHC twenty three days after admission with no permanent neurological deficit or other sequelae.
Case Discussion: Persistent fluctuations in carbamazepine concentrations over 8 days post ingestion of an overdose of immediate-release carbamazepine, despite extracorporeal removal, suggested a pharmacobezoar concretion in the GI tract. Pharmacobezoars are well known to be associated with medications containing film-forming polymers, seen with extended-release formulations. The cause of pharmacobezoar formation with the immediate release preparation in this patient is unclear. However, the apparently large amount of the ingestion combined with the anticholinergic effects of carbamazepine with GI slowing, could have contributed to the bezoar formation.
Conclusions: Massive ingestions of immediate release preparations may form concretions in the gastrointestinal tract. Persistently elevated and fluctuating concentrations of serum carbamazepine may suggest a pharmacobezoar which acts as a pharmacological depot. Clearance of carbamazepine with high flux hemodialysis is fairly rapid and appears effective in reducing elevated serum carbamazepine levels.

KEYWORDS Carbamazepine; bezoar; immediate release preparation

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73. Gimme Fuel, Gimme Fire: Trioxane Ingestion in a Toddler

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Background: Trioxane is a stable cyclic trimer of formaldehyde that exists as a white solid at room temperature. It is the active ingredient in fuel bars used to heat pre-packaged food by the military, outdoorsmen, and some relief organizations. In an acidic environment, trioxane slowly depolymerizes to formaldehyde. The risk of toxicity of trioxane is mainly extrapolated from animal data, as no previous cases of ingestion have been reported to date.
Case Report: A healthy 19-month-old male child, weighing 12.7 kg, was found by his mother chewing through the packaging of a military surplus fuel. The fuel bar was comprised of compressed trioxane and contained 95% trioxane by weight, 2-5% magnesium stearate, and methylene blue for coloring. The child's mother found a hole in the package and called the poison center. No particulate matter was found in the mouth, or on the face or hands. Mom wiped the child's mouth clean and brought him to a local emergency department where he was found to be asymptomatic with normal vital signs. Blood work drawn approximately 1.5 hours after exposure initially showed an anion gap of 10 mEq/L with a bicarbonate of 24 mmol/L. It was recommended that labs be repeated every 2 hrs and the patient be given folic acid, 2 mg/kg IV every 4-6 hours. Repeat labs at 4 hours post-exposure showed an anion gap of 11 mEq/L, with a bicarbonate of 19 mmol/L. Due to the decrease in bicarbonate, he was transferred to a facility with a pediatric intensive care unit. Repeat labs showed a stable bicarbonate level of 20 mmol/L. He was given a total of 3 doses of folic acid and discharged home 20 hours post-exposure.
Case Discussion: Trioxane is a highly concentrated formaldehyde trimer. Unlike other chemical species commonly discussed in the context of "toxic alcohols," formaldehyde is gaseous at standard temperature and pressure. It is most commonly encountered in liquid form as formalin or other aqueous hydrates, solutions or azeotropes. Significant caustic or systemic metabolic aberrations rarely result from inhalation or ingestion of formaldehyde because of the acrid nature of the substance, which typically limits exposure. Though methanol and formaldehyde both metabolize to the same toxic entity, formic acid, fomepizole would have been useless in this case. Had this patient become increasingly acidotic, hemodialysis would have been necessary, and this is why he was transferred to a facility with that capability. Folic acid was given to enhance conversion of formate to carbon dioxide and water.
Conclusions: This case represents the first documented pediatric exposure to trioxane. Formaldehyde solid derivatives may be more amenable to ingestion, with potentially catastrophic consequences.

KEYWORDS Trioxane; formaldehyde; fuel bar

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74. The App Epidemic: Investigating the Accuracy of Mobile Scale Applications

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Background: Mobile applications, or "apps," are ubiquitous to smart phones and similar devices and serve a variety of purposes. However, there is no verification of the accuracy of these readily available programs. This can pose a significant risk of incorrect dosing when used to determine the weight of a substance for medical purposes. We recently encountered a patient who suffered significant side effects after attempting to dose a weight-based medication purchased from the internet as a muscle building supplement using a smart phone "scale" application. The purpose of this study is to assess the accuracy of various apps for determining substance weight.
Methods: Using iOS and Android operating systems, we performed an app store search for scale software by entering "scale" into the search bar of an iPhone 6S and a Samsung Galaxy S6. To correct for potential confounding, we removed protective cases prior to the experiment. We downloaded the first three applications on each platform. Programs that did not produce results or that consistently generated results of "zero" were discarded. Using precision weights of known values, we used each application according to its instructions to obtain measure known weights, determined the average and percent differences between measured and true weights, and charted the results.
Results: Most of the programs downloaded were not functional. Despite investigating more than 10 applications on each platform, only three qualified for the study: two apps (Scale #1 and Scale #2) on Android and one on iOS (Scale #3). Scale #1 consistently generated values between 0.01 and 0.1 grams regardless of weight tested with no appreciable pattern. Scale #2 became more accurate with higher weights, demonstrating a 1913% error at 100 mg compared with a 15% error at 10 grams (the most accurate of all the weights studied). Scale #3 also demonstrated the least accuracy with the lightest weights, with percent error of 1633% at 100 mg compared with 88.3% at 10 grams.
Conclusions: We attempted to mimic a true-to-life situation in which an individual attempts to use a mobile phone as a scale. We presumed that a user would not accept a result of zero, and would move on to the next program. Additionally, we chose to remove the phone covers from the experimental devices to prevent variation due to case composition, a precaution that may not be taken by a layperson. While these measures may have skewed our results, they would do so in favor of obtaining a true value and therefore do not affect our conclusion: mobile phone scale applications are neither accurate nor precise, especially when used to measure weights less than one gram.

KEYWORDS Measurement; app; weight

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75. Alcohol and Energy Drinks: A Dangerous Combination

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Background: Energy drinks mixed with alcohol continue to be a trend that leads to negative health consequences. High doses of caffeine mask the body's natural way of indicating excessive alcohol consumption, thus enabling people to drink more than recommended. In 2010, the Food and Drug Administration (FDA) sent warning letters to four major companies manufacturing caffeinated alcoholic beverages regarding the health concerns surrounding caffeinated alcoholic beverages. Although the FDA has removed caffeinated alcoholic beverages from the market, poison centers continue to receive calls about both alcohol and energy drinks being mixed together for consumption. This study aimed to examine the variables associated with exposures involving energy drinks and alcohol to illustrate characteristics of these cases.
Methods: A retrospective analysis of ingestions containing energy drinks and alcohol as separate products reported to a statewide poison center system from 2001-2015 was conducted. Data were analyzed by year, patient age, gender, route, exposure site, exposure reason, management site, medical outcome, clinical effects, and treatments. Descriptive statistics were used.
Results: A total of 67 exposures were reported over a fifteen year period. Of these, 19% (n = 13) were reported in 2015. 48% (n = 32) were female and 52% (n = 35) were male. 81% (n = 54) of exposures occurred in patients 20 years of age or greater (mean 26 years), 18% (n = 12) were between 13-19 years old, and 1 exposure was reported in the 6-12 age range. The reason for the exposure was 84% intentional, 10% unintentional, and 6% adverse reaction. Of the 56 cases that were reported as intentional exposures, 48% were abuse, 32% were suspected suicide, 16% misuse, and 4% unknown. 67% of patients were already en route to a healthcare facility when the poison center was contacted, 21% of exposures were managed on site, and 12% were referred to a healthcare facility by the poison center. The five most common clinical effects reported for these exposures were 40% tachycardia, 24% agitated/irritable, 22% drowsy/lethargy, 16% vomiting, and 12% nausea. 51% of exposures were treated with IV fluids, 22% were treated with benzodiazepines, and other treatments ranged across various categories. Medical outcomes reported were 5% major effects, 37% moderate effects, 25% minor effects, 19% not followed/minimal effects, 9% unable to follow, and 5% no effects. There were no deaths.
Conclusions: Despite discontinuation by the FDA, consumption of energy drinks mixed with alcohol continues to be a problem. Patients in their mid-20s are at greatest risk. The majority of cases were intentional. A majority of these cases (79%) either sought medical treatment in a healthcare facility or were referred for medical treatment in a healthcare facility. Although few cases had major effects, it is concerning that energy drinks mixed with alcohol exposures are associated with abuse and suspected suicide.

KEYWORDS Alcohol; Energy drink; Misuse

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76. The Antifreeze didn't work! Two cases of hypothermia due to ethylene glycol poisoning

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Background: Ethylene glycol (EG) is a solvent that is often found in anti-freeze and other coolants and is a common cause of poisoning morbidity and mortality worldwide. Clinical effects are widespread, but most often considered are anion gap and osmol gap elevations, oxalate formation, and renal failure. Hypothermia has rarely been reported.
Case Reports: Case 1 - A 45 year-old male with a history of schizophrenia presented to the emergency department (ED) unresponsive after having been reported to be ataxic and dysarthric the night prior. His vital signs were normal other than a respiratory rate 22/min and temperature 32.6 °C. Initial lab work was notable for an anion gap >23, CO2 < 5 mEq/L, creatinine 3.1 mg/dL, pH <6.91, undetectable ethanol, EG 30 mg/dL. The patient was given fomepizole and underwent hemodialysis, during which he suffered cardiac arrest, with return of spontaneous circulation within 4 minutes. Head computed tomography on day 1 showed diffuse cerebral edema with increased hypoattenuation in the deep gray and white matter. He developed worsening cerebral edema, but eventually improved his level of mentation and cognitive function to his baseline. He was discharged to a rehabilitation facility on hospital day 33, still requiring intermittent dialysis. Case 2 - A 78 year old female presented to the ED with a GCS of 4 after becoming progressively altered throughout the day. She had a temperature of 32.4 °C with otherwise normal vital signs and labs showed pH 6.88, creatinine 2.12 mg/dL, CO2 < 5, and EG 16 mg/dL. Despite dialysis and appropriate supportive care, she remained unresponsive with an electroencephalogram showing status epilepticus and requiring tracheostomy placement. Both cases were initially found in their homes with no report of cold ambient temperatures and no known co-ingestants.
Case Discussion: Ethylene glycol poisoning is known to have significant systemic effects. A Pubmed search for "ethylene glycol and hypothermia" only revealed one report. Although the exact mechanism for EG-induced hypothermia has not been well described, there is likely overlap with ethanol-induced hypothermia. Proposed mechanisms include accelerated heat loss due to vasodilation, relaxation of muscles involved in the shivering response, central nervous system depression, and central thermoregulatory dysfunction. Studies on mice suggested that longer chain alcohols are more prone to inducing hypothermia. In this same study, other sedative hypnotic agents also induced hypothermia, suggesting both a similar mechanism of action as well as a possible genetic predisposition.
Conclusions: Ethylene glycol poisoning may result in hypothermia due to mechanisms that are not fully elucidated, however is likely due to CNS depression and autonomic dysregulation.

KEYWORDS Ethylene glycol; hypothermia; overdose

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77. Misrepresented Haloperidol as a Cause of Dystonia: A Case Series

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Background: Antipsychotics have been associated with dystonia due to antagonism at the D2 dopamine receptor. Dystonia causes abnormal muscle contractions of varying types including torticollis, oculogyric crisis, and opisthotonos. Anticholinergic medications are used to treat these symptoms. Haloperidol and diazepam both come in similar orange tablets making them a possible target for substitution by drug dealers. We report a series of 7 cases that presented to a single emergency department with dystonic symptoms refractory to initial diphenhydramine (DPH) treatment after ingestion of haloperidol misrepresented by a drug dealer as a benzodiazepine (BZD).
Case Reports: Seven patients presented to a single emergency department with dystonia after ingestion of orange pills that they believed to be either "Klonopin" or "Valium". Patients reported initial sedation followed by extrapyramidal symptoms. One of the patients produced a pill that was identifiable as haloperidol 5mg. There were no significant complications. Initially, a teenager presented with akathisia on the night of ingestion. He was discharged after BZD treatment for presumed panic attack before returning with torticollis that was recognized due to its similarity to other cases. The poison center was first contacted for a man in his 20's who ingested several pills the night prior. He presented the next day with tachycardia, hypertension, and opisthotonos initially mistaken for a seizure. He received intravenous (IV) DPH and lorazepam with partial symptom resolution and was admitted for further treatment. Three teenagers also presented the day after ingestion with symptoms including: torticollis treated with IV DPH and lorazepam; diffuse muscle contractions treated with IV DPH and oral baclofen; and tachycardia, miosis, and oculogyric crisis treated with IV DPH. All patients had incomplete symptom resolution and required overnight observation and treatment. Another woman presented similarly shortly after ingesting pills from the prior night. One additional patient presented at the same time with no known connection to the others but with similar dystonia after ingesting orange pills she was told were Klonopin.
Case Discussion: The timing and refractory nature of the symptoms in these cases was unusual and may be due to the high doses of haloperidol ingested (20+ mg by history & pill ID). No confirmatory levels were obtained, but the symptoms reported were consistent with dystonia from haloperidol and visual analysis of a round orange scored pill marked "MYLAN 327" supported this.
Conclusions: We report a case series of dystonia from haloperidol misrepresented as a BZD. Clinicians should consider street drug misrepresentation when taking an ingestion history.

KEYWORDS Haloperidol; Misrepresentation; Benzodiazepine

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78. Case and review: Takotsubo cardiomyopathy in xenobiotic exposure

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Background: Takotsubo cardiomyopathy (TC), first described in 2001, involves rapid-onset, reversible apical ballooning, and is associated with periods of catecholamine excess such as stress, exogenous epinephrine, pheochromocytoma, and acute neurologic disorders. Increasingly, case reports document its occurrence after poisonings and overdoses. We present a case report and literature review of TC after xenobiotic exposure.
Case Report: A healthy 25 year old woman presented to the emergency department after a grand mal seizure followed by persistent somnolence and tachycardia after ingesting up to 6g of diphenhydramine in a suicide attempt. She was intubated for airway protection, given activated charcoal, and gastric lavage removed pill fragments. An electrocardiogram (ECG) revealed an incomplete right bundle branch block. Her ICU course was complicated by persistent hypotension and tachycardia. Troponin levels peaked at 1.30 (normal: 0-0.05 ng/mL) and a transthoracic echocardiogram (TTE) revealed a non-dilated cardiomyopathy, an ejection fraction (EF) of 20%, and TC pathology. She was extubated on hospital day 1; a repeat TTE showed an improvement in her EF to 40%. She was discharged from the hospital and made a full recovery.
Methods: A PubMed search (2001 until January 1, 2016) of "Takotsubo," "Tako-tsubo," "stress cardiomyopathy," "ampulla cardiomyopathy," "broken heart," "apical ballooning," "reversible cardiomyopathy," and "neurogenic cardiomyopathy," was combined with the PubMed "Related Articles" feature and keyword query on "poisoning," overdose," "intoxication," "ingestion." Manual article reference screening was also performed. Inaccessible or poorly-controlled (i.e., medically confounded) articles were excluded. Tabulated variables include substances ingested, demographics, past medical history, echo- and ECG findings, peak troponin levels, and outcome.
Results: In 18 resulting cases, TC was reported with illicit substances (cocaine, amphetamines), prescribed medications (antidepressants, benzodiazepines, anti-malarials), over the counter substances (nasal decongestant spray), carbon monoxide and pesticide exposure, and after snake and scorpion envenomations. Patients ranged from 7-84 years old; 14 of 18 (78%) were female. 7 patients had a past medical history of hypertension; 5 patients were tobacco users. 8 patients had ST elevations on initial ECG. Peak troponin levels ranged from 0.23-45 ng/mL. Mean initial ejection fractions were 25%. All cardiac catheterizations performed (n = 12) were normal. All 18 patients had documented normalized or improved cardiac function.
Conclusions: Takotsubo cardiomyopathy is identified as a life-threatening potential consequence of xenobiotic exposure.

KEYWORDS Takotsubo; cardiomyopathy; xenobiotics

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79. Myocardial Infarction in a 16-year-old Male After Marijuana Inhalation

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Background: Marijuana (Cannabis sativa) is the most abused illicit substance in the world. Acute cardiovascular complications, including myocardial infarction and cardiomyopathy have been reported. We present the case of a 16-year-old male who presented with chest pain after smoking marijuana and was found to have an acute myocardial infarction and dilated cardiomyopathy. Exhaustive testing for synthetic cannabinoids ("K2", "spice") was negative.
Case Report: A 16-year-old male presented to an outside hospital with a chief complaint of chest pain. He reported non-radiating substernal chest pain after smoking marijuana and drinking alcohol. Electrocardiogram (ECG) showed diffuse ST segment elevations (>10mm in multiple leads). Labs were notable for a

troponin T 15.04 ng/mL (peak 23.09 ng/mL) and a CK 5818 unit/L, consistent with myocardial injury. ECHO showed mild global left ventricular (LV) systolic dysfunction and a LV ejection fraction of 42%. Cardiac catheterization showed high LV filling pressures, decreased cardiac index, and normal coronary angiography. Right ventricular biopsies were consistent with myocardial infarction. Cardiac MRI performed one month later showed a severely dilated LV with moderate to severely depressed global systolic function. Gadolinium enhancement was across multiple coronary territories, suggesting multi-vessel vasospasm as a cause for the myocardial ischemia. Urine testing was notable for an 11-nor-9-carboxy-THC level of 40 ng/mL by LC/MS. Testing for known synthetic cannabinoid metabolites was negative by GC/MS. A sample of the marijuana smoked by the patient was obtained and GC/MS identified a large Δ-9-tetrahydrocannabinol peak as well as peaks identified as various terpenoid and terpene compounds by NIST library searches. No other peaks were identified that corresponded to known synthetic cannabinoids or unknown compounds.

Case Discussion: Previous research has shown an increased risk of myocardial infarction following marijuana inhalation. This has been substantiated by multiple case reports of otherwise healthy young subjects, typically males with no risk factors for atherosclerosis, presenting with chest pain, ECG changes, and troponin leaks after smoking marijuana. Additionally, mortality in patients who have had a myocardial infarction is significantly higher in those patients who had smoked marijuana more than once per week prior to the infarction. Both coronary thrombus and vasospasm have been reported. Myocardial infarction after synthetic marijuana use has also been described. Our patient had a biopsy confirmed myocardial infarction, with normal coronary angiography after smoking marijuana, supporting a diagnosis of marijuana-induced coronary vasospasm. Testing performed on the patient as well as the sample smoked failed to identify any substance other than marijuana. Interestingly, our patient had evidence of myocardial injury across multiple coronary distributions.

Conclusions: Patients who present with chest pain after smoking marijuana should have an ECG and lab testing, including troponin T, to rule out myocardial infarction.

KEYWORDS Marijuana; Myocardial infarction; Cardiomyopathy

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80. Characterization of Compartment Syndrome Secondary to Drug Overdose

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Background: Drug overdose leads to many physical, psychological, and legal consequences. Compartment syndrome, while classically associated with traumatic injury, is increasingly being reported as a sequelae of drug overdose. Research Question - What are characteristics of compartment syndrome as a sequelae of drug overdose compared with other etiologies?

Methods: This was a retrospective chart review covering the period from 1/1/14-3/1/16, set at a university-affiliated, urban tertiary care emergency department (ED). We included patients who were 1) over the age of 17, and 2) given a diagnosis of compartment syndrome. Exclusion criteria included pediatric patients, prisoners, and pregnant patients. Our hospital ED records were queried for admission diagnoses of compartment syndrome. This study received Institutional Review Board approval.

Results: During the study period, 21 cases were identified. Cases were excluded because of age (n = 1) and lack of confirmed compartment syndrome as a diagnosis (n = 2). Data on the remaining 18 cases were abstracted for demographics, etiology, laboratory values, patient outcome, and number of overdose-related ED visits before and after the diagnosis of compartment syndrome. Of the 18 cases meeting inclusion criteria, 27.8% (n = 5) were associated with drug overdose. 89% (n = 16) of patients were male and 72% (n = 13) were white. The mean age of the overdose and non-overdose groups was 34.8 and 41.6 years, respectively. Average peak creatine phosphokinase (CPK) in the overdose group (45,510 U/L) was significantly higher than the non-overdose group (9,367 U/L; $p < .05$). All patients were treated with fasciotomies, except for one in the non-overdose group who was offered operative intervention, but declined. 60% (n = 3) of patients in the overdose group experienced significant morbidity, including moderate functional deficits, chronic wounds, and/or bilateral leg amputations. This occurred in 23.1% (n = 3) of the non-overdose group and included a post-operative hematoma and chronic lymphedema. The average number of overdose-related ED visits before and after the diagnosis of compartment syndrome was 0.8 visits and 0.4 visits, respectively, for the overdose group.

Conclusions: Over 25% of cases of compartment syndrome during our study period were a consequence of drug overdose. These patients tended to have a higher rate of residual morbidity. We hypothesize this may be due to a delay in diagnosis and more significant muscle damage, as evidence by higher mean CPK levels. Awareness of overdose-related compartment syndrome and early diagnosis are paramount in providing optimal clinical care.

KEYWORDS Compartment Syndrome; Overdose; Rhabdomyolysis

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81. Bromethalin Rodenticide Exposures Reported to US Poison Centers

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Background: Long acting anticoagulants rodenticides (LAARs) have become ubiquitous since the 1950's. Unintentional exposure rarely leads to overt human toxicity. Exposures are commonly managed by poison centers who have great familiarity with these agents. Toxicity is well-characterized and treatments are readily available. Recent restrictions on the registration of LAARs have driven the market towards use of less familiar agents. Bromethalin is a potent rodenticide that works, in part, by uncoupling mitochondrial oxidative phosphorylation. Toxicity in humans is not well characterized and no antidote is available. Ingestions of 1.67 mg/kg have caused serious toxicity and death. Outcomes of human exposures are poorly described, and when encountered, this substance may be unfamiliar to clinicians.

Methods: This retrospective observational study examined data from the National Poison Data System (NPDS) derived from U.S. poison center cases. Inclusion criteria were all human patients with exposure to bromethalin only, from 01/01/2008 to 12/31/2015, and followed to known outcome.

Results: There were 1,698 bromethalin exposures, of which 1,411 (83.1%) were pediatric, age 16 days-17 years. Patients developing no effects comprised 1,358 (96.2%) of pediatric exposures, while 47 (3.3%) had minor effects and 6 (0.43%) had moderate effects. Eleven (0.8%) exposures were attempted suicides, and 1,395 (98.9%) were unintentional. Moderate clinical effects included

nausea, vomiting, syncope, headache, prothrombin time (PT) prolongation and respiratory depression. No Major clinical effects were seen in pediatric exposures, though one exposure with major effects was of unknown age. Adult patient's age 18 - 94 years comprised 281 (16.5%) exposures. Attempted suicides composed 106 (37.7%) exposures and 154 (54.8%) were unintentional. No effects were seen in 179 (63.7%) patients, while 74 (26.3%) had only minor effects. Six patients experienced major effects and 4 had seizures. A single death was reported by suicide. Clinical effects noted in the fatality were cardiac arrest, coma, disseminated intravascular coagulopathy, intracranial bleed, respiratory depression, and PT prolongation.
Conclusions: This is the first study reporting the toxicity of bromethalin in humans from national exposure data. A majority of bromethalin exposures resulted in minor or no effects in pediatrics and adults, though, coagulopathy, death, and seizures were observed. No major effects were seen in confirmed pediatric patients regardless of exposure intent. This study is limited by its retrospective nature, passive reporting, and reliance on caller accuracy. Additional research is needed to describe the minimum toxic exposure, clinical effects, and treatments for bromethalin toxicity.

KEYWORDS Bromethalin; Rodenticide; Surveillance

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82. A case series of Eastern Massasauga Rattlesnake envenomations

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Background: The Eastern Massasauga rattlesnake (Sistrurus catenatus) is a species of rattlesnake endemic to portions of the Midwest United States and Ontario, Canada. It is considered endangered across much of this geographic range. There is minimal published descriptive data related to the clinical effects and potential hematologic abnormalities as a result of envenomation by this specific rattlesnake species. We report the largest case series to date of S. catenatus envenomations.
Methods: This was a retrospective chart review of a regional poison center database. All patient exposures to rattlesnakes reported to a single regional poison control center were reviewed for a 10-year period (2005-2015). Exposures consistent with bites by S. catenatus were selected for further analysis. Clinical effects, patient outcome, and laboratory data were reviewed and compiled.
Results: 23 cases of suspected S. catenatus envenomation were identified over this 10-year period. 78% (18/23) of exposures resulted in clinically significant progression of swelling. 74% (18/23) were treated with Cro-Fab. Severe systemic toxicity evidenced by significant coagulopathy was present in only 17% (4/23) of cases. No deaths were identified. Local pain and progressive edema were the primary symptoms described. All patients had good clinical outcomes.
Conclusions: In addition to being limited in geographic range, S. catenatus also has a relatively low risk of severe systemic toxicity and coagulopathy. Clinical outcomes were excellent in this case series in which the majority of patients received antivenin. Randomized clinical trials are needed to assess the relative contribution of antivenin therapy to clinical outcome.

KEYWORDS Massasauga; Snakes; Crotalids

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83. Poisoning information database covers large proportion of real poisoning cases in Korea

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Background: The poisoning information database (PIDB) provides clinical toxicological information commonly encountered toxic substances in Korea. The aim of this study was to estimate the coverage rate of the PIDB, by comparing the database with the distribution of toxic substances that real poisoning patients presented to 20 emergency departments. The PIDB was started to develop since 2007 and the number of toxic substances was added annually from 50 to 470 substances in 2014.
Methods: We retrospectively reviewed the medical records of patients with toxic exposure who visited to 20 emergency departments in Korea from January to December 2013. Identified toxic substances were classified into prescription drug, agricultural chemical, household product, animal or plant, herbal drug, and others. We calculated the coverage rate of the PIDB for both the number of poisoning cases and the kind of toxic substances.
Results: A total of 10,887 cases of intoxication among 8,145 patients was collected. The 470 substances registered to the PIDB covered 89.3% of identified 8,891 cases with poisoning, while only covered 45.3% among 671 kinds of identified toxic substances. According to the category, 211 prescription drugs, 58 agricultural chemicals, 28 household products, 32 animals or plants could not covered with the PIDB.
Conclusions: This study suggested that the PIDB covered large proportion of real poisoning cases in Korea. However, the database should be continuously extended to provide information for even rare toxic substances.

KEYWORDS Poisoning information; database; epidemiology

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84. A Review of Loratadine Exposures Reported to a Poison Center over a Thirteen Year Period

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Background: Loratadine is a second generation antihistamine which was originally approved by the U.S. FDA for prescription use in 1993 and subsequently received non-prescription status in 2002. Despite widespread availability, little published information exists with regards to the clinical effects of loratadine in single substance overdoses. The purpose of this retrospective review was to evaluate loratadine overdoses, characterize clinical effects observed, and focus on the duration of any moderate to severe effects.
Methods: A retrospective analysis of human adult and pediatric single-agent exposures involving loratadine reported to the Central Ohio Poison Center (COPC) between January 2003 and December 2015. Detailed chart review for verification of clinical effects, including review of case notes, was performed by the investigators for all cases coded with (1) moderate or major clinical effects or (2) any cardiovascular effects. Patient age, gender, reason for exposure, estimated dose, management site, treatment,

noted clinical effects, duration of effects and disposition were recorded for those patients experiencing any moderate effects and minor cardiovascular effects. Cases were excluded if documented as an adverse drug reaction.
Results: A total of 2,295 exposures were reported to the COPC over the 13 year timeframe. Exposures were reported for predominantly children and teenagers and consisted of 2,038 (88.8%) cases. The majority of cases involved unintentional supratherapeutic exposures (n = 2,188; 95.3%), although many intentional overdoses did occur (n = 107; 4.7%). The majority of the cases were managed on-site (n = 2,085; 90.9%), with 187 (8.1%) managed within a healthcare facility. Of this, 136 (72.7%) were treated in the ED or observation unit and released, 8 (4.3%) were admitted to a noncritical care unit, 8 (4.3%) were admitted to a critical care unit, 11 (5.9%) to a psychiatric facility, and 24 (12.8%) were lost to follow-up. A total of 1,206 (52.6%) were followed to a known outcome with 998 (82.8%) of these exposures having no effect, 155 (12.9%) minor clinical effect(s), 18 (1.5%) had moderate clinical effect(s), and 35 (2.9%) deemed as an unrelated effect. No major effect outcomes or deaths were reported. The most frequent clinical effects included drowsiness/lethargy (n = 102), agitation/irritability (n = 36), tachycardia (n = 32), vomiting (n = 32), hypertension (n = 17), nausea (n = 16), hypertension (n = 12) and other (n = 38). Hypertension with or without tachycardia, and not warranting intervention, was the most frequent reason for moderate clinical outcome classification. Most of these cases had resolution of vitals within 8 hours of exposure.
Conclusions: Moderate clinical outcomes were infrequent with no major outcomes or fatalities after loratadine overdose. Cardiovascular effects which did not require intervention were the most frequently noted moderate effect. With only mild symptoms likely, observation at home may be appropriate for the majority of unintentional ingestions.

KEYWORDS Loratadine; Poison Center; Overdose

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85. Increased hospitalizations and ICU admissions in poison center cases involving teenagers

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Background: Our poison control center staff noted an increase in the number of teenagers being admitted to ICUs. Review of our PCC data confirmed this finding, but no U.S. studies addressing this issue were found in the literature. National Poison Data System (NPDS) data was examined to better assess this trend and to look for possible causes.
Methods: Annual U.S. census data for ages 13-19 was obtained for the years 2010 to 2015. American Association of Poison Control Centers (AAPCC) NPDS reports "Management Site by Age" and "Generic Codes by Category by Age" were obtained for each calendar year from 2010 to 2015 and tabulated for the age group 13-19 years old.
Results: U.S. census data revealed an annual decrease in the teen population from 2010 to 2015, with an overall 2.37% decrease (30.1M* to 29.4M). The number of exposed teenagers reported by the NPDS management site reports fluctuated between 153K* and 158K from 2010 to 2014, but rose to 165K in 2015. The number of teens admitted to critical care, non-critical care and psychiatry increased annually from 2010 to 2015 with an overall increase of 28.7% for critical care (12.5K to 16.0K), 44.1% for non-critical care (9.3K to 13.5K) and 85.3% for psychiatry (12.7K to 23.6K). From 2010 to 2015, the percentage of exposures admitted to critical care rose from 7.99% to 9.69%, to non-critical care rose from 6.02% to 8.17% and to psychiatry rose from 8.16% to 14.2%. The number of teens managed on site decreased for 5 of the 6 years with an overall fall of 15.5% (63.2K to 53.4K). Those treated and released fluctuated between 35.9K and 37.5K from 2010 to 2013, but increased to 38.1K in 2014 and 40.2K in 2015. Those lost to follow up or left AMA stayed between 10.2K and 11.8K in all six years. Comparing the 2010 and 2015 Generic Codes by Category by Age tables found that the total number of substances involved in teenager exposures increased from 194.7K to 218.3K. The category with the largest increase was antidepressants, with 9,972 more exposures (a 68.9% increase). This was followed by analgesics (7,113 more exposures; 18.1% increase), antihistamines (4,780 more exposures; 54.2% increase) and stimulants and street drugs (2,293 more exposures; 21.2% increase). The single substance with the largest increase in exposures was diphenhydramine, alone or in combination, with 4,604 more exposures (a 113% increase).
Conclusions: This descriptive study shows that despite a decrease in the U.S. teenage population, the number of exposures involving teenagers has increased. The severity of the exposures has also worsened as evidenced by an increase in both the number and per cent of teenagers admitted to hospital. This data provides an excellent starting point for further research and also for planning educational and public health interventions.
*M = millions K = thousands

KEYWORDS Teenager poisoning; ICU admissions; epidemiology

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86. Comparison of Single and Multiple Person Natural Gas Exposures

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Background: Natural gas is a fossil fuel used as a source of energy for heating, cooking, and electricity generation. It is also used as fuel for vehicles and as a chemical feedstock in the manufacture of plastics and other commercially important organic chemicals. Natural gas is generally methane (with small amounts of short-chain hydrocarbons) and acts as a simple asphyxiant displacing oxygen from hemoglobin. Exposure to natural gas can result in serious adverse effects and even death. This investigation compares multiple-person natural gas exposures to single-person exposures reported to poison centers.
Methods: Cases were all natural gas exposures reported to a statewide poison center system during 2000-2014. Exposures involving other substances in addition to the natural gas and those not followed to a final outcome were included. The cases were divided into multiple- and single-person exposure groups. The two groups were compared by selected variables and descriptive statistics.
Results: There were 469 multiple-person incidents involving 1,478 total people, with a mean of 3.2 (range 2-28) per incident. There were 2,371 cases of single-person exposures. 27% of the multiple- and 14% of the single-person exposures involved patients aged 0-5 years, 15% multiple- and 5% single-person 6-12 years, 9% multiple- and 7% single-person 13-19 years, and 47% multiple- and 71% single-person 20 years or more. The distribution by gender for multiple- and single-person patients were 38% vs 35% male, 56% vs 64% female, and 6% vs 1% unknown. The most frequent routes of multiple- and single-person exposures, respectively, involved inhalation (95% vs 88%). The most common sites for multiple- and single-person exposures, respectively, were the

patient's own residence (83% vs 81%), school (6% vs 2%), and workplace (6% vs 11%). 75% of multiple- and 69% of single-person patients were managed on site, 21% vs 21% were already at/en route to a healthcare facility, and 3% vs 8% were referred to a healthcare facility. 4% of multiple- and 10% of single-person exposures were known or expected to have serious outcomes. No deaths occurred in the multiple-person exposures, but three occurred in the single-person exposures.
Conclusions: Multiple-person natural gas exposures are more likely to involve young children. A greater proportion of multiple-person exposures involved inhalation, occurred at school, were managed on site, and did not have serious outcomes.

KEYWORDS Natural gas; poison center; multiple-person

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87. Exposures Involving Homeless Persons Reported to Poison Centers

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Background: According to the US Department of Housing and Urban Development 2015 Annual Homeless Assessment Report to Congress, in January 2015, over 560,000 people were homeless on a given night. "Homeless" was defined as lacking housing and included people who stayed in a supervised public or private facility (e.g., shelters), unsheltered places (not meant for human habitation), and transitional housing. Homeless persons may differ from the rest of the population with respect to poisoning risk. This study describes exposures involving homeless persons reported to poison centers in one state.
Methods: Cases were all exposures reported to a statewide poison center system during 2003-2015 where the record notes mentioned that the patient was homeless or a street-person. Exposures involving more than one substance and those not followed to a final medical outcome were also included. The distribution of cases by selected variables was determined. Descriptive statistics were used.
Results: During 2003-2015, there were 581 exposures where the person was identified as homeless. There was no clear annual trend in the number of cases. The patient age distribution was 2% 0-5 years, 6% 6-19 years, 91% 20+ years, and 1% unknown age; of the patients aged 20+ years, the mean was 43 years (range 20-74 years). 73% of the patients were male. The most common routes of exposure were ingestion (86%), inhalation (9%), and bite/sting (3%). The exposure reason was 74% intentional (38% suspected attempted suicide, 20% abuse, 7% misuse), 18% unintentional, 1% adverse reaction, 1% other, and 6% unknown. 66% of the exposures involved a single substance. The management site was at/en route to a healthcare facility (81%), referred to a healthcare facility (9%), on site (8%), and other/unknown (2%). The medical outcome was no effect (16%), minor effect (23%), moderate effect (28%), major effect (9%), death (<1%) (acetaminophen and lorazepam), not followed-nontoxic (1%), not followed-minimal effects (10%), unable to follow-potentially toxic (10%), and unrelated effect (3%). Ethanol or pharmaceuticals were reported in 425 (73%) of the cases, and other substances in 222 (38%). Of the cases with ethanol or pharmaceuticals, the most common substances were ethanol (14%), quetiapine (8%), acetaminophen with hydrocodone (6%), trazodone (6%), and alprazolam (5%).
Conclusions: Toxicological exposures involving homeless persons were most commonly adult and male patients, ingestions, and intentional (particularly suspected attempted suicide), and managed at a healthcare facility. Almost half involved serious outcomes. Most of the exposures involved ethanol or pharmaceuticals.

KEYWORDS Homeless; street-person; poison center

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88. Black Widow Spider Bites At Work Reported to Poison Centers

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Background: The black widow spider (BWS, Latrodectus mactans) is one of the more dangerous spiders in the US with over 1,700 exposures reported to US poison centers in 2014. BWS are nocturnal, solitary, not aggressive unless threatened, inhabit dark places, and possess a potent venom (alpha-latrotoxin), which opens presynaptic cation channels and indiscriminately releases acetylcholine. BWS bites usually cause localized pain (pinprick), local muscle cramping, and may spread to large muscle groups. Bites occur more commonly in warmer months (April to October) and may occur in a variety of locations including the patient's workplace. These spiders can be dangerous to outdoor workers but may also enter structures and present a risk to indoor workers including machine operators, janitors, and cashiers. This investigation sought to examine whether BWS bites that occurred at the workplace differed from bites reported from callers at home or elsewhere.
Methods: This study included suspected BWS bites reported to a state-wide poison center system during 2000-2014 with patients aged 18 years or more. Bites not followed to a final medical outcome were included. Cases were grouped into those where the exposure site was coded as "workplace" (Work) and all others (Other). The distribution of cases by various factors was determined for the two groups. Medical outcomes of interest included moderate effect, major effect, death, and unable to follow-potentially toxic.
Results: Results were assessed using descriptive statistics. Of 1,854 total BWS bites, 117 (6.3%) occurred at work. The mean patient age was 35 years (range 19-73 years) for Work and 39 years (range 18-94 years) for Other. 93.2% of Work and 61.3% of Other patients were male. Bites occurred during June-August for 47.9% of Work and 38.5% of Other cases. The management site was on site in 56.4% of Work and 53.4% of Other. The medical outcome was moderate effect, major effect, death, or unable to follow-potentially toxic in 28.2% of Work and 31.4% of Other cases. The most common clinical effects for Work and Other cases, respectively, were puncture (58.1% vs 63.3%), dermal pain (55.6% vs 57.5%), erythema (16.2% vs 18.0%), edema (12.0% vs 12.9%), and unspecified pain (8.5% vs 10.2%).
Conclusions: Only a small proportion of BWS bites occurred at the workplace. Bites that occurred at the workplace were more likely to occur during June-August and involve younger and male patients, and slightly less likely to involve the most commonly reported clinical effects. In spite of differences in patient demographics and clinical effects, the management and outcome of BWS bites that occurred at the workplace were similar to all others. It is important for employers to educate their workers about their risk of exposure to venomous spiders and how they can prevent and protect themselves from spider bites.

KEYWORDS Black widow spider; occupational exposure; poison center

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89. Correctional facility exposures reported to a regional poison control center over a 10-year period

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Background: Correctional facility (CF) health care spending has increased due to an aging, and growing prison population. Implementation of telehealth services within CFs has been recommended by government agencies to contain health care costs. Currently, the regional poison control center (RPCC) provides treatment advice to health care providers caring for inmates exposed to toxic substances. The objective of this study is to describe trends in toxic exposures occurring within CFs reported to a RPCC over a 10-year period.
Methods: This is an observational case series of exposures in incarcerated adults reported to a single RPCC between January 1, 2006 and December 31, 2015. Cases were included if the caller site code was CF or state prison infirmary.
Results: A total of 555 cases met inclusion criteria. Exposures were most commonly reported in patients between 20 to 29 years of age (242; 43%). Exposures were more common in men (397; 72%) than in women (158; 28%). The most common reason for ingestion was intentional (332; 84%), and more than half of all cases were reported for suspected suicide (252; 53%). The top 5 pharmaceutical categories reported in exposures were anticonvulsants (135; 17%), antidepressants (125; 16%), analgesics (107; 13%), sedative/hypnotics/antipsychotics (75; 9%), and antihistamines (48; 6%). The majority of patients were managed within CFs (441; 79%). Of patients managed within CFs, the most common treatment was observation only (173; 39%), followed by decontamination only (78; 18%) and other supportive therapy (72; 16%). Of patients managed within a health care facility (HCF), the most common treatment was other supportive therapy (31; 28%), followed by observation only (28; 26%), and decontamination only (12; 11%). IV fluids, benzodiazepines and antiemetics were the most common supportive therapies performed at both management sites. Most patients were followed to a known outcome (455; 82%). The majority of patients experienced no to minor effects (294; 64%). Moderate to major effects were recorded in 94 patients (17%) and there were no reported deaths. Patients managed within a HCF were more likely to experiences moderate to severe outcomes compared to patients managed within a CF (OR 2.5, 95% CI 1.5 to 4.2).
Conclusions: RPCCs play an important role in managing toxicity within CFs through consultation services. Future partnerships between state CFs and the RPCC present opportunities for preventing, and managing exposures within CFs.

KEYWORDS Correctional facility; Prison; Prison Poisonings

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90. Bites by native and non-native snakes in Austria from 2003-2014

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Background: In our country venomous native snakes are Vipera berus and Vipera ammodytes. Non-native snakes are kept either in zoos or by private collectors, some of them are kept illegally. We wanted to obtain the numbers, types, and symptoms of snakebites documented in our Poisons Information Centre (PIC). Also antivenom recommendations and fatalities after snakebite were evaluated.
Methods: We analyzed retrospectively enquiries to the PIC concerning snakebites, and statistical records for fatalities due to snakebites in our country from 2003 to 2014.
Results: During the period of 12 years the PIC was consulted regarding 496 patients exposed to snakebites. 474 (95.6%) patients were bitten by native snakes. In 259 (54.6%) of these, the type of the snakes were not identified. In 42 (16.2%) cases medical observation was recommended due to symptoms like fang-marks, local reactions, pain, vomiting, hypotension, and vertigo.
In 194 (40.9%) cases of 474 native snakebites European viper (Vipera berus; n = 189) or horned viper (Vipera ammodytes; n = 5) were identified. Documented local symptoms were: fang-marks, edema, pain, hematoma in 168 (86.6%) cases; gastrointestinal symptoms (nausea, vomiting, gastralgia or diarrhoea) in 24 (12.4%) cases; hypotension in 9 (4.6%) cases; neurologic symptoms (vertigo, muscle fasciculation or paresthesia) in 9 (4.6%) cases; changes in laboratory parameters were: anaemia, leukocytosis, thrombocytopenia, haemolysis, rhabdomyolysis or coagulation disorders in 9 (4.6%) cases. Eight patients received antivenom after recommendations by PIC. Compartment syndrome developed in a 12 and a 14 years old boy (1%). In these two cases PIC was contacted some days after the bite, both of them did not receive antivenom. Twenty-one (4.4%) of all 474 native snakebites were identified as non-venomous: Aesculapian snake (Elaphe longissima; n = 12), grass snake (Natrix Natrix; n = 9). 22 patients were bitten by non-native snakes. In 15 cases hospitalisation was recommended after the bite by: Agkistrodon spp, Atheris spp, Atropoides spp, Bitis spp, Cerastes spp, Crotalus spp, Eristicophis spp, Naja spp, Trimeresurus spp, Sistrurus spp. Two deaths following non-native snake envenomation were documented: a reptile zoo keeper died 1 hour after a bite by a South American rattlesnake (Crotalus durissus); a 32 years old man, who kept many exotic snakes, committed suicide by injecting himself with a mixture of venoms from 11 different snakes.
Conclusions: In 12 years no fatalities were reported after 474 bites by native snakes (including Vipera berus and Vipera ammodytes). Antivenom was applied in only 8 cases. Two children developed compartment syndromes presumably, because antivenom was not given. In contrast, 2 fatalities occurred in 22 cases due to envenomation by non-native snakes.

KEYWORDS Snake bite; vipera berus; antivenom

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91. Carbon monoxide poisoning in Austria from 2004 to 2014

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Background: In order to reduce carbon monoxide (CO) intoxications in households, an EU-legislation (Energy-using Products Directive) regulating boiler exchanges was implemented as of September 2015. Carbon monoxide intoxications including fatalities are common in our country. In order to find a method to evaluate a benefit of this regulation, several different statistical datawere analyzed and compared.
Methods: We analyzed 3 different data sources representing CO poisonings in our country from 2004 to 2014: calls to our Poisons

Information Centre (PIC), nationwide hospital discharge diagnoses, and nationwide statistical records of fatal casualties.
Results: Over the period of 11 years the PIC was consulted regarding 184 patients exposed to CO. One hundred and fifty-one (82.1%) were older than 15 years, 11 of them consumed additionally alcohol and/or medicaments, 130 were exposed accidentally, 14 patients attempted suicide and in 7 cases the intent remained unclear. Thirty-three (17.9%) patients were under the age of 15, all of whom were exposed accidentally to CO. In 167(90.8%) cases (31 patients <15 years) symptoms were mild or moderate: nausea, vomiting, headache, vertigo, and drowsiness. Seventeen patients (9.2%; 2 persons <15 years) suffered from severe symptoms: collapse, somnolence, coma, and seizures. In all hospitalized cases oxygen therapy was recommended and in severe cases hyperbaric oxygen therapy was advised. No fatality was documented.
In the national statistical register, hospital discharge diagnoses for CO intoxication were recorded in 3064 cases (228 - 373 per year) from 2004 to 2014. During this 11-year period, CO intoxications were registered as the cause of death in 669 cases (45 - 74 per year). The fatalities were usually already found dead at the scene and therefore were neither hospitalized nor was the PIC contacted. The only utilizable hyperbaric chamber in Austria (Graz) reported the treatment of 129 patients from 2004 to 2014 (7 - 20 per year) due to CO intoxications.
Conclusions: Retrospective analysis of the available data shows the same trend. The number of calls to the PIC as well as the number of hospitalizations and deaths due to CO intoxication are slightly increasing. The analysis shows that the combination of these data can be used to evaluate regulations concerning CO intoxications.

KEYWORDS Carbon-monoxide; fatalities; statistic

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92. SUDS (single-use detergent sacs) Exposures Reported to a Statewide Poison Control System: Impact of Packaging and Labeling Changes

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Background: Exposure to single use detergent sacs (SUDS) can cause severe clinical effects, particularly in children who may mistake them for toys or candy. In a prior study (PMID 24580062) we showed that some clinical signs (neurological and respiratory effects) and certain brands are associated with more serious outcomes following SUDS exposures. The present study is a follow-up investigation, examining whether these associations still persist, and how exposure trends have changed in response to packaging and labeling improvements in some of these products.
Methods: Retrospective chart review of SUDS exposures over a 20-month period as reported to a poison control system (PCS) serving a population of 38 million. All cases were included unless the following exclusion criteria were met: non-human exposure, non-SUDS exposure, informational call or cases with only demographic information. Abstraction of data was performed by four abstractors and a mean kappa score of 0.82 on 3% of the cases was obtained. Data was analyzed for clinical associations and trends using SPSS v 23 and then compared to the previous study.
Results: After 241 results were excluded, there were 3,502 SUDS exposures from January 2013 to August 2015. Table 1summarizes demographic, clinical, and outcome data from the current and previous studies. The average number of calls per month increased from 67 in 2012 to 97 in 2013 and 125 in 2014. Binary logistic regression showed that metabolic, neurological, and respiratory effects, and the All Mighty Pacs® brand were significantly associated with moderate or severe outcomes ($p < 0.05$). In order to improve their safety profile, lid packaging and label changes were introduced for boxes of Tide Pods® in the spring and August of 2013; call volumes displayed a small, brief decrement in exposures following these changes. Specifically, PCS received 72 Tide Pod® calls in June 2013, 75 in July, 74 in August, 78 in September, 68 in October with a low peak of 53 in November. This downward trend was followed by an upward trend back to baseline numbers similar to those seen in June thru September.
Conclusions: SUDS exposures cause minor GI effects in the majority of children. CNS and respiratory effects remained the most useful predictors of serious outcomes after SUDS exposures, although metabolic derangements may also contribute. In this larger sample, the All Mighty Pacs® brand, but not Purex Ultrapacks®, were significantly correlated with moderate or severe outcomes. Packaging changes aimed at increasing the safety margin of these products showed a weak, transient benefit on exposure trends for SUDS between 2013 and 2015.

KEYWORDS Single-Use Detergent Sacs; Tide Pods(R); Laundry Products

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93. Naloxone Use by Hawaii EMS and Poison Center

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Background: Naloxone administration to an opioid-poisoned patient may improve survival chances. Because of the "US opioid epidemic", 30 states have approved bystander naloxone administration to decrease time to treatment until Emergency Medical Services (EMS) arrival. The relative importance of Time-to-patient, and time to Health Care Facility (Time-to-HCF), has not been studied. We also characterized poison center (PC) opioid calls and naloxone administration in a state where a patient's EMS records are routinely linked to the HCF record.
Methods: We queried statewide EMS provider pre-hospital medical records from 2012-2013. Pre-hospital charts were linked to hospital records. Multivariate logistic regression models of death among the transported patients were developed for EMS Time-to-patient and Time-to-HCF; control variables were patient age group, gender, and county of EMS provider. Time-to-patient and Time-to-HCF were modeled by tertiles of their distributions. NPDS queries included human, closed exposure cases by NPDS opioid generic codes (GC) and naloxone use from 2000-2015. PC calls were described by graphical and regression analyses of changes over time.
Results: Hospital records were linked to 2,427 patients who received naloxone from EMS over the 2-year period (101/month).

Adjusted odds of death for EMS patients receiving naloxone, 2012-2013

Predictive variables	Odds ratio [95% Confidence Interval]	
Patient age group	Time to patient	Time to HCF
30 years or younger	0.38 [0.21, 0.64]	0.34 [0.19, 0.57]
31-40 years	1.01 [0.60, 1.61]	0.87 [0.52, 1.38]
41-60 years	1 (reference)	1 (reference)
61-70 years	1.77 [1.21, 2.56]*	1.65 [1.15, 2.35]*
71 years and older	4.21 [3.14, 5.69]*	3.91 [2.96, 5.2]*
Male gender	1.13 [0.88, 1.46]	1.06 [0.84, 1.35]
1st terfile (<7 minutes)	1 (reference)	1 (reference)
2nd tertile (7-10 minutes)	1.14 [0.84, 1.56]	1.31 [0.97, 1.78]
3rd tertile (11 min. or more)	1. 6 [0.83, 1. 61]	1.57 [1.16, 2.12]*

*95% Confidence Interval excludes 1. 00 (p-<0. 05

Most (97%) were ≥18 years of age, mean age 53 years (SD+ 20), and 58% (1,414) males. Patient condition was described by EMS as "serious" for 82% (1,985), and critical for 17% (403). EMS recorded patient response to naloxone as "improved" for 15% (371) of patients, unchanged for 31% (748) and worsened 0.2% (4); there was no report of patient response to naloxone for 54% (1,304) of the patients. Hospital-assigned diagnoses included drug poisoning 31.0% and opiate poisoning/dependence 17.5%. Of these patients, 35% had Medicaid or similar insurance, and 7% were self-pay. These 2 forms of payment accounted for 30% ($11.1 million) of the annual $37.0 million in medical charges for all 2,427 patients. The table shows the odds of death for these patients was more closely associated with Time-to-HCF than Time-to-patient. The PC reported 2,529 exposures to 1 or more of the 33 opioid GCs during the 16 years (465 during the 24-month period of EMS data). The number of opioid exposures was increasing [95% CI] over time for 2002-2015 (9.33 [6.54, 12.1] exposures/year (n = 14, rsquare =0.815, p < 0.0001). Naloxone was reported as performed (P) or recommended and performed (RP) in 212 (14%) of 2,529 cases over the 16 years. The proportion of patients receiving naloxone has increased from 0% to 15% over 2000-2015, increasing 0.870 [0.644, 1.10] %/year (n = 16, rsquared =0.830, p < 0.0001).

Conclusions: PC calls related to opioids show the expected increase over time, and % of naloxone use of is increasing on top of that increase. Opioid exposures strain health care resources. Age over 60 years is clearly a risk factor in patients receiving pre-hospital naloxone. Time-to-HCF showed a statistically significant relation to mortality. While Time-to-patient showed an increase, it was not statistically significant. Most of the patients receiving naloxone, however, were not finally diagnosed as opioid poisoned. Thus this experience does not argue strongly against the use of bystander naloxone.

KEYWORDS Naloxon; Opioid overdose treatment; Emergency Medical Services

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94. Ibuprofen overdose: a common but minimally toxic poisoning

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Objectives: Ibuprofen is a common poisoning due to its ready availability as an analgesic. Like most non-steroidal anti-inflammatory drugs it does not appear to be associated with major toxicity but there are reports of severe toxicity with larger doses. This study aimed to investigate the epidemiology and clinical effects in a large series of ibuprofen overdoses.

Drug	N	Dose (g)	LOS (h)	ICU	Coma (GCS <9)	Ventilation	Death
Ibuprofen	84	4.6 (2.4-9.2)	9.4 (5.4-15)	0	0	0	0
Ibuprofen-Codeine	40	6 (4.8-9. 6)	16.2 (8.4-25)	2	0	0	0
Ibuprofen + Coingestants	621	2.4 (1.6-4.8}	15.4 (9-24)	32	22	23	0
All Cases	745	3 (1.6-5.3)	14.9 (8-23)	34	22	23	0

Methods: All presentations of ibuprofen or ibuprofen-codeine overdoses to a tertiary toxicology service (1987-2013) were extracted from a prospective database and reviewed. The following data was extracted: demographics, complications (coma [GCS <9], hypotension [systolic BP <90mmHg], seizure), treatments and outcomes (length of stay [LOS], intensive care [ICU] admission, death).

Results: There were 745 ibuprofen ingestions, 594 ibuprofen, 142 ibuprofen-codeine and one with both. The commonest co-ingestants were paracetamol, alcohol, benzodiazepines, selective serotonin reuptake inhibitors and atypical antipsychotics (quetiapine). There were 124 cases where only ibuprofen [84] or ibuprofen-codeine [40] were taken without co-ingestants. For these 124 admissions, the median age was 24y (interquartile range [IQR]: 18-37; range: 15-59), and 92 (74%) were female. The median ingested dose was 5g (IQR: 2.7-9.6; range: 0.4-60). The median LOS was 11.5h (IQR: 5.7-18.4). There were no deaths, no cases of coma and no requirement for intubation. Only two patients, both ingesting ibuprofen-codeine alone were admitted to ICU. The LOS, ICU admission rate, intubation rate and coma were longer and higher respectively in patients co-ingesting other drugs (Table). Patients ingesting other drugs took a lower dose of ibuprofen.

Conclusions: Ibuprofen and ibuprofen-codeine combination alone overdoses cause only minor effects in overdose, with no major complications or interventions required. Ibuprofen alone overdoses represents a younger or more female group compared to deliberate self-poisoning in general. In the majority of cases other drugs were also ingested and the other drugs appeared to result in a longer LOS and greater requirement for critical care services.

KEYWORDS Ibuprofen; overdose; epidemiology

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95. International Perspective on Prescription Benzodiazepine Exposures Reported by Poison Centres in the Global Toxicosurveillance Network

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Objectives: To describe characteristics of exposures to prescription benzodiazepines reported by Global Toxicosurveillance Network poison centres (PCs) in Europe.

Methods: Human benzodiazepine exposures reported in 2014 by PCs in France (Paris), Germany (Göttingen), Italy (Milan), Lithuania,

	France N = 209	Germany N = 737	Italy N = 1452	Lithuania N = 40	Netherlands N = 1448	UK N = 948
Age, median	33.5	41.0	42.0	22.5	41.0	39.0
Gender, %female	63.6	57.7	67.5	60.0	70.2	59.4
Route, % oral	99.5	97.7	98.7	100	99.9	98.9
Reason, % intentional	53.6	77.1	86.9	62.5		70.4
Rate per 100,000 population						
Alprazolam	1.0600	0.3083	1.0084	0.3026	1.3132	0.0828
Diazep a m	0.5173	2.3770	0.3997	0.5043	3.2206	1.1566
Larazepam	0.1950	2.3633	1.0201	0.5380	4.0703	0.2423
Rate per 1000 standard units						
Alprazolam	0.0011	0.0136	0.0016		0.0115	0.0322
Diazepam	0.0027	0.0288	0.0022		0.0154	0.0019
Larozepom	0.0005	0.0138	0.0014		0.0250	0.0029

the Netherlands, and the UK (Birmingham, Cardiff, Edinburgh, Newcastle) were examined. UK and Dutch PCs provide medical management advice to healthcare providers only, while other PCs also offer services to the public. Defined regions of coverage exist for each PC except Milan, which handles 65-70% of calls in Italy. Descriptive statistics are provided for the gender, age, exposure reason, exposure route, and rates of enquiries for particular benzodiazepine drugs (alprazolam, diazepam, lorazepam). Exposure reasons are classified as intentional and unintentional; the Netherlands are excluded as reason is not collected. Routes are classified as oral and non-oral. Rates per 100,000 population and per 1,000 standard units are presented to account for country differences. For PCs without full country coverage, the distribution of standard units sold is assumed proportional to the population. Drug utilization data are acquired from IMS Government Solutions, Inc.

Results: In each country, the greatest proportion of exposures involved a female. The median age was lowest in Lithuania (22.5 years) and highest in Italy (42.0 years). In general, the proportion of intentional exposures was substantially greater than unintentional exposures. The majority of exposures were oral. Population-based exposure rates were relatively high in the Netherlands for each drug. Adjusting for standard units sold appears to mitigate the highest population-based rates within drug classes in each country.

Conclusions: The greatest proportions of exposures in each country were oral, of an intentional nature, and occurred in females. Despite methodological differences in PC data collection, these characteristics may be explored further to identify preventive measures.

KEYWORDS Benzodiazepines; exposures; international poison centers

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96. A Characterization of Fentanyl Exposures Reported to the National Poison Data System from 2000-2015

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Background: In the US, fentanyl abuse is recently reemerging. Medical diversion and clandestine synthesis has made fentanyl increasingly available to the public for abuse. Due to its potency, fentanyl is becoming a significant cause of morbidity and mortality. Fentanyl is also used in combination or as an adulterant with illicit drugs such as heroin and cocaine with deaths reported. It is unclear whether fentanyl abused as a sole agent or in combination with other illicit drugs increases the likelihood of a major adverse outcome. We sought to characterize fentanyl exposures reported to the National Poison Data System (NPDS) to determine which factors lead to major outcomes.

Methods: We queried NPDS for all closed, human exposures to fentanyl using AAPCC Generic Code 0200628 within our 5-state regional poison center (RPC). Cases without a known outcome were excluded. Descriptive statistics for age, gender, level of health care facility (HCF) care, presence of other drugs (categorized by drug class), route of administration, medical outcome, and reason for exposure were performed. Findings are summarized in Table 1.

Results: A total of 236 exposures were identified and 69% (n = 164) met inclusion criteria.The mean age was 41 years. There were 86 (52.4%) female exposures. 54 (32.9%) patients exposed required critical care unit admission. 109 (66.5%) were single-substance fentanyl exposures. Route of administration is illustrated in Table 1. The majority of exposures involved fentanyl transdermal systems; 105 of these cases were patch ingestions. The majority of exposures involved minor outcomes (n = 78, 47.6%), followed by moderate (n = 65,39.6%), and major (n = 20,12.2%). There was one reported death. There were 103 intentional exposures, including 33 suspected suicide attempts. There were 49 cases of unintentional exposures, including adverse drug events. Of these, 33 (67.3%) resulted in only minor outcomes. Co-ingestants are shown in Table 2.

Conclusions: Cases involving parenteral exposures were surprisingly uncommon and may reflect regional trends. Historically, fentanyl has been used as an adulterant to heroin; however, this combination was not reported. As shown in Table 2, the percentage of concomitant exposures to opioids or amphetamines was high amongst exposures resulting in major outcomes and these combinations may represent increased risk of toxicity. Intentional exposures resulted in a high percentage of moderate or major

Table 1

Route of administration	Occurrences
Ingestion	105
Dermal	54
Inhalation/Intranasal	3
Parenteral	10

Table 2

Co-ingestants	Major outcomes Occurrences	Moderate outcomes	Minor outcomes	Total
Benzodiazepines	4	14	7	25
Opioids	6	13	11	30
Cocaine	0	1	0	1
Amphetamines	2	2	0	4

outcomes and may represent the use of higher doses or misuse of transdermal systems for the purposes of recreational abuse or suicide.

KEYWORDS Drugs of abuse; fentanyl; exposures

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97. A Poison Center's Experience with Pediatric Exposure to Novel Anticoagulants

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Background: Serious morbidity and mortality from pediatric accidental exposure to new anticoagulant medications have not been well studied. In the only report to date, a retrospective review from eight poison centers reported on 20 children less than 12 years of age with exposure to rivaroxaban and apixaban only. While there were no serious morbidity or mortality reported, outcome data was available for only 12 patients. The goal of this study was to identify pediatric exposures at a single poison center to novel anticoagulants and to report any major adverse outcomes.
Results: Twelve patients were identified with an age range from 10 months – 3 years. All cases originated at home and all were referred to HCF (one patient's family refused). Seven exposures were to rivaroxaban. Of these 7 patients, INR was checked in three patients. Two of the three patients had an INR of 0.9. One patient had an INR of 2.3 and a factor anti-Xa level of 75%. The other five exposures were to dabigatran. Of these patients, an INR was checked in only one patient and was reported as "normal." None of the 12 patients had any bleeding and, except for the one patient with coagulopathy following the rivaroxaban exposure, there were no major outcomes. The one patient with an elevated INR was observed overnight and released the next day with improvement of INR.
Conclusions: There were very few pediatric exposures to novel anticoagulation in a five-year period at a large single center and all exposures were associated with rivaroxaban. One patient did develop coagulopathy following rivaroxaban, emphasizing the need for continuing surveillance of pediatric exposure to these novel drugs.

KEYWORDS Rivaroxaban; dabigatran; pediatric

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98. Characteristics of Prescription Opioid Exposures in Young Children – A Retrospective Review of California Poison Control System Data

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Background: Unintentional pediatric opioid exposures are commonly reported to the poison control system in the United States. Previous studies have described the characteristics of accidental pediatric exposures specifically to methadone, buprenorphine, tapentadol and fentanyl. Few studies have focused on unintentional pediatric exposures to other commonly prescribed opioid analgesics.
Methods: This was a retrospective, descriptive study of unintentional pediatric exposures to commonly prescribed opioid analgesics (including codeine, hydrocodone, hydromorphone, morphine, oxycodone, oxymorphone, tramadol, and their derivatives) reported to the California Poison Control System (CPCS) for the 3-year period between January 1, 2012, and December 31, 2014. Inclusion criteria were single drug exposures in patients aged 0 to 6 years, and who were followed to a known outcome. Duplicate cases were excluded. We further reviewed each patient's text note and excluded cases that were incorrectly coded, were very unlikely to have had an exposure, or were judged as having a toxic effect from an adjunctive medication (such as acetaminophen). Chi-square test or Fisher's exact test was used to compare the frequency of each symptom occurrence, treatments, and coded outcomes. Statistical significance between groups was adjusted for multiple comparisons.
Results: There were 1,905 pediatric patients unintentionally exposed to a target opioid in CPCS database. Of these, 1,490 cases met our inclusion and exclusion criteria. About two thirds (64.16%) were between one and three years old. Hydrocodone and its derivatives accounted for the biggest percentage (45.1%), followed by tramadol (20.75%), codeine (19.6%), oxycodone (8.12%) and morphine (4.36%) (table 1). 639 patients (42.9%) patients were sent or referred to the hospital. Of them, 19 (1.28%) were admitted to the floor, and 25 (1.68%) to the intensive care unit (ICU). The majority of patients were asymptomatic, while 87 (5.84%) had minor effects, 28 (1.88%) had moderate effects and 6 (0.4%) had major effects. No deaths were reported. The most frequently reported symptom was altered mental status (8.12%), followed by nausea/vomiting (2.16%) and respiratory depression (2.12%). 28 cases (1.88%) were given intravenous naloxone (table 2). Three patients (0.2%) were intubated, two of them ingested oxycodone and one took tramadol. One oxycodone ingestion case developed severe bradycardia and hypotension and underwent cardiopulmonary resuscitation. Excluding hydromorphone and oxymorphone cases (due to extremely small case numbers), patients who were exposed to morphine or oxycodone were more likely to have altered mental status and worse outcome code (moderate and major effect). They were also more likely to be treated with naloxone and admitted to the ICU. In addition, oxycodone exposed patients were associated with a higher rate of respiratory depression when compared to those with codeine, hydrocodone or tramadol exposures.
Conclusions: Although uncommon, unintentional pediatric prescription opioid ingestion may lead to significant respiratory depression and bradycardia that may require intensive medical management. Compared to hydrocodone, tramadol, and codeine, unintentional exposures to morphine or oxycodone were more likely to develop significant clinical effects, to be treated with naloxone, and to be admitted to the ICU.

KEYWORDS Opioid; pediatrics; poisoning

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99. Assessing the impact of legislation on dextromethorphan use reported to a single PC

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Background: Dextromethorphan (DXM) is an over-the-counter cough suppressant that is widely abused for its hallucinogenic

and dissociative properties. Secondary to widespread abuse, there is increasing legislation in attempt to curtail abuse. This study evaluates reports of regional poison center (PC) calls before and after the January 2015 legislation in one state restricting the sale of DXM to minors.

Methods: A single PC database was queried for all records indicating exposures to products containing DXM between Jan 1, 2011 through Dec 31, 2015. Demographic characteristics (age, sex) and exposure reason were assessed. The proportion of DXM calls in 2014 and 2015 were compared through chi-square analysis. In order to account for overall changes in PC utilization, annual trends from 2011-2015 for all human exposure calls to this center, DXM specific calls, as well as intentional DXM calls were analyzed using Poisson regression.

Results: There were slightly more DXM exposures reported among males than females (52.5%, vs 46.0%), and intentional use of DXM did not vary by gender ($p = 0.187$). Children $<=5$ years accounted for approximately one-third of all DXM exposures, though the most frequently identified groups among intentional DXM exposures included teenagers 13-19 years (44.3%), and those 20-29 years of age (32.7%). The rate of all DXM exposure calls was lower in 2015 compared to 2014 (10 vs 16 per 1,000 calls, $p < 0.0001$). Similarly among intentional exposures only, there was a reduction in the rate of intentional DXM exposures per 1,000 intentional exposures between the two years (36.2 vs 23.8, $p = 0.006$). Annual trend analysis, however, has shown continuous decline in the number of calls for each type. The number of human exposure calls to the PC decreased at a rate of 641 fewer calls per year (95% CI: [-840.38, -443.22; $p < 0.001$). Similarly, the numbers of all DXM exposure calls and intentional DXM exposure calls has been declining as well with 51(95% CI: [-69.23, -33.17; $p < 0.001$] and 17 [95% CI: -25.10, -8.10, $p < 0.001$] fewer calls per year, respectively.

Conclusions: Virginia legislation effective January 1, 2015 restricted the sale or distribution of DXM or DXM-containing products to minors less than age 18. Review of a single PC data showed there was a significant decrease in 2015 compared to previous years. The data suggest legislation may have contributed towards this. However, other factors such as increased attention to these products and awareness about abuse may have also contributed to the trends. Furthermore, as PC calls are decreasing as well, the decline in DXM may not be reflective of the true burden in the community, but rather a result of decreased reporting to the PC. Further studies comparing trends between states that passed the legislation compared to those who did not may indicate the impact on DXM abuse.

KEYWORDS Dextromethophan; abuse; legislation

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100. Four Year Trend Analysis of Childhood Exposures to Liquid Laundry Detergent Packs in the U.S

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Background: Prior to introduction of Single-use Liquid Laundry Detergent (LLD) Packs in North America (Feb 2012), a prospective observational study was initiated among a representative sample of Poison Control Centers (PCCs) to evaluate reporting rates, situational variables and biological response to this product category.

Methods: Analysis of childhood LLD Pack exposures (age $<= 5$ years) reported to participating PCCs during the first four years on market, Feb 2012-Dec 2015. The case narrative was reviewed to verify medical outcome, clinical course, and key situational and product characteristics. Multi-route exposures were assigned a 'primary route' based on clinical presentation and exposure history. Analyzable cases were categorized as 'Serious' (major/moderate), 'Non-serious', or 'Unable to Assess', based on medical outcome. Coding discrepancies were reconciled with the contributing PCC. Exposure reporting rates were normalized using Nielsen consumption data.

Results: During the study period, a total of 10,709 LLD Pack childhood exposures were reported. The primary route of exposure was ingestion (84%), followed by ocular (13%), and dermal (3%). Normalized exposure reporting rates for all routes of exposure peaked during the first ten months on market (1.24 exposures per million units sold [EMUS]), and subsequently declined by 37% to a sustained rate of 0.78 EMUS (Nov 2013-Dec 2015). The mean exposure rate for serious ingestions between Feb 2012-May 2013 was 0.07 EMUS, and declined by 58% to a sustained rate of 0.03 EMUS (Jun 2013-Dec 2015). Ingestions involving single-compartment LLD Packs were 2.5 times more likely to result in a serious outcome (10.7%) as compared to multi-compartment LLD Packs (4.3%). The normalized rate of serious ocular exposures demonstrated a similar sustained reduction (0.04-0.02 EMUS, 45.3%), however there was no observed difference in the proportion of serious cases for single vs multi-compartment LLD Packs. There was no observed trend in the environmental variables evaluated. Children continue to access the LLD Pack outside of the original container in roughly 40% of analyzable exposures ($N = 2,182$). When accessed from the original packaging, the container was left open in roughly 1/3 of cases evaluated.

Conclusions: During the four year period, normalized reporting rates demonstrate a sustained reduction in exposure frequency and severity of childhood exposures. Voluntary safety standards defined in 2015 were not implemented during the time period evaluated. Continued emphasis on reduction of accidental exposures is needed.

KEYWORDS Laundry Detergent Pack; Safety Surveillance; Exposure Prevention

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101. Poison Center Deaths by Quarter; Is it a Flip of a Coin?

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Background: Poisoning deaths in the US as tracked by the CDC have risen markedly in the past 10 years. Fatalities reported by a regional poison center (RPC) are increasing as well. The final quarter of fatalities reported to the RPC in 2015 was an all time high. The aim of this study is to define the fatality trend over the last 10 years.

Methods: All fatality abstracts entered into the National Poison Data System (NPDS) by a RPC from 2006 through 2015were retrieved. Group 1 (2006 and 2007) fatalities were compared to those from Group 2 (2014 and 2015) in order to calculate a percentage change over time. Total numbers for each month (and quarter) for all 10 years were tallied for comparison.

Results: A total of 581 fatalities were submitted during the study period. Fatalities in Group 1 and Group 2 were 97 and 146 respectively (a 51% increase over time). Overall, the quarter with the highest number of fatalities was the fourth (168) while the third quarter (128) had the least. The first quarter of 2006 revealed the lowest number of fatalities (8) compared to all quarters from all years, while the fourth quarter of 2015 revealed the greatest number of fatalities (27) during the study period (a 238%

increase). Monthly distribution of fatalities for the study period ranged from 34 (September) to 71 (November).
Conclusions: Fatalities reported to a RPC appear to be increasing and there appears to be an association by both month and quarter. Further study is warranted to determine if this phenomenon is noted collectively by other US poison centers.

KEYWORDS Death; Poison Center; Seasonality

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102. Drug Abuse Patterns among Patients Living with HIV Reported to the Toxicology Investigator's Consortium

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Background: Substance abuse among those living with the human immunodeficiency virus (HIV) is a concern given the additional effects of drugs on virus-associated immune suppression. Thus, substance abuse has the potential to contribute to transmission, disease progression, morbidity, and mortality. Recent studies and data regarding drug abuse patterns among those with HIV, however, are lacking. Therefore, the purpose of this study is to describe the patterns of drug abuse related toxicity among those with HIV.
Methods: All cases of intentional exposures for intentional abuse in patients documented positive for HIV reported to the Toxicology Investigator's Consortium (ToxIC) from January 15 to March 1 2016 were extracted and analyzed. Descriptive statistics for age, gender, drug abused, treatment, chronicity, and clinical signs were performed.
Results: A total of 51 cases were extracted and included for analysis. The mean age was 38.3 years (range 16-67) and 39 (76.5%) were male. The most common drug classes abused were opioids (N = 16, 31.4%), sympathomimetics (N = 14, 27.5%), antidepressants (N = 5, 9.8%), antipsychotics (N = 4, 7.8%), and sedative hypnotics (N = 3, 5.9%). Among opioids, heroin abuse was identified in 9(17.6%) cases while prescription opioids were in 7(13.7%). Cocaine (N = 8, 15.7%) was the most common drug among the sympathomimetics. Treatment was required in 36(70.6%) cases and 8(15.7%) patients were intubated. Of the cases, 30(58.9%) were acute. The most common clinical signs were coma/CNS depression (N = 20, 39.2%), respiratory depression (N = 11, 21.6%), and agitation (N = 9, 17.6%).
Conclusions: Opioids and sympathomimetics were the most common drug classes abused among patients with HIV. Similar to national trends, among opioids, the abuse of heroin and prescription opioids appear very comparable. The youngest patient was 16 and CNS toxicity involved the vast majority of cases. This is important in that young patients with HIV may be at greater risk than adults for negative CNS outcomes related to substance abuse given their combination of immune suppressed state and ongoing brain development. Understanding these substance abuse patterns among patients with HIV can help guide and intensify education programs aimed at abuse prevention.

KEYWORDS Drug abuse; HIV; education

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103. Case Series of daycare attendees exposed to elemental mercury vapors

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Background: Recognition of the potential health dangers of mercury exposure has led to the near-complete disappearance of consumer products and medical equipment containing significant amounts of elemental mercury. Although rare today, chronic exposure to spilled elemental mercury in an enclosed space can lead to panoply of symptoms including neurobehavioral changes, abdominal ailments, peripheral neurological complaints and skin rash. Fetuses and small children are particularly vulnerable to the toxicity of mercury.
We describe a series of children and day care workers exposed to mercury vapors for three months from a broken sphygmomanometer. The index case was a three year old child admitted to the hospital for failure to thrive and a desquamating rash. As part of a broad work-up blood and 24-hour urine mercury levels were 7 mcg/dl and 79 mcg/L respectively. A search of the patient's home failed to reveal a source however environmental testing of the day care center confirmed the source (>49,000 ng/m3).
Methods: All children, day care workers and recurrent visitors were encouraged to see their physicians for mercury testing. A total of twenty-five children and four adults were evaluated. All patients were followed until urine mercury levels returned to normal.
Results: Thirteen children and 4 adults had elevated urine mercury levels. Initial levels ranged from undetectable to 182mcg/g creatinine. A variety of non-specific symptoms were reported including cough and coryza, however all symptoms except for the index case were considered mild. The index case experienced wasting and hypertension requiring supplemental feeding and anti-hypertensive therapy. Two full-time daycare workers had 24-hour urine levels of 144 and 131 mcg/g creatinine and were chelated.
Five children and two adults completed a 19 day course of oral 3-dimercaptosuccimer. Post-chelation urine mercury levels dropped to 53 mcg/g creatinine or less. All children, including the index case, clinically returned to baseline four months after cessation of exposure.
Conclusions: Well-established chelation thresholds exist for asymptomatic children with elevated lead levels from acute exposure (Dietrich 2004). Similar data is not available for mercury. The decision to chelate involves the potential benefit of more rapid elimination of body burden against cost, potential risks of metal redistribution and depletion of vital micronutrients. Challenges in this case series included rapid clearance of mercury from blood, difficulty in obtaining 24-hour urine specimen in toddlers and non-specific symptoms omnipresent in children. Benefits of chelation therapy are known to diminish with chronicity of metal exposure.
ACGIH has established an adult biological exposure index (BEI) of 35 mcg/g creatinine. It was decided that chelation with DMSA 10 mg/kg (weight-based) would be recommended for children whose symptoms were likely related to mercury and for those children whose urine mercury exceeded twice the BEI. Children whose urine mercury was less than twice BEI but was above normal would be considered on a case by case basis. A total of 5 children with urine mercury 76-182 mcg/g creatinine were chelated with resultant reduction of urine mercury to 14-53 mcg/g creatinine. Based upon our experience, all children tolerated chelation well.

KEYWORDS Mercury; chelation; pediatrics

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104. Acute toxicity associated with the recreational use of the novel psychoactive benzofuran N-methyl-5-(2 aminopropyl)benzofuran (5-MAPB)

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Background: N-methyl-5-(2 aminopropyl)benzofuran (5-MAPB) is a novel psychoactive benzofuran, created by N-methylation of 5-(2-aminopropyl)benzofuran (5-APB), with similar structure to methylenedioxymethamphetamine (MDMA). No case of 5-MAPB related toxicity has been published to date. The only sources of information are users' reports on Web discussion forums, and data from metabolic studies.
Case Report: A 24-y/o previously healthy Caucasian male was brought to the ED from a rave party, where he had orally consumed an unknown amount of 5-MAPB for recreational purposes. He presented with paleness, cold and clammy skin, sweating, disorientation in time and place, mydriasis, tremor and progressive psychomotor agitation. Physical examination revealed hypertension (157/105 mmHg) and tachycardia (169 bpm); body temperature was 38.2 °C (100.8 °F), respiratory rate was 20/min, and room air pulse oximetry was 95%. He had an increased muscle tone, and symmetric hyperreflexia without clonus. The ECG showed sinus tachycardia (138 bpm) with a minimal ascending ST-segment elevation and QTc-prolongation (465 ms). Laboratory analysis revealed creatine kinase of 305 U/L (normal <190 U/L), myoglobin of 94 µg/L (28-72 µg/L), and high-sensitive troponin T of 31 ng/L (< 14 ng/L). 2.5 h after admission hallucinations and a convulsive episode occurred. Signs and symptoms resolved substantially within 14 hours with aggressive symptomatic treatment, including sedation with benzodiazepines, fentanyl and propofol, external cooling, and treatment with urapidil. The patient remained somnolent for further 24 hours. 5-MAPB exposure was analytically confirmed by LC-MS, and 5-MAPB and its main metabolite 5-APB (Ref) were quantified. All other detected substances were used therapeutically (Table). 5-MAPB showed first-order elimination kinetics with a half-life of 6.5 hours.
Case Discussion: Due to the similar structure and according to this case it is probable that 5-MAPB acts comparable to MDMA and 5-APB, which are potent monoamine transporter blockers, and monoamine releasers. Affinity to 5-HT2A receptors and to adrenoreceptors may considerably contribute to vasoconstricting effects.
Conclusions: 5-MAPB appears to have a similar acute toxicity profile to MDMA, with marked psychomotor stimulation and pronounced vasoconstricting action.

Table. Concentrations of 5-MAPB and 5-APB, and other detected substances

Time after admission [h:min]	5-MAPB serum [µg/L]	5-APB serum [µg/L]	5-MAPB urine [mg/L]	Other substances urine
0:20	502	44		
0:40			33.1	
1:39	480	48	22.3	
03:30	308	47		caffeine, fentanyl, lidocaine lorazepam, midazolam, metabolites of nicotine and paracetamol, quinine
06:30	274	39		

KEYWORDS 5-MAPB; Novel psychoactive substances; Benzofuran

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105. Cannabinoid Hyperemesis Syndrome: An Underdiagnosed Disease Entity. A Retrospective Study

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Background: Cannabis is one of the most popular illicit drugs of abuse in the United States and worldwide. Cannabinoid hyperemesis syndrome (CHS) was first described in 2004 in patients with known heavy habitual use of cannabis who complain of recurrent cycles of vomiting. The diagnosis of CHS is often missed initially due to the similarity of the clinical presentation with other more serious etiologies of intractable vomiting. In addition, the history of cannabis use is not usually readily provided by the patient. We aimed to evaluate the prevalence of establishing the diagnosis of CHS among patients presenting with suggestive history and lab findings but with no other confirmed alternative diagnosis.
Methods: After obtaining approval from the institutional review board, a retrospective study was conducted in which we searched in the electronic medical records of a community-based teaching hospital in the time period from January 2014 to August 2015. Patients were screened for the presence of intractable vomiting and cannabis use with the terms hyperemesis, emesis and vomiting, as well as the terms cannabinoid, cannabis and marijuana, respectively. Toxicology results were further explored for possible evidence of Marijuana use.
Results: Our search returned nine patients. One patient was excluded due to a confirmed alternative diagnosis. All included patients (6 males and 2 females, median of age 27y, range 19-52y), had a final diagnosis of CHS. Only three patients (38%) had the initial working diagnosis of CHS. Gastritis and cyclic vomiting syndrome were the most common initial diagnoses. Symptoms included vomiting (100%), abdominal pain in seven patients (88%) and relief with hot showers in four patients (50%). Majority of patients admitted to daily use of Marijuana for at least one year (5 patients, 63%). Seven patients (88%) had a history of previous in-patient hospitalization due to similar symptoms. The diagnosis of CHS was established after a median of two hospital encounters (range 1-6). A history of asthma was found in three patients (38%). Six patients underwent imaging or endoscopic studies (75%). Hospital stays ranged from one to two days with three of the patients (38%) eloped or were discharged against medical advice.
Conclusions: CHS is often underrecognized in patients presenting with intractable vomiting and abdominal pain. Patients frequently undergo extensive laboratory, imaging and endoscopic workup which later proves to be non-diagnostic. Without routine screening, prompt diagnosis and appropriate addressing of cannabis use, patients remain at risk of having recurrent hospital admissions. Consensus on diagnostic criteria to facilitate early recognition is needed.

KEYWORDS Cannabinoid; Hyperemesis; Marijuana

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106. Severe Adverse Events Associated with Intentional Abuse via an Unintended Route for Fentanyl Patches Compared to Other Schedule II Opioids

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Background: Fentanyl transdermal patches are long-acting opioid products indicated for chronic pain. The extended-release (ER) nature may make them appealing for intentional abuse. Abuse via unintended routes may also increase the likelihood of a severe adverse event (SAE). The association of SAEs between fentanyl patches and other schedule II opioids in intentional abuse exposures via an unintended route was examined.
Methods: Data from the Researched Abuse, Diversion, and Addiction-Related Surveillance (RADARS®) System Poison Center Program collected between 1Q2010 to 4Q2015 were analyzed. Intentional abuse exposures via an unintended route of administration for fentanyl patches and other Schedule II opioids (oxycodone, hydromorphone, morphine, oxymorphone, tramadol, tapentadol) were examined. Schedule II opioids were restricted to tablets/capsules and stratified by immediate-release (IR) and ER. Unintended route was defined as use via any route other than dermal for fentanyl patches and any route other than swallowed whole for other Schedule II opioids. SAEs were defined as exposures that resulted in a major medical outcome, death, admittance to a critical care unit, non-critical care unit, or psychiatric facility. Multiple logistic regression adjusted for age was used to determine the association of SAEs among fentanyl patches and Schedule II IR and ER opioids via unintended route.
Results: There were 10,404 intentional abuse exposures to Schedule II opioids that had a known route and age. Of these, there were 1,850 fentanyl patch exposures and the majority occurred via an unintended route (88%). Of the 2,641 ER exposures, 37% involved an unintended route. Seventeen percent of the 5,913 IR reported an unintended route. After adjusting for the effects of age, there was a significant increase in the odds of SAE for those using a fentanyl patch via an unintended route compared to those using an IR or ER opioids via an unintended route (Table 1 - Adjusted Odds Ratio (AOR) for SAE among different opioid groups and age).
Conclusions: Abuse of fentanyl most commonly occurred via an unintended route while abuse of Schedule II IR and ER opioids most commonly occurred via the intended route. Those who abused fentanyl patches via an unintended route were more likely to experience a SAE than those who abused ER or IR tablets/capsules via unintended routes. Abuse deterrent formulations should be explored to reduce harm related to the abuse of fentanyl patches.

KEYWORDS Opioids; RADARS(R) System; fentanyl exposures

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	AOR (95% CI)	p-value
Drug group		
Fentanyl patch/ER opioids	1.66 (1.41, 1.96)	<0.001
Fentanyl patch/IR opioids	1.93 (1.63, 2.28)	<0.001
Age	0.99 (0.98,0.99)	<0.001

107. Cooking with Cannabis: A Case Report of Severe Toxicity Following the Use of Cannabutter

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Background: Tetrahydrocannabinol (THC) is generally considered to be the primary active substance in cannabis. Along with other psychoactive cannabinoids, THC is a hydrophobic oil, meaning it is insoluble in water but soluble in lipids and alcohol. "Cannabutter" is a popular butter-based solution which has been infused with cannabinoids. Cannabis edibles have become more available in recent years as the medical marijuana market has matured. We report three cases of accidental THC toxicity by ingestion of cannabutter used in home cooking.
Case Report: A 34 year old female and two children (13-year old male and 14-year old female) were brought to the emergency department (ED) by friends for mental status changes. Symptoms began approximately 2-3 hours after ingestion of a cake made with cannabutter, and included near-syncope, confusion, difficulty ambulating, blurred vision, hallucinations and myoclonic twitching. Urine drug screens in all patients was positive for cannabinoids. All three patients were admitted to the hospital for detoxification; one adolescent spent the day in pediatric intensive care for blood pressure monitoring. All responded to supportive treatment (IV fluids, lorazepam). The patients ultimately had a full return to neurologic baseline and stabilization of vitals within 36 hours and were discharged from the hospital. Child protective services were notified.
Case Discussion: This case illustrates the potential danger associated with recreational edible marijuana use. Systemic THC levels and psychoactive effects after ingestion are highly variable because of differences in bioavailability, rate of gastrointestinal absorption, and metabolic first-pass effect whereby an orally administered drug is partially metabolized (principally in the liver) before reaching systemic distribution. Clinical signs are variable and depend on both the absorbed quantity and duration of exposure. Management of patients intoxicated by cannabinoids is supportive with careful attention to the airway and breathing in children. Safe cannabis detoxification typically requires 24 hours, but sometimes longer for patients with unstable vital signs or persistent psychosis.
Conclusions: Edibles have become more available in recent years as the marijuana market has matured. With key differences in dose, onset, duration and metabolism, oral cannabis presents a considerable risk of accidental overdoses, especially in inexperienced users. Clinical signs are variable and depend on both the absorbed quantity and duration of exposure. Children with marijuana exposure are much more likely to demonstrate severe or life-threatening toxicity. Because medical cannabis is recommended for specific health conditions, regulation and quality assurance are needed.

KEYWORDS Edible Cannibas; THC; Drug of Abuse

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108. Death from diffuse alveolar hemorrhage temporally related to the use of MAB-CHMINACA and N-methyl-2-aminoindane

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Background: Deaths from recreational use of a synthetic cannabinoid (SC) or an aminoindane (AI) derivative are rare. No cases have been reported in the literature of the use of either an SC or an AI derivative causing death from diffuse alveolar hemorrhage (DAH).
Case Report: A previously healthy 18 year old teenager collapsed when smoking "K2." The teen had taken two drags from a "K2" joint with no untoward effects. Several minutes later, the decedent took another two drags from the joint and "started freaking out." Within seconds to a few minutes after the second two drags, the decedent vomited, coughed up blood and then collapsed. CPR was initiated and EMS was summoned. Resuscitative efforts were unsuccessful. The autopsy was notable for extremely hemorrhagic, edematous lungs with the right lung weighing 1300 g and the left weighing 1090 g. Average post-mortem lung weight for the decedent's gender for the right lung is 445 g (95% C.I. 155-720 g) and for the left lung is 395 g (112-675 g). Microscopically, the alveolar septal capillaries were markedly congested and erythrocytes were scattered throughout the alveolar spaces. There was no acute inflammation. Aspirated vegetable material was identified within some of the larger bronchioles with no inflammatory reaction. Also notable was blood within the stomach and proximal small intestines without any discrete or localized sites of bleeding noted.
Post-mortem toxicology testing revealed: Blood (tested by LC/quadrupole Time of Flight MS) – MAB-CHMINACA 2.7 ng/mL, A possible MAB-CHMINACA metabolite (M6) was also identified but not quantitated – N-methyl-2-aminoindane 95.4 ng/mL. Urine (tested by LC/MS/MS) – UR-144 metabolites, N-(4-hydroxypentyl) 1.7 ng/mL – N-pentanoic acid 2.6 ng/mL.
Standard forensic drug screen on whole blood was negative for 129 pharmaceuticals and chemicals.
Case Discussion: Acute DAH associated with recreational use of either an SC or an AI derivative has not been previously reported in the literature. The rapidity with which the DAH developed in an otherwise healthy teenage is quite concerning. Whether the DAH was caused by the SC or the AI is unknown. Of note, this is the first report of this particular amionindane being used in humans. Also unknown is whether or not there were other chemicals in the "K2" that caused/contributed to the DAH as the substance specifically smoked was not analyzed. The urine UR-144 metabolites are consistent with previous SC use.
Conclusions: We report the first case of a death from DAH temporally related to the use of MAB-CHMINACA and N-methyl-2-aminoindane.

KEYWORDS MAB-CHMINACA; N-methyl-2-aminoindane; Diffuse alveolar hemorrhage

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109. Evaluation of opioids/opiates involved in fatalities in one region 2012–2015

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Background: Opioid intoxication has been increasing for more than two decades. For much of this time period prescription opioid drug diversion was the primary source of the increase. Coordinated efforts at controlling the number of dispensed opioids have shown results in decrease prescriptions dispensed. In Ohio dispensed opioid prescriptions have decreased 10% from 2010 to 2015 (780M/yr to 700M/yr). However in the same period deaths caused by lethal intoxication have increased.
Methods: review of Franklin county coroner's office records for 2012 to 2015 for lethal intoxication. The office also provides service for 6 surrounding counties, providing a service for central Ohio. The drugs attributed to the fatality were based on the coroner's legal determination after autopsy and post mortem toxicology analysis.
Results: Between 2012 and 2015, 1323 deaths were attributed to lethal intoxications (OD). There was a 29% increase from 301 deaths in 2012 to 388 deaths in 2015. 973 (73.5%) of OD deaths were attributed to an opiate/opioid or polysubstance intoxication including an opioid. Heroin accounted for 504 lethal intoxications (38%), with a linear increase ($R2 = 0.953$) from 93 in 2012 to 155 in 2015. Heroin constituted 52% of opiate/opioid related intoxications. Prescription opioid related deaths decreased 24% since 2012: from 108 deaths in 2012 to 82 deaths in 2015. Fentanyl related deaths increased 240% in 2015 with 58 deaths, from a mean of 17 cases/yr (2012-2014). Prior to 2015 the source of fentanyl was prescription drug diversion (patch). In 2015 illicit fentanyl via the heroin distribution market appeared and is responsible for the sudden increase. Cocaine showed a 50% increase in 2015 with 47 cases, from a mean of 31 case/yr (2012-2014).
Conclusions: The increase in the study period is attributed to dramatic increase in illicit heroin and illicit non-pharmaceutical fentanyl fatalities, During this same period there was a noted decrease in prescription opioid fatalities. Opioid/opiate fatalities continue to increase, despite a decrease in available prescription opioids. The increase appears related to illicit heroin and illicit non-pharmaceutical fentanyl.

KEYWORDS Heroin; opioids; fatality

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110. Intentional Abuse Cases Mentioning Prescription Opioid Products Following the Hydrocodone Rescheduling

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Background: In an effort to curb abuse, the Drug Enforcement Administration rescheduled hydrocodone combination products (e.g. Vicodin®) from Schedule III to Schedule II in October 2014. This study examined whether the trend in intentional abuse cases

Table 1. Average Quarterly Change in Intentional Abuse Cases Pre/Post Hydrocodone Rescheduling

Drug	Pre (95% CI)	Post (95% CI)	p-value for difference in trends	Interaction p-value
Hydrocodone	−3.4% (−4.2, −2.6)	−6.2% (−9.9, −2.3)	0.211	REF
Oxycodone	−2.7% (−3.5, −1.9)	3.1% (−0.9, 7.3)	0.010	0.007
Other Schedule II Opioids	−3.1% (−4.0, −2.2)	4.1% (−0.3, 8.7)	0.004	0.003
Tramadol	−1.1% (−2.1, −0.1)	−3.1% (−7.8. 1.8)	0.476	0.817

mentioning hydrocodone products changed following rescheduling and whether this change differed from comparator opioids.
Methods: Data from the Researched Abuse, Diversion, and Addiction-Related Surveillance (RADARS®) System Poison Center Program were used. Intentional abuse cases from 1Q2011-4Q2015 mentioning products within drug categories of interest were included. Four drug groups were assessed: hydrocodone, oxycodone, other Schedule II opioids (fentanyl, hydromorphone, morphine, oxymorphone, tapentadol), and tramadol. The Pre-Rescheduling period was 1Q2011-3Q2014, and Post-Rescheduling was 4Q2014-4Q2015. The analysis was restricted to poison centers that provided data every quarter during the study period (n = 44). Negative binomial regression was used to compare the Pre-Rescheduling quarterly trend to the Post-Rescheduling trend with hydrocodone as the reference for interactions.
Results: Table 1 shows the average quarterly change for each drug group Pre/Post-Rescheduling. On average, hydrocodone abuse cases declined 3.4% per quarter Pre-Rescheduling to -6.2% Post-Rescheduling, a statistically non-significant change. Oxycodone declined 2.7% Pre-Rescheduling but displayed a statistically significant change to +3.1% per quarter Post-Rescheduling. Other Schedule II opioids declined 3.1% Pre-Rescheduling but increased to +4.1% per quarter Post-Rescheduling, a statistically significant change. Changes in tramadol trends were not statistically significant. The difference in oxycodone and other Schedule II opioids trends were significantly different from the change in hydrocodone.
Conclusions: After rescheduling, hydrocodone showed non-significant decline while oxycodone and other Schedule II opioids showed significant increases. This suggests regulatory changes in one drug may impact the patterns of abuse in others. Continued monitoring is needed to further determine the potential impact of this intervention on prescription opioid abuse.

KEYWORDS opioids; hydrocodone rescheduling; abuse cases

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111. Pediatric Cardiac Toxicity Associated with Fentanyl Ingestion

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Background: In the United States, opioids such as fentanyl account for up to 40% of drug related deaths. Between 2012-2014, The National Forensic Laboratory Information system reported an eight-fold increase in fentanyl seizures and drug deaths secondary to illicit drugs have increased substantially.
Case Report: A 14-year-old male was found unresponsive, hypotensive, cyanotic (50% oxygen saturation) and diaphoretic 2 hours after a reported ingestion of a half of a round, blue-green unidentified pill. There was hematemesis on his pillow and the patient had continued emesis en route to the hospital. An initial chest X-ray suggested right-sided aspiration pneumonia. Baseline investigations showed a lactate of 6.4 mmol/L, a high sensitivity troponin of 206 ng/L (normal 1-14 ng/L) and undetectable acetaminophen and salicylate levels. GC/MS was positive for fentanyl and its metabolites, cannabinoids, ondansetron, metoclopramide and ranitidine but was negative for xylazine. Chest pain was reported 8 hours post arrival. An electrocardiogram showed ST elevation over the anterior leads and the echocardiogram demonstrated borderline systolic function, though both tests were normalized on subsequent examinations. High sensitivity troponin peaked at 311 ng/L (normal 1-14 ng/L) within 24 hours whereas lactate normalized within a few hours. Cardiac inflammation in the RCA and LAD distributions possibly secondary to vasospasm was evident on a cardiac MRI performed 2 days post hospital admission. Management involved the administration of non-invasive positive-pressure ventilation, and intravenous naloxone, dopamine, norepinephrine and ceftriaxone. Patient was successfully weaned off inotropes 24 hours post admission, switched to room air a few days later and discharged home with a normal physical exam on day 5. A repeat cardiac MRI 6 months later was normal.
Case Discussion: Complications of fentanyl ingestion are usually secondary to respiratory depression. Adverse cardiovascular events have been seen when fentanyl is adulterated with xylazine. We present an unusual pediatric case of cardiac injury in the context of a fentanyl ingestion. In this case, xylazine was not detected and despite the fact that the urine drug screen was positive for cannabinoids and fentanyl, the patient's history and presentation was more consistent with recent fentanyl use. Alternative considerations that could have contributed to cardiac toxicity include a cannabinoid effect, another unidentified cardiotoxic illicit substance or a takotsubo cardiomyopathy incident.
Conclusions: Non-prescription fentanyl may cause cardiac toxicity.

KEYWORDS Fentanyl; Cardiotoxicity; Drug Abuse

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112. Adverse effects after the use of ADB-CHMINACA – a case report from the EU Spice II plus project

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Background: In 2014, the 'European Monitoring Center for Drugs and Drug Addiction' (EMCDDA) reported about thirty novel synthetic cannabinoids (SC). These included indazole-based valine derivatives carrying a cyclohexyl methyl side chain such as ADB-CHMINACA, which represents a new class of SC. It is a full agonist with a significantly higher affinity and activity at the CB1 receptor compared to JWH-018.
Case Report: A 20 yo male smoked SC with vaporizer. After 3 hours he vomited, grew restless and developed severe headache followed by an increasing clouding of consciousness. On admission in hospital, disorientation, somnolence, and impaired coordination were evident. Subsequent cerebral computed tomography findings and clinical findings were compatible with posterior reversible leucencephalopathy syndrome (PRES), according

to the neurologist. After 12 hours, the patient gradually became more alert. Complete regression of the neurologic symptoms was observed within the next 48 hours. In addition, the patient developed fever (38.8 °C) and rhabdomyolysis (creatine kinase 3102 U/L). Serum und urine samples of ED patients were analysed using LC-ESI-MS/MS for SC and their metabolites. ADB-CMINACA was identified in serum (31 ng/mL), and in urine. Ketamine, which was therapeutically applied, was also found in serum (240 ng/mL). The patient could be discharged home four days after admission.
Case Discussion: The etiopathogenesis of PRES is not fully understood. Among other potential causes a release of vasoconstricting agents leading to vasospasm and ischemia is discussed. Cannabinoids were shown to induce changes in vascular resistance including vasoconstriction, and they also modulate the actions of vasoactive compounds. Together with reports about cerebral infarction after consumption of SC this case points to the potential neurotoxicity of SC, especially of compounds such as ADB-CHMINCA, which show a much higher affinity (Ki 0.3 nM) to the CB1 receptor than the respective affinity of Δ9-THC (Ki 41 ± 2 nM) or many other SC. Furthermore, serotonin release is modulated by CB1 receptors. That may explain rhabdomyolysis and fever in case of SC overdose.
Conclusions: Clinical features of this case are characterized by PRES, and rhabdomyolysis. The latter has been reported before in case of SC overdose, while PRES has yet not been reported after the intake of SC. The case underlines the potential of the new SC for inducing severe toxicity. The third generation SC such as ADB-CHMINACA seem to have a higher potential of toxicity than former SC like JWH-018.

KEYWORDS ADB-CHMINACA; synthetic cannabinoids; new psychoactive substances

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113. Pulmonary Talcosis and Pericarditis in a Patient with Intravenous Drug Abuse

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Background: Pulmonary talcosis is a granulomatous inflammatory process resulting from talc exposure. Cases have been reported with intravenous (IV) use of drugs adulterated with talc as well as IV administration of oral drug tablets containing talc. Computed tomography (CT) findings in patients with IV exposure include diffuse micronodularity and ground glass opacities.
Case Report: A 37-year-old male presented to the Emergency Department complaining of diffuse, sharp chest pain worsened with inspiration and improved by leaning forward. Vital signs were within normal limits. Physical examination was notable for a thin man clutching his chest in moderate discomfort. Cardiac and pulmonary examination were unremarkable. An electrocardiogram (EKG) showed mild ST elevation in aVR, V1 and V2 as well as T wave inversions in leads V1 and V2 not noted on an old EKG. Troponin levels were not elevated. Cardiology was consulted and diagnosed the patient with pericarditis and recommended nonsteroidal anti-inflammatories and colchicine. Further history was obtained significant for IV injection of 15 milligram immediate release morphine tabs. The patient reported this activity approximately 6-9 times over the last week as his chronic leg pain was not controlled with his oral morphine. He denied a prior history of IV or other illicit drug use. Given this new history a CT chest was performed showing innumerable nodules with diffuse lower lobe ground glass attenuation consistent with pulmonary talcosis. A urine drug screen was positive for opiates. Confirmation by mass spectrometry found morphine, hydromorphone, and 6-mono-acetyl morphine at greater than 20,000, 2,590, and 30 nanograms per milliliter respectively.
Case Discussion: The presence of 6-mono-acetyl morphine in the patient's urine is consistent with active heroin use. As immediate release morphine tends not to contain talc, adulterated heroin is the likely source of exposure in this case. IV administration of other oral tablets containing talc is another alternative. Pulmonary talcosis does present solely with chest pain, however, given the patients' EKG findings and classic chest pain he likely had concurrent pericarditis. Autopsies on patients with pulmonary talcosis from IV exposure have found talc deposits in extra-pulmonary sites including the myocardium. It is feasible that myocardial talc deposits result in local inflammation leading to pericarditis.
Conclusions: To our knowledge, pulmonary talcosis and pericarditis have yet to be reported concurrently. As talc deposition in the myocardium is known, this raises the question of whether IV talc exposure can cause pericarditis. Further study is needed to make stronger conclusions.

KEYWORDS Talcosis; Pericarditis; Intravenous

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114. Clonazolam: A New Synthetic Drug Of Abuse

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Background: We present a case series of two patients presenting to the ED with acute ingestion and toxicity of clonazolam. These are the first published cases of clonazolam abuse in the US.
Case Reports: Case 1 - A 20 y/o male with a history of anxiety and depression presented to the ED via EMS after being found slumped over in his car. He and a friend had just ingested several tablets of a drug "Pinzor"' purchased online for recreational abuse. He had ingested three 0.7 mg pills subsequently identified as clonazolam, a new synthetic benzodiazepine. Upon arrival he had stable vital signs but became progressively hypotensive and bradycardic (HR 51, BP 79/46). Blood pressure was responsive to IV fluids and he did not require atropine. He was drowsy, although arousable, and had 3 mm pupils. ED workup was unremarkable except for a UDS positive for benzodiazepines. He was admitted to the ICU and was subsequently discharged the following morning after return of normal mental status, stable vitals, and a mental health evaluation. Case 2 - An 18 y/o male with no PMH arrived via EMS after falling asleep in the same car. He was alert upon arrival and admitted taking two 0.7 mg "Pinzor" tablets prior to becoming drowsy, and was awakened by EMS. His vitals in contrast to the other patient were significant for tachycardia and hypertension (HR 135, BP 174/107). His mentation was normal throughout his ED course except for some restlessness and anxiety. His work-up was remarkable for a creatinine (1.10 mg/dl), a TSH (517 mlU/L), and a positive UDS for benzodiazepines, THC, and opiates. He was admitted and had an uneventful hospital course. His creatinine and vital signs normalized after IV hydration, and he was discharged the following day. Laboratory analysis of the purchased pills by GCMS and liquid chromatography time of flight mass spectrometry confirmed clonazolam.

Case Discussion: We believe these are the first reported cases of clonazolam ingestion and subsequent toxicity in the US. The Poison Center and Toxicology service were consulted from the ED and had no knowledge of this particular drug. Online searches revealed that clonazolam is a designer benzodiazepine intended for "research use" produced in Europe and only available for online purchase. A PubMed search for 'clonazolam' results in only one publication in which a case report of flubronazolam, another designer benzodiazepine, ingestion is described. Of note there are multiple online social forums in which the use of clonazolam is described. Our case series is notable due to the markedly different presentations of the two patients in regards to vital signs and mental status. Whether this was a dose dependent relationship of clonazolam or due to a combination of the clonazolam, THC, and opiates in the second patient is uncertain.
Conclusions: We hope this case series will raise awareness for the use of clonazolam, a new designer benzodiazepine in recreational abuse, as well as encourage further studies of this drug.

KEYWORDS Clonazolam; Toxicity; Drug of Abuse

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115. The Myth of the Gateway Drug: Comparing Nonmedical use of Prescription Medications to Traditional Drugs of Abuse in the Adolescent Population

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Background: The general public perceives nonmedical use of prescription medications as safe compared to illicit drugs, despite growing concerns among US health care professionals. Some illicit drugs, such as alcohol and marijuana, are labeled "gateway" drugs which lead to misuse of more dangerous agents, but to the authors' knowledge, no studies have investigated prescription medications as potential "gateway" drugs, particularly among adolescents. We hypothesize that prescription medications are used earlier than traditionally accepted "gateway" drugs and lead to similar levels of healthcare facility (HCF) utilization.
Methods: This is a retrospective cohort study of Poison Control Center data from 2005-2014. Cases with exposures to an identifiable prescription medication or an illicit substance for the purpose of intentional misuse or abuse among those aged 9-21 years were analyzed. Cases were assigned to cohorts based on accepted developmental models of "Pre" (9-11 years), "Early" (12-14), "Middle" (15-17) and "Late" adolescence (18-21) and to ordinal levels of HCF utilization (pre-hospital, emergency department, floor, or intensive care units). Comparisons were made between prescription and illicit drug groups by age, developmental stage and level of care using ANOVA and Chi-square.
Results: 24,335 cases were analyzed (559 pre-, 3801 early-, 9640 middle- and 10,335 late-adolescent cases). Younger cohorts used more prescription stimulants and inhalants ($p<0.001$) while older cohorts used more alcohol, illicit stimulants and opioids, ($p<0.001$). All prescription agents with the exception of opioids were used at a younger age than all illicit substances except inhalants ($p < 0.002$). The use of all substances peaks at age 16. Prescription medications and illicit drugs required comparable levels of HCF utilization except middle adolescents using illicit substances, who required a higher level of care than all others ($p < 0.001$).
Conclusions: Prescription medications use occurs at earlier ages, preceding commonly perceived "gateway" drugs, while using equally high levels of HCF resources. Substance use peaks during middle adolescence, which is widely accepted as a developmental stage characterized by increased impulsivity and altered perception of risk-reward balances which may reflect changes in the nucleus accumbens and prefrontal cortex. Limitations exist, including lack of confirmatory drug studies and standard limitations of poison center data. Educational efforts aimed at healthcare providers and parents may be warranted to abate the "gateway" phenomenon of prescription medications in adolescents.

KEYWORDS Drug of Abuse; Pediatrics; Addiction

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116. Drug Exposure Trends Since Decriminalization of Marijuana in Colorado

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Background: Marijuana was decriminalized in Colorado in 2012 and the first retail stores opened January 1, 2014. Much speculation surrounded whether legal marijuana would affect the rates of abuse of other popular, illicit drugs of abuse. The purpose of this study was to determine whether marijuana decriminalization and legalization affected rates of illicit and prescription opioid drug abuse in Colorado.
Methods: All cases of marijuana, fentanyl, prescription opioids, heroin, and cocaine exposures reported to the National Poison Data System between 2010-2015 in Colorado were extracted using retrospective generic codes for each drug. A descriptive analysis was then performed on the data. The total drug exposures reported to our regional poison center were quantified by year for each category. The yearly exposures for each drug were then divided by population to reflect yearly per capita data. The data was then plotted on a line graph to illustrate trends (Figure 1).
Results: Prior to decriminalization of marijuana in 2012, prescription opioid exposures increased from 591 to 641 from 2010 to 2012, while marijuana exposures increased from 95 to 110. Since marijuana decriminalization in 2012, marijuana exposures have increased each year, from 110 to 235 (Table 1). While marijuana exposures increased, prescription opioid exposures decreased from 641 to 576. However, no significant changes were evident in the numbers of fentanyl, heroin, and cocaine exposures.
Conclusions: After the decriminalization of marijuana, ease of access and decreased legal ramifications may have led to increased exposures. At the same time, increased use of marijuana may have led to decreased exposures of prescription opioid abuse. On the other hand, this increased access to marijuana may also refute the belief that marijuana can serve as a gateway drug as there has not been a correlating increase in illicit drug use since the decriminalization of marijuana.

KEYWORDS Marijuana; decriminalization; drug abuse

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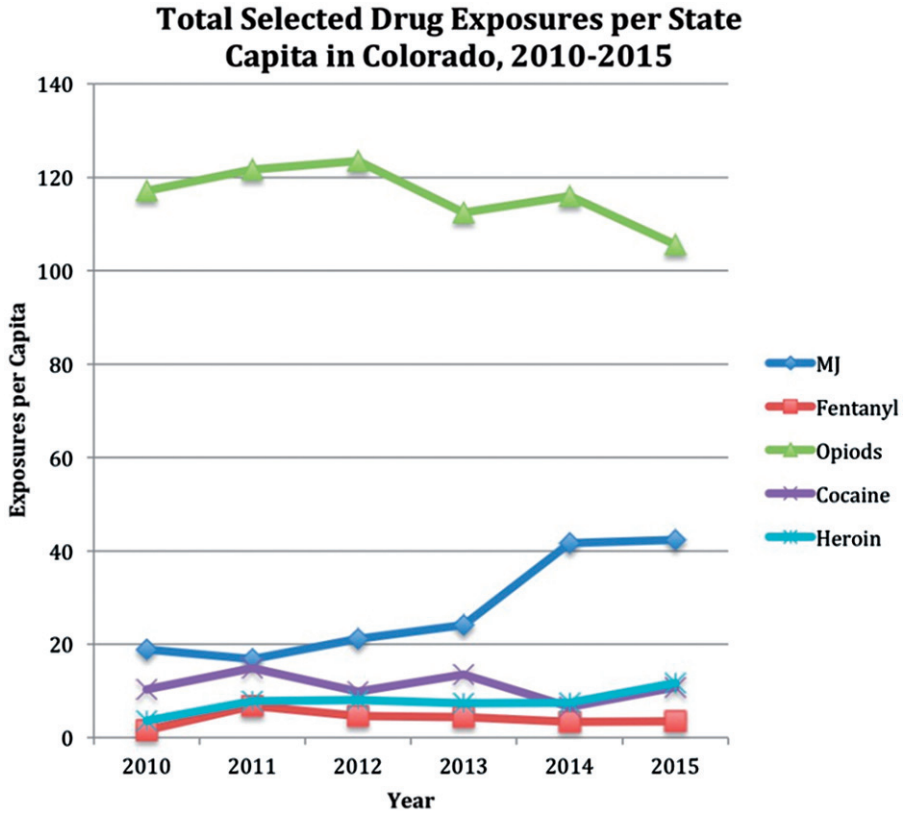

Figure 1. Total selected Drug Exposures per State Capita in Colorado, 2010-2015

Table 1. Total exposures

Year	2010	2011	2012	2013	2014	2015
MJ	95	86	110	127	223	231
Fentanyl	8	35	24	23	18	19
Opiods	591	623	641	593	621	576
Cocaine	52	76	51	71	35	59
Heroin	18	40	42	39	40	64

117. Analytically confirmed exposure to novel psychoactive substances in patients presenting to hospital with severe clinical toxicity in the United Kingdom. Results from the Identification Of Novel psychoActive substances (IONA) study

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Objective: The emergence of novel psychoactive substances (NPS) as recreational drugs has challenged emergency departments and poisons centers because of the large numbers of substances involved and the lack of information about their pharmacology and toxicology. The use of branded 'legal high' products is a particular challenge as the chemical constituents may not be known.

The UK Identification Of novel psychoActive substances (IONA) study is collecting biological samples from patients presenting to hospitals with severe toxicity and aims to identify the NPS involved and link these to the clinical features documented. Here we describe results for the first 49 participants recruited from 7 hospitals in England and Scotland.

Methods: With ethical approval, adults (≥16y) presenting to participating hospitals with severe acute toxicity (according to specific definitions) after NPS exposure were recruited with informed consent or, for those individuals without capacity at the time of presentation, with the agreement of an appropriate relative/representative. Clinical features recorded using a structured data collection sheet. Blood and urine samples were collected and analysed by liquid chromatography-tandem mass spectrometry.

Results: Samples were analysed for 49 patients (42 male, 7 female; median age 33, range 16-58 years) presenting between March and November 2015. NPS were detected in 37 patients (76%), most often synthetic cannabinoid receptor agonists (SCRAs, n = 24), which included MDMB-CHIMICA (7), 5F-AKB48(4), 5F-ADB-PINACA (3), STS-135 (3) 5F-NPB-22 (3) FUB-PB-22 and 5F-PB-22 (3). Other NPS identified included methiopropamine (8), 25I-NBOMe (7), ethylphenidate (4) and mephedrone (3). Traditional drugs of misuse were also identified in 23 patients and included methamphetamine (10), amphetamine (9), methadone (10), diazepam (3), MDMA (2), and morphine (1). Clinical and laboratory features

most commonly recorded with analytically confirmed NPS exposure were confusion (23, 62%), reduced level of consciousness (22,59%), agitation (21, 57%), tachycardia >140/min (20, 54%), acidosis (13, 35%), paranoia, elevated creatine kinase (each 12, 32%), hypertension, aggression, hallucinations or increased liver transaminases (each 8, 22%). Seizures were reported in 7 patents (19%). Nine patients (24%) required intubation and ventilation. Comparing SCRA-exposed and other NPS-exposed patients, the only significant differences in clinical features identified were reduced frequencies of tachycardia (Odds Ratio 0.21, 95% CI 0.05, 0.98, P = 0.047) and pyrexia (OR 0.05, 95% CI 0.005, 0.495, P = 0.0104) in SCRA users.

Conclusions: A wide range of substances can be identified in samples from patients presenting with severe toxicity after suspected NPS use with SCRAs most commonly identified in this UK cohort. Further data collection is needed to establish how well substances involved can be inferred from the clinical features observed.

KEYWORDS Novel psychoactive substances; drug misuse; synthetic cannabinoid receptor agonists

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118. Breaking Bad: Which Benzo is Baddest?

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Background: An increasing number of benzodiazepine related prescriptions, abuse, and emergencies have been reported. The aim of this study is to compare morbidity and mortality between 4 different benzodiazepines in patients reported to US poison centers.

Methods: The National Poison Data System (NPDS) was retrospectively queried for all cases involving alprazolam, clonazepam, diazepam, and lorazepam. Patients 12 years and older that were managed between January 1, 2004 and December 31, 2014 were included. Total number of cases, intubations, and deaths were obtained in order to define trends over time. Group 1 (2004-05) was compared to Group 2 (2013-14) for each drug and reported as percent change.

Results: Table 1 lists the absolute numbers of cases, intubations, and deaths for each of the 4 benzodiazepines. The percentage included refers to the degree of change occurring between Group 1 and Group 2. Alprazolam had the highest number of cases, intubations, and deaths while diazepam had the lowest. When comparing Group 1 with Group 2, all 4 benzodiazepines demonstrated an increase in deaths with alprazolam and clonazepam having the largest increase (162% and 120% respectively). Diazepam was the only drug resulting in reduced percentages (cases and intubations) between the 2 time periods.

Conclusions: US poison center data indicate that benzodiazepine related morbidity and mortality has increased over time. Several reasons may contribute to this phenomenon. Further study is warranted to better define this evolving public health dilemma.

KEYWORDS Benzodiazepines; intubations; deaths

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	ALPRAZOLAM	CLONAZEPAM	DIAZEPAM	LORAZEPAM
CASES	583999 (24%)	516666 (38%)	132065 (−23%)	256057 (39%)
INTUBATIONS	55480 (32%)	52899 (51%)	12766 (−6%)	24187 (62%)
DEATHS	5738 (162%)	3285 (120%)	785 (7%)	1255 (79%)

119. Drug/Cannabis Associated Delirium Treated with Dexmedetomidine-Two Cases

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Background: Dexmedetomidine (dxm) is increasingly used to treat major withdrawal symptoms and hyperadrenergic crisis. We present 2 cases of cannabis/drug induced delirium unresponsive to traditional tranquilizer treatment but is responsive to dxm as adjunct therapy.

Case Reports: Case 1 - A 21 y/o male, with a history of attention deficit hyperactivity disorder, (Rx amphetamine-dextroamphetamine 30 mg daily, non compliant for 2 weeks), smoked hashish up to 9 times daily for several months. Last use was 6 days prior to admission. He had been extremely agitated, combative, had incoherent speech with word salad speech pattern, and was unable to sleep 1 week PTA. BP was 147/76 with heart rate of 116 to 159 (sinus). Agitation, paranoid ideation, and hallucinations increased over 13 hours despite receiving chlorpromazine 25 mg IM, benztropine 1 mg IM, olanzapine 20 mg IM, haloperidol 30 mg IM and lorazepam 6 mg IM. He was placed in locked restraints. A low-grade fever of 102° F and rhabdomyolysis (peak serum creatinine kinase 13,745 IU/L) developed. Upon ICU transfer, lorazepam (totaling 11 mg IV over 5 hours) and dxm mean dose 0.4 μg/kg/h were administered. After 3 hours the fever resolved, he slept, and locked restraints were removed. Tachycardia resolved in 5 hours. Dxm was continued a total of 19 hours. The urine carboxy-THC was >500 ng/ml 7 days after the last reported use; other drugs were negative. Serum THC and carboxy-THC were 14.7 ng/ml and >100 ng/ml, respectively 7 days after last use. Case 2 - A 20 y/o female was admitted with a 40 mg alprazolam and 100 mg olanzapine overdose. She also admitted to smoking hash daily for 5 weeks. The patient became delirious 3 hours after ingestion. Her blood pressure was 140/53, with a pulse of 174. Her pupil size was small with incomprehensible speech. Urine drug analysis was positive for Carboxy-THC at 444 ng/ml and alpha OH-alprazolam at 8013 ng/ml. Dxm at a mean dose of 0.25 ug/kg/hr was started. After one hour on dxm, the patient was described as "less aggressive at this time, seems to be calming down" and was sleeping at 2 hours. The patient was sedated with tachycardia resolution at 9 hours and dxm was discontinued at 13 hours. A total of 5 mg of diazepam was also given.

Case Discussion: Dxm, a parenteral presynaptic α2-agonist, has been increasingly used to manage drug withdrawal. It was effective in stabilizing and treating our patients with hyperadrenergic delirium from cannabis/drug intoxication that was refractory to various psychotropic and tranquilizing agents. Also, dxm prevented administration of escalating doses of tranquilizers.

Conclusions: Dxm with benzodiazepine were effective in treating delirium due to cannabis/drug related delirium in these patients.

KEYWORDS Cannabis; Delirium; Dexmedetomidine

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120. U-47700, a synthetic opioid obtained online

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Background: U47700 is a synthetic opioid developed by a team at Upjohn in the 1970s that is selective for the μ-opioid receptor, 7.5 times the potency of morphine in animal models. U-47700 has not been studied in humans but would be expected to produce typical opioid effects, including sedation, euphoria and respiratory depression. U-47700 is most often used as a substitute for other opioids and has a short duration of action. It has emerged as a drug of abuse and can be bought online for as little as $40 per gram. Sweden made U-47700 illegal in January 2016. We report two patients presenting after insufflation of U-47700.

Case Report: A 26 yo male and 24 yo female consumed alcohol and alprazolam, then insufflated a substance they believed to be "synthetic cocaine" named U-47700, purchased on the internet. The male was later found face down on the lawn, with agonal breathing and cyanosis. Exact down time is unknown, but he was reportedly last seen normal 30 minutes before by his girlfriend who called EMS.

EMS reported the male patient to be cyanotic with O2 sats of 50%. He was intubated in the field and placed in a c-collar for transport. In the ER, physical examination revealed pinpoint pupils. Vital signs: HR 125, BP 150/63, T 97.4, Sat 84% on the ventilator which rapidly improved to 100%. CT chest exam showed patchy consolidation. UDS was negative. The patient was admitted to ICU, sedated on propofol and given antibiotics for presumed pneumonia. The patient self extubated in the ICU and was discharged 3 days after presentation with a normal exam. The female presented with anxiety, shivering, nausea and abdominal pain. She reported no breathing difficulty but was drowsy. She noted she felt "cool and relaxed" after using the agent. Pupils were normal sized and reactive. Vital signs: HR 97, BP 111/77, T 97.8, RR 18, Sat 100% room air. Her UDS was (+) for cannabinoids. She stayed for 24 hour observation and did well.

Analysis of the male patient's urine detected U-47700 at a concentration of 0.05 ng/mL

Case Discussion: These cases are concerning because this is a relatively new agent that the authors were previously unaware of. It is easily available on the internet. Users may not understand its effects or that it is an opioid, thus, potentially resulting in more dangerous use than that exists with use of an illicit drug. In this case, the users were expecting a cocaine high but instead got opioid overdoses.

Conclusions: Use of U-47700 has been reported to the European Monitoring Center for Drugs and Drug Addiction. Limited information about the drug is available in scientific literature. Anecdotal reports describe the substance being administered by a number of different routes.

KEYWORDS Opioid; Synthetic; Abuse

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121. A review of the illicit and prescription drug trade on the darknet

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Background: The development and globalization of the internet has provided novel and increasingly stealth means to purchase and sell prescription drugs and drugs of abuse. Heightened police surveillance has led to the relocation of many online drug markets to the more secure darknet. The darknet is best described as a network requiring specific software, configurations, and authorizations, often using non-standard communications protocols and ports in order to ensure identity protection. Global illicit drug use is increasing and represents a growing socioeconomic burden. Currently our knowledge of trends in drug use is based upon surveys and lagging indicators based on reported toxicity. Illicit and prescription drug availability on the darknet could act as a proxy for drug demand and be a leading indicator for global drug use trends. The purpose of this study is to categorize the availability of illicit and prescription drugs of abuse in darknet drug markets.

Methods: The darknet drug marketplaces Alphabay, Abraxas, Dream Market, and Nucleus were accessed through the Tor browser. Totals for each drug class were recorded. Total individual drug products available for purchase from each marketplace were recorded as well. A product's drug class was site specific and based on the site's own designation. Drug class and individual drug availability was characterized and described.

Results: Cannabis (N = 37,577) had the highest number of products available across all four sites among drug classes, (Table 1). Empathogens (N = 20,674), stimulants (N = 20,044), benzodiazepines (N = 10,247), psychedelics (N = 9,965), and opioids (N = 9,798) followed in popularity. Among individual drugs, "weed" was the most common drug available followed by cocaine, methylenedioxymethamphetamine (MDMA), heroin, and lysergic acid diethylamide (LSD) (Table 2). Fentanyl products (N = 1,133), including the non-traditional opioids carfentanil, W-18, and U-47700 were identified for sale. Novel cathinones, phenylethylamines, and unclassified research chemicals were also identified.

Conclusions: Our findings are the first to categorize illicit drug availability on the darknet. Cannabis was the most abused drug globally in 2014, which is reflected by its continued high demand

Table 1.

Drug Class	Number of Products by Drug Class
Cannabis	37577
Empathogens	20674
Stimulants	20044
Benzodiazepines	10247
Psychedelics	9965
Opioids	9798

Table 2.

Individual Drugs	Number of Products
Weed	18687
Cocaine	8121
Methylenedioxymethamphetamine	7852
Lysergic acid diethylamide	4464
Amphetamine salts	3423
Heroin	3127
Meth amphetamine	2975
Ketamine	1755
Mushrooms	1203
Fentanyl & derivatives	1133

on the darknet in 2015. The availability of fentanyl and its derivatives, as well as ketamine and other commonly diverted drugs of abuse is of increasing concern. The availability of illicit drugs on the darknet for purchase may reflect global drug use trends. Monitoring illicit drugs of abuse availability on the darknet may serve to identify novel drugs of abuse and be a leading indicator predicting future drug epidemics.

KEYWORDS Drugs of abuse; darknet; drug markets

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122. Critical Illness Due to Use of Phenibut as a Recreational Drug of Abuse

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Background: Phenibut (4-amino-3-phenyl-butyric acid) is a psychoactive xenobiotic that has recently seen an uptrend in utilization as a drug of abuse. It is readily available through unregulated internet sources as a dietary supplement. Structurally, it bears resemblance to baclofen and gamma-aminobutyric acid (GABA) and is theorized to cause agonism at the GABA-B receptor. We present a series of patients who either self-report or were found on drug testing to have ingested phenibut.
Case Report: This is a case series of three consecutive patients who ingested phenibut between May 2015 and March 2016. All three were men, ages 32, 38, and 62. All patients presented with a period of alternating agitated delirium and sedation. All three patients underwent endotracheal intubation for airway protection in the emergency department. Phenibut was detected in urine of one patient by gas chromatography/mass spectrometry. The other two patients were known to be using phenibut through self-report or historical data. All three patients were taking other prescribed or illicit psychoactive xenobiotics. All three required intensive care unit (ICU) admission, with mean ICU length of stay of six days (range 3-8 days). The oldest patient developed acute respiratory distress syndrome (ARDS) requiring prone ventilation and neuromuscular blockade. Eventually he suffered multi-system organ dysfunction leading to death.
Case Discussion: We describe three patients who presented with phenibut toxicity. They demonstrated a similar pattern of mixed agitation and delirium consistent with GABA-B agonist toxicity. In each case, the amount of consumed phenibut is unknown. However, the reported amount ingested by the patient who perished was "two teaspoons". Coingestion of other agents may likely have contributed to toxicity, thereby confounding interpretation the clinical syndrome of phenibut exposure alone. The patient who died as a result of phenibut toxicity had several medical comorbidities, including chronic obstructive pulmonary disease and ischemic cardiomyopathy. Protracted delirium seen in one patient may have been the result of phenibut withdrawal that, based upon its short half-life and similarity in mechanism to baclofen, could be expected after cessation of chronic use.
Conclusions: Phenibut is a readily available substance, the use of which is rising in popularity. Our experience shows that patients use this xenobiotic can experience life-threatening complications including respiratory failure and multiorgan damage due to a clinical picture similar to other GABA-B agonists, such as baclofen and gamma-hydroxybutyrate.

KEYWORDS Phenibut; GABA-B; dietary supplement

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123. Reverse Takotsubo Cardiomyopathy Secondary to d,l-Amphetamine Ingestion

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Background: Adderall® is a mixture of amphetamine stereoisomers, 25% d- and 75% l-amphetamine. It is an indirect sympathomimetic indicated in treatment of attention deficit-hyperactivity disorder and obesity. Reverse, or inverted, Takotsubo cardiomyopathy is a condition associated with excessive catecholamine stimulation of cardiac tissue leading to a characteristic pattern of contractility mirroring the classical Takotsubo pattern. We present a case demonstrating an association between the abuse of d,l-amphetamine and development of "reverse" Takotsubo cardiomyopathy.
Case Report: A healthy 23-year-old woman with a history of depression presented for evaluation of shortness of breath, chest pain and palpitations. She endorsed non-prescribed ingestion of an unknown amount of d,l-amphetamine six hours prior to evaluation in the emergency department. On EKG, she was found to have ST-elevation in aVL and aVR and ST-depression in the inferior and lateral leads. Chest radiograph was consistent with pulmonary edema; troponin and brain natriuretic peptide levels were elevated. She was intubated for respiratory distress and placed in intensive care. Cardiac catheterization was significant for apical hyperkinesis with basal hypokinesis and normal coronary angiography. Ejection fraction was 20-25%. She had a prolonged ICU course secondary to delirium. Urine gas chromatography/mass spectroscopy analysis was significant for amphetamine as well as methadone and oxycodone. She was extubated and discharged to home on hospital day 13 and had a normal ejection fraction and wall motion on repeat echocardiography at a two-month follow-up visit.
Case Discussion: Previous case reports have demonstrated that amphetamine-like substances, such as 4-fluoroamphetamine, methylphenidate, and methamphetamine, are associated with development of both classical and "reverse" Takotsubo cardiomyopathy, although no previous case reports demonstrate the inverted pattern associated solely with abuse of d,l-amphetamine. In general, approximately 10-25% of cases of stress-induced cardiomyopathy possess the "inverted" configuration, possibly due to the regionally differentiated expression of beta-2 receptors in the cardiac tissue.
Conclusions: The massive catecholamine release due to ingestion of d,l-amphetamine was associated with development of reverse Takotsubo cardiomyopathy in the absence of other sympathomimetic drugs.

KEYWORDS Adderall; amphetamine; Reverse Takotsubo

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124. Preliminary data from a prospective observational study: harms from cannabis exposures

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Background: 23 states currently have medical marijuana laws and 4 states and 2 territories have legalized recreational marijuana. Since legalization, there has been a proliferation of cannabis products including concentrates, extracts, resins, and edibles. However, little is known of the medical harms from acute exposures. We prospectively characterize acute exposures to cannabis products.

Methods: This is initial data (2/1-3/13/2016) from a prospective study. We solicited calls to the poison center (PC) from all emergency departments (EDs) in a state that recently legalized recreational cannabis. All toxicologists and SPIs received study education, monthly reminders, and used standardized data collection forms. EDs were encouraged to call the PC on all cannabis exposures via: co-written letters from the state health department and PC that were distributed to EDs; a flyer (for posting) sent to all EDs; ED directors contacted by phone. When EDs called the PC, the toxicologist interviewed the treating practitioner and the patient/family by phone.

Results: To date, 29 cases have been reviewed. Fifteen (52%) were female, and patient age ranged from 1-91y. Exposures to cannabis have included dried plant material, concentrates, extracts, oils, and baked goods. Signs and symptoms include nausea/vomiting (11), anxiety/panic (7), dizziness/ataxia (7), altered mental status (6), tachycardia (5), somnolence (4), "out of body" sensation (3), obtundation (2), syncope (2), myoclonus (2), agitation (2), and respiratory failure with intubation (1). When reported, the source of the exposure was homemade (12), dispensary (5), and friend (4). When reported, the intent of use was accidental (8), recreational (7), use for chronic conditions (4), and other therapeutic use (3).

Representative cases - 1yoM ingested homemade BHO (~80% THC) with subsequent agitation, tachycardia, obtundation, respiratory failure, with mechanical ventilation and aeromedical transfer. 8yoM ate 2 commercial cannabis cookies (50mg THC total) and developed tachycardia, anxiety, and chest tightness. 38yoM ate 2 commercial cannabis cookies (1gTHC total), experienced tachycardia, anxiety, palpitations, vomiting, and was treated with benzodiazepines and prolonged ED observation. 60yoF ingested homemade Rick Simpson Oil (500mgTHC in a capsule) in an attempt to treat basal cell carcinoma and experienced tachycardia, agitation, vomiting, ataxia, and required anti-emetics and prolonged ED observation. 91yoF was given a cannabis extract in tea by a friend to treat glaucoma and experienced dizziness, vomiting, and required anti-emetics and ED observation.

Conclusions: Cannabis exposures occur in a variety of age groups, from a variety of sources, and a variety of products. Symptoms range from tachycardia and anxiety to respiratory depression with mechanical ventilation.

KEYWORDS Marijuana exposures; Cannabis products; prospective observational study

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125. Infectious complications after intravenous buprenorphine/naloxone use: a report of 4 cases

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Background: Buprenorphine/naloxone (BNX) is available via the sublingual route for the treatment of opioid dependence. Buprenorphine, a partial agonist at mu-opioid receptors, decreases cravings and blocks the effects of most other opioids due to high receptor affinity. Naloxone is an opioid antagonist which is active when used intravenously (IV) and serves as an abuse deterrent in this formulation. Data suggest lower rates of abuse with BNX as compared to buprenorphine alone. We report four cases of intravenous BNX abuse hospitalized with infectious complications related to this use.

Case Reports: Case 1 - 25 year-old male with history of IVDU presented with right upper extremity pain. He was found to have cellulitis after injecting BNX with resultant staphylococcus aureus bacteremia and tricuspid valve vegetations concerning for endocarditis. He was treated with an extended course of antibiotics and transitioned to naltrexone. Case 2 - 28 year-old male with history of opioid abuse presented with bilateral dorsal foot pain after BNX injection. He was diagnosed with cellulitis and had areas of necrosis. He was treated with an extended course of antibiotics and continued on BNX with close follow up. Case 3 - 22 year-old male presented with left upper extremity swelling and erythema. He admitted to injecting BNX and was found to have an abscess with surrounding cellulitis. The abscess was drained and he was treated with antibiotics and given clonidine and hydroxyzine for opioid withdrawal. Case 4 - 19 year-old male presented with paroxysmal fevers, Janeway lesions, and splinter hemorrhages. He admitted to BNX injection into external jugular vein. Blood cultures were negative with no vegetations identified on echocardiogram, but had history of doxycycline use prior to admission. He was treated with an extended course of antibiotics and was tapered off BNX in the hospital.

Case Discussion: We report 4 cases of IV BNX abuse in patients hospitalized with complications of IVDU. Whether BNX has particulate matter or any ingredients that may enhance bacterial growth or increase risk of infection is unclear. Although there are lower rates of abuse than buprenorphine alone products, abuse by intravenous injection is not completely mitigated for BNX.

Conclusions: Despite formulation with naloxone, BNX can be abused intravenously. Patients are at risk for standard IVDU infectious complications including cellulitis, abscess, and endocarditis.

KEYWORDS buprenorphine; addiction; infection

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126. Prescription Opioid Abuse or Misuse in Pregnancy Using Poison Center Data

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Background: The prescription opioid epidemic is a significant public health problem that affects all segments of the United States population. One particularly vulnerable population is pregnant women. In this analysis, we compared intentional abuse and misuse exposure pregnant female cases reported to poison centers to non-pregnant female controls to understand differences in opioid exposures.
Methods: The Researched Abuse, Diversion, and Addiction Related Surveillance (RADARS) System collects data from multiple programs to assess prescription drug abuse, misuse and diversion. A retrospective matched case-control study was conducted with intentional abuse or intentional misuse exposures between the ages 15 years to 45 years who reported a prescription opioid to a participating poison center between January 2005 and September 2015. Confirmed non-exposures and death by an indirect report were excluded. Cases were pregnant women who were matched to three non-pregnant female controls on year, poison center, and age (treated as categorical by decades). Pregnant women who did not have three matches were excluded (n = 3). Exposure frequencies for active pharmaceutical ingredients (API; oxycodone, fentanyl, hydrocodone, hydromorphone, morphine, oxymorphone, methadone, buprenorphine, tramadol) were compared for pregnant women and non-pregnant women using conditional logistic regression.
Results: 304 cases and 912 matched controls were analyzed. Teenagers represented 9.2% of the population; the majority were in their twenties (65.5%), followed by 23.7% in their thirties and 1.6% in their forties. Table 1 below displays the percentage of cases and controls exposed to each API. The odds of intentionally misusing or abusing an API differed among pregnant vs. non pregnant for two of the API's: For hydrocodone, the odds among pregnant women was 1.65 (95% CI 1.25 to 2.18) times the odds among non-pregnant women whereas the odds for tramadol among pregnant women was 0.33 (95% CI 0.20 to 0.52) times the odds among non-pregnant women.
Conclusions: In this matched case-control study, pregnancy status was associated with an increased risk of hydrocodone abuse/misuse and lower risk of tramadol abuse/misuse after matching on year, poison center, and age.

	Statistics	Non-pregnant N = 912	Pregnant N = 304	p-value
Oxycodone	N (%)	213 (23.4)	83 (27.3)	0.153
Hydrocodone	N (%)	341 (37.4)	147 (48.4)	**<0.001**
Tramadol	N (%)	172 (18.9)	22 (7.2)	**<0.001**
Methadone	N (%)	88 (9.6)	32 (10.5)	0.651
Buprenorphme	N (%)	69 (7.6)	26 (8.6)	0.564
Morphine	N (%)	34 (3.7)	9 (3.0)	0.523
Fentanyl	N (%)	33 (3.6)	4 (1.3)	0.050
Hydromorphone	N (%)	22 (2.4)	6 (2.0)	0.658
Oxymorphone	N (%)	14 (1.5)	3 (1.0)	0.482

KEYWORDS opioids; pregnancy; poison centers

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127. Two PINACAs, multiple patients: A backcountry EMS mass casualty requiring air and ground transport

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Background: Synthetic cannabinoid (SC) use and toxicity including seizure, agitation, and death has surged in the last few years. SCs pose challenges in the emergency department (ED) and in laboratory analysis, complicated by an ever-changing array of compounds. An as-yet unreported challenge is the prehospital triage and treatment of SC intoxication. We describe an SC mass casualty incident (MCI) in the backcountry requiring helicopter emergency medical services (HEMS) and transport to multiple local EDs.
Case Report: Six previously healthy male prison inmates (age 28-59 years) working as seasonal forest firefighters in the California Sierra Nevada wilderness smoked cigarettes containing "spice" and required emergency medical services (EMS) evaluation. Four patients had tonic-clonic seizures and were transported by HEMS; three required prehospital intubation. A fifth altered patient with abnormal vital signs received ground transport. A sixth altered patient with normal vital signs was transported by private vehicle. A 7th patient required HEMS transport the following night after smoking a remaining cigarette butt. Patients were evaluated at 3 local EDs. All patients were discharged same day or day after presentation without complication. Biological specimens from 5 patients were obtained for testing with liquid chromatography-time-of-flight-mass spectrometry (LC-TOF/MS) (TOF 6230- LC1200, Agilent). Results from 2 patients showed the presence of AB-PINACA, its metabolite AB-PINACA pentanoic acid, ADB-PINACA, and its metabolite ADB-PINACA pentanoic acid.
Case Discussion: AB-PINACA and ADB-PINACA are indazole-based SCs recently placed into Schedule I classification with very limited toxicity reports. Intoxication by a combination of these two compounds has not yet been reported in the medical literature. These SCs, causing simultaneous toxicity in multiple patients, created logistical challenges for EMS. The initial call was for 1 patient. Due to the remote location, EMS contact took 1 hour. On arrival, EMS learned of 5 more patients and that helicopters had been deployed at request of on-site correctional officers. The first EMS crew set up incident command to coordinate and triage. The closest landing zone was 45 minutes away with no radio contact available between HEMS and EMS. Communication occurred via intermittent cell phone contact with frequent signal loss. One HEMS crew went to the incident site to triage helicopter resources; 2 helicopters remained on standby. Patients were ferried by EMS crews to helicopters as needed. One helicopter experienced mechanical problems causing them to abort from the MCI. The time from initial contact to EMS departure from the scene was 4 hours, utilizing 6 ambulances and 3 helicopters.
Conclusions: We report a novel case series of AB-PINACA and ADB-PINACA intoxication in patients presenting with AMS and seizures requiring intubation. Due to multiple symptomatic patients in a remote location, HEMS and ground transport were both required, placing a strain on the local EMS agency.

KEYWORDS Synthetic Cannabinoid; Emergency Medical Services; PINACA

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128. NBOMe intoxication with confirmatory testing

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Background: 25-I-NBOMe (2 iodo 2, 5 dimethoxy N (2 methoxybenzyl) phenethylamine) is an ultrapotent substituted phenethylamine. The threshold dose is 100-200 times lower than that of its parent compound amphetamine. Since first showing up as a "research chemical" in 2009 it has been identified in blotter paper samples and sold as LSD. Reports of excited delirium including fatal cases have been reported after use in this manner. Many of these cases lack confirmatory testing.
Case Report: 18 year-old male presented to the emergency department (ED) for altered mental status after possible overdose. He was at a friend's house until early morning when he was requested to leave due to odd behavior. Approximately 20 minutes later, he was found wandering barefoot in the snow and was brought to the ED. On arrival, he was aggressive with tachycardia to 146 beats/minute, tachypnea to 22 respirations/minute, and had a blood pressure of 147/86 mmHg. He demonstrated loud, pressured speech with incomprehensible sentences. Pupils were dilated to 8 mm and reactive bilaterally. Labs demonstrated a leukocytosis to 22.3 × 103/μL, hyperglycemia at 221 mg/dL, serum creatinine of 1.02 mg/dL, and lactate of 3.8 mmol/L. The patient received 2 mg intravenous (IV) lorazepam with good effect. He was amnestic to much of the preceding events but did subsequently report using "one more tab" of LSD than his friends. Assays for both LSD (negative) and NBOMe (NMS Labs) were obtained and the patient was positive for both 25I-NBOMe and its metabolites.
Case Discussion: We report a case of NBOMe intoxication with laboratory confirmation after reported "LSD" use. Due to its potency compared to other amphetamine-like compounds, small dose changes or double dosing may lead to severe toxicity. Complications of NBOMe intoxication may include sympathomimetic effects, serotonin toxicity, and concomitant environmental exposures or trauma. Treatment involves aggressive sedation with benzodiazepines and IV fluid resuscitation. For severe cases, intubation along with active cooling and serotonin antagonists such as cyproheptadine may be utilized. Our patient presented with symptoms consistent with NBOMe toxicity which responded to benzodiazepines; however, he required hospital admission for management of frostbite.
Conclusions: NBOMe toxicity presented with mixed sympathomimetic/serotonergic features and our patient responded well to supportive. Our patient had concomitant environmental injuries related to the intoxication.

KEYWORDS NBOMe; LSD; designer stimulant

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129. Survey of prescription opioid knowledge and use patterns among ED patients that were prescribed opioid medications

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Objectives: The USA is experiencing a prescription opioid abuse epidemic; however, the role that knowledge about prescription opioids contributes is unclear. Daily opioid consumption of greater than 200 milligram morphine equivalent (MME) has been associated with a 3-fold mortality increase in patients with chronic nonmalignant pain.1 We surveyed Emergency Department (ED) patients with prior opioid prescriptions about overdose risk behavior and knowledge of overdose prevention strategies.
Methods: This was an ED-based survey conducted at one urban, tertiary-care hospital. English speaking ED patients >18 years were screened for inclusion if they had previously been prescribed an opioid medication; non-English speakers and those <18 years were excluded. All patients provided written informed consent and were reimbursed for participation. The survey included the following information: patterns of use, sources of prescription(s), doctor-shopping, preferred opioids, reasons for using, history of substance abuse, history of mental illness, and knowledge of overdose prevention strategies.
Results: 50 patients completed surveys, of whom 60% were female, 64% visited the ED for a pain-related complaint, 56% reported taking opioids daily, and 94% reported taking a prescription opioid before arriving to the ED. All 50 patients reported taking prescription opioids primarily to treat pain, one patient additionally reported taking prescription opioids to treat or prevent withdrawal. 9/50 reported taking >200 MME on an average day. 8/50 patients reported receiving a prescription for an opioid from an emergency physician within the past month. 12/50 reported a history of drug abuse treatment in the past. 3 patients reported a history of opioid overdose in the past. 33/50 were unaware of the existence of out-of-hospital naloxone programs to prevent opioid overdose. Of the 9 patients taking >200 MME daily, 3 patients were aware of out-of-hospital naloxone programs.
Conclusions: In this ED-based survey, the majority of participants were unaware of the existence of important overdose prevention strategies, such as naloxone programs, even among those at risk for overdose due to high daily opioid use. These results suggest a lack education from providers to patients with prescribed opioids, and further highlight the importance of education regarding community naloxone training programs to treat prescription opioid overdose in at-risk patients.

KEYWORDS Opioid-related disorders; Drug abuse; Drug overdose

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130. Naloxone Use after Opioid Pain Reliever Exposures 2000-2015

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Background: Opioid pain reliever (OPR) misuse is common and has been increasing markedly nationwide over the last fifteen years. As opioid overdoses and deaths continue to rise, the use of naloxone to treat these patients has continued to increase. It is not known whether the use of naloxone is influenced by key variables such as opioid type, age, gender, and clinical effects contributing to medical outcome.
Methods: This study aimed to examine the impact of variables associated with cases of naloxone treatment (Naloxone group) vs. no naloxone treatment (None) for prescription OPR exposures reported to a state poison center network during 2000-2015. Demographic information was obtained for age group, sex, and urban vs. rural location. Data gathered included OPR type used, route of exposure, exposure reason, exposure site, and clinical outcome. Descriptive statistics were used, and the 95% confidence intervals (CI) were calculated for select variables.
Results: Of 89,076 total reported OPR exposures, 10,975 (12.3%) received naloxone. The annual percentage of cases receiving naloxone remained stable during this period ranging from 11.5-13.6%. The most common age group receiving Naloxone vs. None was over 20 years (84.8% vs. 68.0%, CI 1.24-1.26). We found no effect conferred from gender (58% female in both groups), urban vs. rural location (80.8% vs. 85.4%), or route of exposure, the latter primarily being ingestion. The prevailing OPRs used in Naloxone and None groups were hydrocodone 54.6% vs. 54.1%, followed by tramadol 7.4% vs. 14.8%, codeine 6.2% vs. 11.4% and propoxyphene 6.3% vs. 7.8%, all differences which were statistically significant. When examining cases deemed as suspected suicide, naloxone was used almost twice as frequently (60.5% vs. 34.2%). Approximately 80% of unintentional exposures in both groups did not use naloxone. Single-agent exposures were 34% vs. 56.6% for Naloxone vs. None. For patients who received naloxone, a significant proportion were already en route to or in a healthcare facility (93.2% vs. 48.6%). Significantly fewer patients receiving Naloxone compared to None were referred by the poison center to seek emergency care (6.6% vs. 16.9%). Finally, clinical outcomes revealed more major effects (17.2% vs. 2.9%, CI 5.54-6.22), and deaths (0.8% vs. 0.2%, CI 3.11-5.21) for Naloxone vs. None groups, respectively.
Conclusions: Naloxone administration after prescription OPR use was associated with age over 20 years, hydrocodone use, suspected suicide, treatment in a healthcare facility, and more major effects and deaths.

KEYWORDS Opioid; Naloxone; Drug Abuse

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131. Inpatient compared to outpatient prescription opioid misusers – What's the difference?

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Background: Few studies have characterized patients who self-referred to outpatient rehabilitation (e.g., Suboxone™ clinics) following misuse of prescription opioids vs. patients who received a medical toxicology consult plus inpatient care due to adverse events following recreational opioid use. Our aim was to describe the demographics, drug use history, medication source, and other characteristics of patients presenting for emergency care after prescription opioid misuse resulting in medical toxicology consultation (Inpatient, or IN) compared to those who self-referred to outpatient medication assisted treatment clinics (Outpatient, or OUT) for rehabilitation.
Methods: The ToxIC Prescription Opioid Misuse subregistry is a prospectively collected, de-identified, national dataset of patients who required hospital admission and a medical toxicology consultation following prescription opioid misuse. Intentional self-harm patients were excluded. We descriptively analyzed medical history, drug source, use and diversion patterns, and other factors that have been shown to increase misuse risk.
Results: Of 110 entries (60 IN, 50 OUT) from 2013-2015, 37 vs. 58% were aged 31-50 years, and 37 vs 26% were female, respectively. PMH included 37 vs 20% reporting a chronic pain syndrome ($p < 0.003$), and 8 vs 48% misused alcohol in the past ($p < 0.001$) for IN vs OUT. Long-term heroin, cocaine, and IV drug use were less common in the IN group vs OUT (8 vs 32% [$p < 0.001$], 7 vs 26% [$p < 0.001$], and 17 vs 74% [$p < 0.001$], respectively). Long-term unemployment was more common in OUT (46 vs 18%). OUT reported anxiety 36%, depression 52%, and a low rate of bipolar or post traumatic stress. Reported reason for use was recreational high (85 vs 0%) and to avoid withdrawal (0 vs 100%) in IN vs OUT groups. Most common agents used in IN vs OUT were oxycodone/hydrocodone 32 vs 18%, methadone 13 vs 0% ($p < 0.01$), buprenorphine 5 vs 30% ($p < 0.001$), and heroin 3 vs 44% ($p < 0.001$), respectively. Use was acute 53 vs 0%, acute on chronic 27 vs 6%, and chronic 0 vs 94% for IN vs OUT, respectively ($p < 0.001$). The majority of OUT patients were insured: 84% Medicare/Medicaid, 8% private, 6% unknown. OUT group initially obtained the drugs by purchase (60%), prescription (12%), and stealing (2%). For OUT, the opioid that led to first use was hydrocodone 40%, heroin 30%, and oxycodone 14%. OUT reported ethanol and drug treatment program use in 38 and 94% respectively. For the OUT group the prescription drug monitoring program was available in 100%, accessed in 88%, and found patients in 84%.
Conclusions: IN patient group was more likely to have chronic pain, seek recreational high, and use hydro-/oxycodone and methadone. OUT patient group was more likely to use ethanol, heroin, and cocaine, had long-term unemployment, was insured, and sought to avoid withdrawal.

KEYWORDS Prescription opioid misuse; buprenorphine; medication assisted treatment

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132. Case Report: 2 5-dimethoxy-4-chloroamphetamine: A Longer Than Expected Trip

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Background: 2,5-dimethoxy-4-chloroamphetamine, (DOC), is a laboratory synthesized hallucinogenic stimulant with both psychedelic and sympathomimetic properties. Although recreational use

of synthetic stimulants is rising, there have only been a handful of reported cases of phenyethylamines. Reports have ranged from seizures to death. Drug formulations come in a wide variety and are often sold and consumed under the false label of more common drugs, such as lysergic acid diethylamide (LSD). When compared to LSD, the relative scarcity of DOC and combined lack of consumer awareness may serve to confound the patient history and management. Furthermore, these newer synthetic compounds cannot be detected on routine urinalysis or hematological screen. Considered alongside the potential for seizure, death, and sympathomimetic toxicity, the awareness and consideration of 2-C class compounds is of clinical importance in critical care settings.

Case Report: The patient initially presented with subjective visual hallucinations, euphoria, altered mental status, tachycardia of 128, and fever of 100.6. Given the history of toxicologic ingestion, fluid bolus and Ativan were administered. Although the tachycardia and fever resolved, he remained symptomatic and his clinical examination was only slightly improved. An EKG was performed and without pathologic changes. The metabolic panel and CBC were returned and demonstrated CPK of 511 but was otherwise normal. Four hours into the visit (roughly 21 hours after ingestion) the patient was still symptomatic. At this time the patient admitted to having taken "DOC". He was kept overnight in the ED with moderate improvement in his symptoms, returning closer to baseline. He was subsequently discharged home with family for observation in stable condition with ultimate symptom duration of 33 hours. Blood and Urine was sent out for analysis and DOC was identified by LC/MS.

Case Discussion: There exists little information about synthetic phenylethylamines in the current medical literature. To date, this is the fourth laboratory confirmed DOC intoxication. In two of the other cases the drug was initially thought to be LSD. In contrast to the other three case reports, our patient did not suffer seizures, status epilepticus, or death. Like two of the reported non-fatal cases, our patient responded well to supportive management and was discharged in stable condition. Questions still remain about the concentration of dose taken, reported by our patient as one paper wafer. Clinically, and similar to other non-fatal case reports, our patient did have objective sympathomimetic findings, which responded to fluid resuscitation and benzodiazepines. In our case, the hallucinatory effects lasted well into the next day following ingestion, roughly 33 hours. When compared to other cases, our patient did not require endotracheal intubation or ICU placement. It is unclear why our patient had fewer complications, but this could be secondary to interaction between Δ(9)-Tetrahydrocannabinol (THC), cannabidiol (patient was THC positive) and CYP2D6 metabolism and the potent anti-epileptic effects of THC.

Conclusions: DOC is a rare and highly toxic synthetic phenyethylamine and should be considered when hallucinatory and sympathomimetic symptoms present together. Given the half lives of LSD (3-5 hours) and MDMA (7-12 hours), symptoms lasting 24hr should prompt consideration of phenylethylamine intoxication.

KEYWORDS DOC; phenylethylamine; toxicology

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133. Can elevated lactate and LDH produce a false positive enzymatic ethanol result in live patients presenting to the emergency department?

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Background: There have been allegations in the courtroom that elevated serum lactic acid in trauma victims can result in a falsely elevated serum ethyl alcohol assay result. Most hospitals utilize an indirect method of ethanol measurement where a serum sample is added to a mix of alcohol dehydrogenase and oxidized nicotinamide adenine dinucleotide (NAD+). This allows any ethanol in the patient's serum to be metabolized to acetaldehyde, and in the process results in the reduction of NAD+ to NADH. NADH is then measured using spectrophotometry. The courtroom allegation stems from the concept that oxidation of lactate to pyruvate by lactate dehydrogenase (LDH) results in the same molar-for-molar reduction of NAD+ to NADH, and could therefore theoretically cause patients with elevated lactate and LDH to have a falsely-elevated ethanol level.

Methods: Patients with elevated lactic acid and LDH levels who presented to a university hospital from April 20, 2015 to December 13, 2015 were identified to provide possible test specimens. Any samples that had elevated lactic acid and LDH levels on retesting and yielded a positive ethanol level were sent for confirmatory gas chromatography testing. If a sufficient amount of serum was available, the sample was used to re-run the lactate and LDH level simultaneously with the enzymatic ethanol assay to eliminate changes that may have occurred during sample storage.

Results: A total of 37 samples were included in the final analysis. Only 4 patients had an elevated enzymatic ethanol level, and all 4 also had a measurable GC ethanol level. The lactate in this dataset ranged from 3.0 to 24.2 mmol/L, with a mean of 6.53 mmol/L (normal value 0.5-2.2). The LDH ranged from 242 to 8838 U/L with a mean of 1695 U/L (nml 122-225 U/L). 20 control samples were run on patients with normal lactate and LDH, none of which yielded a positive enzymatic ethanol result.

Conclusions: This data does not support the contention that an elevated LDH and lactate can yield a false positive serum ethanol result as run by enzymatic ethanol assay in live patients presenting to the emergency department.

KEYWORDS Ethanol; LDH; Lactate

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134. *Withdrawn*

135. False Positive Cocaine Immunoassay Screen in a Patient Taking Levomilnacipran

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Background: Cocaine screening involves urine immunoassays for benzoylecgonine (BE). Immunoassays for BE generally are highly specific, and false positive results are infrequent.
Case Report: Parents of a 17 year-old girl with attention deficit hyperactivity disorder (ADHD) and depression found her confused upon awakening and brought her to an urgent care clinic. An initial urine drug screen (UDS) was "non-negative" for tricyclic antidepressants ("TCA"), tetrahydrocannabinol ("THC"), and methadone ("MTD"). Upon transfer to a pediatric hospital emergency department, she reported hallucinations including formication and hearing animal noises. She was tachycardic, heart rate 132 bpm, and had mydriasis. A repeat UDS immunoassay on a Roche Cobas 8000 was presumptive positive for cocaine, cannabinoids, and amphetamine. Her medication list included amphetamine and dextroamphetamine (Adderall®) and levomilnacipran (Fetzima®). Liquid chromatography with tandem mass spectrophotometry (LC-MS/MS) confirmed the amphetamine, but was negative for benzoylecgonine and cannabinoids. LC-MS/MS also revealed a peak consistent with N-desethylmilnacipran, a metabolite of levomilnacipran. Urine was re-run on a Siemens Dimension RxL Max which produced a signal that did not meet the positive cutoff value. The patient denied cocaine, TCA, methadone, and cannabis use but did endorse taking numerous naproxen-diphenhydramine (Aleve PM®) tablets the previous evening. She recovered without adverse events.
Case Discussion: Levomilnacipran (Fetzima®) is a novel serotonin and norepinephrine reuptake inhibitor approved for treatment of depression. Two different immunoassays were non-negative. However, confirmatory LC-MS/MS and a third immunoassay failed to confirm benzoylecgonine but potentially show a levomilnacipran metabolite. Other false positive results occurred in this case, although the naproxen-diphenhydramine overdose may explain the false positive results for THC and methadone. The remaining false positive for BE is less common, and we believe that levomilnacipran may be the cause.
Conclusions: Because false positive screens for benzoylecgonine are rare, this case justifies further investigation into whether levomilnacipran or its metabolites can create a false positive signal for BE on immunoassay screening.

KEYWORDS benzoylecgonine; false-positive; levomilnacipran

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136. Lamotrigine Causes False Positive Field Test for Methylenedioxypyrovalerone

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Background: 3,4-Methylenedioxypyrovalerone (MDPV) is a designer drug of the phenethylamine class that is structurally and pharmacologically similar to 3,4-methylenedioxymethamphetamine (MDMA), cathinone, and methamphetamine. Since the Synthetic Drug Abuse Prevention Act of 2012 placed MDPV into DEA Schedule I, field tests for presumptive identification of MDPV have been developed for use by law enforcement. We present a case of false positive result for MDPV that was obtained when lamotrigine (LTG) powder was tested by law enforcement officers (LEOs) using a field drug test kit at the scene of a drug overdose.
Case Report: A 16-year-old female was found in an altered state of consciousness in her home by LEOs. She had white powder in her nares, around her lips, in her underwear, and on the floor. LEOs obtained a sample of the powder and tested it using a NARK-II MDPV Reagent test kit, manufactured by Sirchie. The LEOs observed a distinct yellow color, indicating the presence of MDPV. Samples of the material were collected from the floor, her shorts, and her underwear and sent to the state laboratory. In the ED her heart rate was 107 beats/minute, blood pressure 98/42 mmHg, respiratory rate 14 breaths/minute, and temperature 98.4 °F. Naloxone was administered IV without effect. A urine drug immunoassay was negative and expanded urine toxicology drug testing (GC/MS) was ordered. She was admitted with a working diagnosis of MDPV overdose. LEOs reported to the treating team that a suicide note was found, as were empty bottles of LTG. She developed involuntary movements of her four extremities, and became obtunded, requiring endotracheal intubation on hospital day (HD) 2. A serum LTG level was found to be 80.8 ug/mL (therapeutic range =2.5–15 ug/mL). Urine toxicology testing (GC/MS) was positive for LTG. Serial LTG levels were measured; the last level was 4.2 ug/mL on HD 11. Upon regaining consciousness the patient admitted to taking LTG with the intent to harm herself. She was discharged from the hospital on HD 13. The samples of powder collected from her home and clothing were found by state police to be LTG on GC/MS.
Case Discussion: To our knowledge, there are no cases in the peer-reviewed literature describing cross-reactivity of LTG with any presumptive drug analysis reagents. Although these screening tests are known to trigger false positives or negatives, no specific mention of cross-reactive substances are mentioned in the product labeling.
Conclusions: Considering the significantly elevated LTG level and that the forensic laboratory identified three samples of drug material from her home as LTG, we believe that the NARK II MDPV reagent triggered a false positive in this case.

KEYWORDS methylenedioxypyrovalerone; lamotrogine; illicit drug testing

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137. Cobalt toxicity from subcutaneous injection

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Background: Cobalt chloride is currently marketed online as a body building supplement because of its role in hematopoiesis. Previously, cobalt has been reported to cause cardiac, thyroid, neurologic, hematologic and dermal manifestations.
Case Report: A 30 year-old male presented to the Emergency Department (ED) 2.5 hours after injecting cobalt chloride purchased online for body building into his abdominal subcutaneous

tissue. The amount injected was uncertain but he intended to inject 160 mg dissolved in 1 ml of water. He measured the required amount using a mobile application-based scale. The patient developed nausea and vomiting fifteen minutes after injection. His initial vital signs were heart rate 90 bpm, blood pressure 111/78 mm Hg, RR 20/min,T 98 F. Laboratory and electrocardiogram findings were unremarkable except for a blood glucose of 182 mg/dl and a WBCC of 18,000/mm3. 9 hours after injection, his abdominal radiograph did not show any radiopaque bodies, but serum cobalt level was greater than 1600 ng/ml (normal range 0.0-0.9 and <10 ng/ml for Metal on Metal Implants). The patient was given 4 mg ondansetron IV, 10 mg of metoclopramide IV, 40 mg pantoprazole IV, 1 Liter IVF and discharged from the ED. Over the following week, the patient complained of generalized weakness and myalgias that gradually resolved. 6 weeks later, the patient reported small cutaneous nodules at the injection site but felt normal otherwise.

Case Discussion: This is a case of subcutaneous cobalt chloride toxicity that presented with vomiting, diffuse myalgias and fatigue. No neurological or cardiac effects were apparent.

The patient used a mobile application to weigh the dose. The use of this application can lead to hazardous consequences when relied upon for dosing of drugs. Fortunately, to date, the patient has not suffered any serious morbidity. We assume that the cobalt level was obtained post-peak of absorption. Based on his body weight and a estimated Volume of Distribution of 0.93 kg/L, we extrapolate that the amount injected was greater than 96 mg.

Conclusions: This is the first report of toxicity from subcutaneous injection of cobalt chloride with a very high serum level and no serious complication up to 6 weeks from the incident. The use of application-based scales is erroneous and can lead to overdose and complications.

KEYWORDS Cobalt; subcutaneous; toxicity

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138. Diagnostic Tools for Clotting Abnormalities from Green Pit Viper Envenomation, an In-vitro Study

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Background: Green pit viper (GPV) bite is common in Thailand. The GPV venom acts as a thrombin-like substance which consumes fibrinogen. Since the test for fibrinogen levels is not widely available in the country, current practice guidelines suggest using the venous clotting time (VCT) or 20-minute whole blood clotting test (20WBCT) as a standard diagnostic tool. However, performing and interpreting those bedside tests is challenging. A previous study showed that the prothrombin time (PT), a more standardized test, provided the highest accuracy for hypofibrinogenemia, but the study was small and results were questionable. The objectivewas to find the most accurate diagnostic tool for hypofibrinogenemia from GPV envenomation.

Methods: We conducted an in-vitro study. Blood samples were obtained from 26 healthy subjects who were between 20-45 years old and had not been taking any medications during the previous month. We titrated different doses of GPV venom in blood samples of the first subject to find two optimal doses of venom to achieve two target fibrinogen ranges (less than 1 g/L and between 1-1.7 g/L) which were simulated as severe and moderate systemic envenomation. The two optimal venom doses from the first subject were used for the tests of the 25 remainders. For those 25 subjects, we grouped blood samples from each subject into three groups, group 1 with target fibrinogen below 1 g/L, group 2 with target fibrinogen 1-1.7 g/L, and group 3 as a control (no venom added). Blood samples from each group were tested for platelet count, PT, INR (international normalized ratio), activated partial thromboplastin time (aPTT), VCT, and 20WBCT. As fibrinogen levels of less than 1 g/L and 1-1.7 g/L were the target endpoints, we calculated sensitivity, specificity, accuracy, correlation coefficient, and area under the receiver operating characteristic (ROC) curve of those tests.

Results: According to the following normal ranges for our tests: platelet count >100,000 cells/mm3, VCT <15 min, normal 20WBCT, INR <1.2, aPTT <30 seconds, and fibrinogen level >1.7 g/L, we found abnormalities in 0%, 0%, 0%, 3.4%, 22.0% and 23.7%, respectively. PT/INR provided the highest correlation, -0.34 ($p = 0.010$) for PT and -0.33 ($p = 0.010$) for INR, with the fibrinogen level less than 1.7 g/L. Using a fibrinogen level of below 1 g/L, PT/INR revealed the highest area under the ROC curve which was 0.761 (95%CI 0.568-0.954, $p = 0.037$) and 0.764 (95% CI 0.572-0.956, $p = 0.035$), respectively. INR was found to provide the highest accuracy (91.5%) compared with other diagnostic tools for detecting hypofibrinogenemia from GPV envenomation.

Conclusions: PT/INR could be used as a diagnostic tool to evaluate hypofibrinogenemia from GPV envenomation due to high accuracy, high inter-laboratory standardization, and high reproducibility.

KEYWORDS Trimeresurus spp; coagulopathy; viper bite

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139. Serial Nonsense? Clinical Characteristics and Trends in Cases with Serial Carboxyhemoglobins

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Background: Carboxyhemoglobin (COHgb) levels are commonly obtained to evaluate for carbon monoxide exposures though their true prognostic value remains controversial. For unclear reasons, serial COHgb levels are sometimes obtained despite the well-established half-life of COHgb with various degrees of oxygen supplementation and the absurdity of rising COHgb in the absence of ongoing exposure. We sought to evaluate the trends and clinical characteristics of cases where serial COHgb levels were obtained.

Methods: A retrospective review of all patient encounters with at least one COHgb level resulted from 4/2010 – 4/2015 at a quaternary care academic medical center was performed. All charts were individually reviewed and inter-rater reliability assured through training and evaluation of Kappa scores. Data collected included age, gender, pregnancy status, smoking history, month of encounter, admission level of care, administration of oxygen, history of fire or burn, vital signs, presenting symptoms, use of hyperbaric oxygen therapy (HBO), initial pH, troponin, lactate, and COHgb levels. Emergency department (ED) or inpatient cases with serial COHgb levels were identified and the timing of each level was also obtained and delta-times were calculated. Characteristics of patients with serial COHgb results were compared to those

Table 1. Comparison of Serial COHgb vs. Single COHgb cases.

	Serial COHgb (n = 106)	Single COHgb (n = 515)	p-value
Average Age [range]	49.8 (9-88)	47 (1-98)	.3636
Percent male (n)	63.2 (67)	62.0 (321)	.3948
Percent smokers (n)	46.2 (49)	40.5 (210)	.8346
Mean Initial COHgb (range)	9.0 (0.2-45.2)	3.0 (0-40.6)	.3883
Percent Burn Injury (n)	64.2 (68)	50.0 (259)	.0103
Percent Admitted (n)	86.8 (92)	75.3 (390)	.0001
Percent ICU admit [n]	64.2 (68)	35.1(182)	.0001
Percent receiving hyperbaric oxygen therapy[n]	14 (15)	3.5 (18)	.0001
Death (n)	7.5(8)	5.4 (28)	.3675

with a single COHgb result. Statistical significance was calculated using Fisher's exact test and Student's t-test where appropriate.
Results: The search identified 2,213 encounters with a COHgb result. From this group, 624 cases had an associated ED or inpatient encounter. A total of 106 (17%) cases had serial COHgb levels obtained during their ED/inpatient encounter. The mean number of COHgb levels obtained was 2.6 (range 2 - 9). The average initial level was 8.9%, subsequent levels averaged 2.8% at 353 minutes, 1.8% at 663 minutes, 1.1% at 1,095 minutes. The longest interval was 3,807 minutes (2.6 days) to obtain a total of four levels. Table 1 compares the characteristics of the cases with serial COHgb values versus those with a single COHgb value. A total of four patients (3.8%) were identified as having a change in COHgb level from normal (defined as <2% by the institution lab) to abnormal on serial level measurement. The largest interval increase was 1.9%. All four of these patients were current smokers, three were presenting for burn injuries and one was presenting with altered mental status; none of their COHgb levels exceeded 4% at any time and there were no deaths.
Conclusions: In an academic medical center, ordering of serial COHgb levels occurred in over 100 cases in a 5 year period. Despite being more likely to have a burn injury, be admitted the ICU, and receive HBO there was not a statistically significant difference in mortality in those with serial COHgb levels. Four patients were observed to increase from normal to abnormal COHgb levels based on the laboratory standard however the clinical significance of this increase is questionable. This study suggests that there is minimal value in obtaining serial COHgb levels and the practice should be discouraged.

KEYWORDS Carbon monoxide; Carboxyhemoglobin; HBO

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140. Two cases of falsely elevated iSTAT® serum creatinine in moderate to severe acetaminophen poisoning

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Background: Laboratory results play an important role in the diagnosis and management of acetaminophen (APAP) poisoned patients. Laboratory interferences could complicate diagnosis and management. We report two cases of falsely elevated iSTAT® serum creatinine (SCr) in moderate to severe APAP poisoning.
Case Reports: Case 1: A 58-year old female was found unresponsive with several near empty or empty pill bottles, including APAP. Blood was analyzed using the iSTAT1® Handheld Analyzer with a iSTAT® Chem 8+ cartridge and the Roche/Hitachi MODULAR P® analyzer with Cobas® assays. Initial APAP level was 668 mcg/mL. Results were similar between methodologies with the exception of SCr, which was 7.6 mg/dL on iSTAT® Chem 8+ and 1.7 mg/dL on Cobas® CREA. Two and half hours later, repeat SCr was 6.4 mg/dL on iSTAT® Chem 8+ and 1.3 mg/dL on Cobas® CREA. The patient declined over the next few days and expired secondary to complications from fulminant hepatic failure. Case 2: A 22 year-old female presented altered and tachycardic after a suspected overdose of APAP and diphenhydramine. Blood was analyzed using the iSTAT1® Handheld Analyzer with a iSTAT® Chem 8+ cartridge and the Roche/Hitachi MODULAR P® analyzer with Cobas® assays. A 4-hour APAP level was 318 mcg/mL. Results were similar between methodologies with the exception of SCr, which was 1.6 mg/dL on iSTAT® Chem 8+ and 0.6 mg/dL on Cobas® CREA. She made a full recovery and was discharged in her normal state of health a day later.
Case Discussion: Point-of-care iSTAT® SCr was elevated when compared to the Cobas® laboratory SCr in two cases of moderate to severe APAP poisoning. The iSTAT® Chem 8+ package insert lists APAP as a possible positive interferent but does not provide a mechanism or the magnitude of the interaction. This electrochemical assay converts SCr into hydrogen peroxide, which is oxidized on a metal electrode, producing a current proportional to SCr concentration. APAP is readily oxidized and we believe it produced a current on the iSTAT® electrode that was falsely read as SCr. The magnitude and linearity of the interaction is unclear. As it is not widely reported, we believe that this interference is only clinically relevant after large APAP overdoses.
Conclusions: iSTAT® SCr was elevated compared to Cobas® CREA in two cases of moderate to severe APAP poisoning. These differences can be misleading and clinicians should be aware of the possibility in patients with significant APAP poisoning. If an elevated iSTAT® SCr is observed after a significant APAP poisoning, we recommend repeating a SCr using an alternative methodology.

KEYWORDS Acetaminophen; Serum creatinine; Laboratory interference

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141. Cardiac arrest after use of phenylpiracetam and CDP choline

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Background: Phenylpiracetam (phenotropil), is used in Russia to improve cognition. It is marketed online as a psychostimulant. Cytidine diphosphate (CDP)- choline (Citicoline) is prescribed in Europe for cognitive impairement. We report a case of cardiac arrest in a young man who used both substances in addition to cocaine and ethanol.
Case Report: 24 year-old male presented to the emergency department (ED) in cardiac arrest. He was found at home having convulsions by his father who initiated CPR. He had return of spontaneous circulation in the ED. The patient's pattern of drug use is unknown. The father reported finding 3 empty packets of each of the suspected substances at home. Each packet contained 20 capsules of phenylpiracetam and 10 capsules of CDP choline and were delivered by mail on the previous day. ED labs showed a lactate 15.2 mmol/L, Troponin 0.01 ng/ml, creatine kinase 302 IU/L, Creatine 1.1 mg/dl and ethanol 65 mg/dl. CT brain

day 1 was normal but follow-up 12 days later showed bilateral subacute globus pallidus infarcts. The patient improved and was extubated on hospital day 2. His course was complicated by a left arm DVT and left leg compartment syndrome that required fasciotomy. Analysis of his serum and urine against the phenylpiracetam reference standard, identified the substance using liquid chromatography- quadrupole time-of-flight mass spectrometry (LC-QTOF-MS). Additionally, cocaine, benzoylecognine (BE), cocaethylene, mitragynine and 2,4,6- trimethoxyamphetamine (TMA) were also identified in the serum and urine samples. The levels of drugs quantified in the patient's serum are- phenylpiracetam (3098 ng/mL), cocaine (54 ng/mL), benzoylecgonine (4447 ng/mL), cocaethylene (1.5 ng/mL), mitragynine (8.3 ng/ml) and TMA (12. 9 ng/mL). We did not detect any CDP-choline in the patient's samples as it is most likely metabolized to CDP and choline right away. Because both are endogenous metabolites, their measurement would be hard to interpret without the patient's baseline levels.

Case Discussion: Phenylpiracetam has gained popularity as a nootropic that can improve mental function and as a performance enhancer. CDP-Choline is an endogenous substance that is a precursor of phospholipid phosphatidylcholine. Citicoline is the pharmaceutical which is identical to the naturally occurring metabolite. Recently, phenylpyracetam and citicoline have become available online as dietary supplements purported to enhance memory and focus. There are no case reports of toxicity or toxidrome associated with these two substances.

Conclusions: The contributing role of these two supplements to the cardiac arrest is unknown because of the confounding presence of cocaine in serum and urine. The use of these substances should be discouraged because of their potential association with serious adverse effects as suggested in this case.

KEYWORDS Phenylpiracetam; dietary supplement; citicoline

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142. Evaluation of expired breath as a sample source for cocaine screening

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Background: Hospital-based testing for cocaine usually utilizes a urine matrix. Urine collection is non-invasive, but requires time and privacy to collect, and is subject to adulteration and substitution. Typically, urine "cocaine" assay tests for the metabolite benzoylecognine (BE) which is detectable for several days, but is not considered the most active form of the drug. Expired breath serves as an alternative method of specimen collection that allows for rapid, non-invasive collection under direct observation, and can test for cocaine itself. Cocaine detection would reflects the patient's current clinical condition better than BE, which may alter management in patients with excited delirium, acute coronary syndrome, and other conditions. The objective of this study was to examine the concordance between toxicology screening for cocaine in paired urine and expired breath samples from the same patient encounter in the ED.

Methods: This study was approved by the Johns Hopkins University IRB. Expired breath specimens were collected using the Sensabues DrugTrap® device (Sensabues; Sweden) from a convenience sample of 22 patients with existing urine toxicology screening results. After collection, the devices were stored at 4oC until processing. For processing, the sample recovery was performed by removing the collection filter from the device and rinsing twice with methanol, and collecting the eluent. The collected eluent was evaporated to dryness under methanol, and the residue was reconstituted in HPLC mobile phase. Each sample was analyzed by HPLC coupled with high resolution-accurate mass (HRAM) mass spectrometry (MS) using an Orbitrap Exactive instrument from Thermo Fisher Scientific (San Jose, CA). Peaks representing cocaine were identified based on retention time, accurate mass (within 5 ppm tolerance), and the presence of known mass fragments compared to a cocaine standard. LC-HRAM MS results from collected breath specimens were then compared to the corresponding urine result from the patient.

Results: Twenty-two patients were enrolled. Four (18%) had a negative urine toxicology screening test result for cocaine metabolite. Of the 18 (82%) patients with a positive result for cocaine metabolite on urine toxicology screening, 1 patient had positive cocaine detection on expired breath. In this small sample, the sensitivity of breath testing was 5.5%, specificity 100%, positive predictive value 100% and negative predictive value 19%.

Conclusions: We identified cocaine in a breath specimen from an emergency department patient. Overall, the sensitivity of breath testing was low compared to urine testing for BE. Breath testing may not be optimal for identifying BE, which can be detected in urine for 48 hours or longer. The expected concentration of cocaine in breath is not established, so the clinical relevance of this positive result is unclear.

KEYWORDS Cocaine; Breath; Sensabues

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143. Bobbing for Mothballs: An algorithm for identifying mothball composition

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Background: Camphor, naphthalene, and paradichlorobenzene mothballs are difficult to distinguish without packaging. Symptoms and management differ significantly based on the ingested compound. Previous studies have used a variety of techniques including water, 50% dextrose, saturated salt solutions, chloroform, turpentine, radio-opacity, flame color, and melting point to classify mothballs. A simple identification technique using materials readily available in an emergency department is lacking.

Methods: Mothballs made of naphthalene and paradichlorobenzene were tested, along with camphor tablets. Each material was tested both in its intact form and after being fragmented with a hammer to simulate a partially ingested and recovered mothball. Each of these six sample types were then tested by immersion in 40 ml each of eleven different commonly available fluids: sterile water, 0.45% NaCl, 0.9% NaCl, lactated ringers, 5% dextrose in water, 5% dextrose in 0.9% NaCl, 50% dextrose in water, 8.4% NaHCO3, 3% H2O2, 70% isopropanol, and 91% isopropanol. All tests were conducted in standard urinalysis sample cups to replicate commonly available materials in an emergency department. Three toxicologists blinded to the identities of samples and solutions visually evaluated each sample at two different time points. Observations included assessing response to immersion: sink, float, or dissolve.

Results: All three evaluators agreed in their description of 62/66 (94%) of the samples. In all four cases of disagreement, one evaluator described the sample as both sinking and dissolving, while the other two described sinking only. A two fluid algorithm utilizing 50% dextrose and 91% isopropanol was sufficient to distinguish the sample types. Camphor will dissolve in 91% isopropanol while both paradichlorobenzene and naphthalene will sink. In 50% dextrose, both naphthalene and camphor will float while paradichlorobenzene will sink. These results were consistent with

both intact and fragmented mothballs, and were confirmed with a second focused unblinded trial.
Conclusions: Mothball materials of both intact and fragmented samples can be distinguished by immersion in 91% isopropanol and 50% dextrose, with camphor dissolving in isopropanol, naphthalene floating in 50% dextrose, and paradichlorobenzene sinking in both. Limitations of this study include using camphor tablets as a substitute for mothballs given lack of availability.

KEYWORDS Camphor; naphthalene; paradichlorobenzene

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144. Pediatric Dextromethorphan Ingestion Causing False Positive Qualitative Urine Drug Screen for Opiates

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Background: Dextromethorphan is a commonly available non-prescription antitussive. Dextromethorphan and its metabolite, levorphanol, both bear structural similarity to morphine and other opiates. In therapeutic doses, it does not reliably cross-react with the urine enzyme multiplied immunoassay technique (EMIT) for qualitative detection of opiates. Previous literature shows uncorroborated and conflicting evidence that this analog can provoke a false positive result for opiates. We present a case of a massive pediatric exploratory ingestion resulting in a false positive opiate screen.
Case Report: A previously healthy 2 year old girl presented to the Emergency Department after she was found to have ingested an entire bottle of dextromethorphan-containing cough syrup. Initially she complained of a headache and was found to be ataxic with slurred speech. An electrocardiogram, basic metabolic profile, and complete blood count were unremarkable, and serum ethanol, salicylate and acetaminophen levels were undetectable. Her urine enzyme immunoassay (Beckman Coulter UniCel DXC600i, using Opiate 300 Ng reagent) was positive for opiates and PCP, although no other opioid medications were reportedly available in the home. Her comprehensive gas chromatography/mass spectrometry (GC/MS) screen was positive only for caffeine and dextromethorphan. Specific gas chromatography analysis of urine for dextromethorphan (or levomethorphan) returned an undetectably high level, reported as greater than 2000 ng/mL. She was admitted for observation and her drowsiness and gait disturbances improved without intervention over two days, and she was discharge from the hospital.
Case Discussion: We present a case of a pediatric dextromethorphan overdose resulting in false positive for opiates and phencyclidine (PCP) on the EMIT drug screen. Although false positive PCP results are well known in dextromethorphan overdoses, this case demonstrates that very high urinary levels can cause a false positive opiate screen, likely due to structural homology. No other xenobiotics known to cause positive qualitative opiate screen were reported to be available to the child, and no other cross-reactive substances were detected on GC/MS testing.
Conclusions: Dextromethorphan cross-reactivity is well known to cause a qualitative false positive for PCP on EMIT testing, however this case demonstrates that in large ingestions, it can also cause a false positive for opiates on the EMIT drug screen, especially in large ingestions.

KEYWORDS Dextromethorphan; opiates; EMIT

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145. Acute systemic skeletal fluorosis confirmed with radiography and urine fluoride concentration in the setting of 1,1-difluoroethane abuse

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Background: Dust-Off Duster contains the hydrocarbon 1,1-difluoroethane (1,1-DFE). Hydrocarbon abuse is reported in 10% of adolescents and chronic abuse is reported in adults. Chronic exposure to fluorinated hydrocarbons may cause skeletal fluorosis with osteosclerosis, vitamin deficiency, and fractures.
Case Report: 27-year-old male presented to an Emergency Department (ED) with frostbite. The injury occurred 2 days prior after syncope from abusing Dust-Off Duster. He reported using at least 6 cans daily for 11 months. His vital signs and electrocardiogram were nonactionable. Physical exam findings included: grade 2 frostbite on the abdomen, left hand, and right arm, contracted scarring, a hypertrophic nodule on the right elbow, hard anterior nodules on the right tibia, and hard growths on the digits of the right hand. He stated the growths started 5 months ago and were increasing in size. Laboratory assessment was notable for: alkaline phosphatase, 624 U/L; vitamin D, 10 ng/mL; serum fluoride, 0.3 mg/L (0.2-3.2 mg/L); urine fluoride, 52 mg/dL (0.2-3.2 mg/dL); and urine creatinine, 1 g/L (0.3-3 g/L). Imaging included: periosteal bone formation on the bilateral tibia, right phalanges, right radius and ulna, and a focal nodule of bone on the lateral epicondyle of the distal right humerus. He was treated with oral vitamin D and asked to stop abusing Dust-Off Duster. A bone biopsy was scheduled but he was lost to follow-up.
Case Discussion: Skeletal fluorosis is reported after chronic ingestion of fluoride contaminated water and industrial exposure. Findings include joint stiffness, osteosclerosis, osteoporosis, ossification of ligaments, and vitamin D deficiency. Rapid skeletal fluorosis is not well reported but is associated with fluorinated hydrocarbon abuse. Our patient was using at least 6 cans of 1,1-DFE daily for 11 months. Imaging demonstrated periosteal new bone, which coincided with his use history. His serum fluoride was not elevated but was collected four days after his last use. His urine fluoride was elevated and consistent with an exogenous exposure. His low serum concentration of vitamin D and elevated alkaline phosphatase are consistent with fluoride toxicity. 1,1-DFE serum concentrations and quantitative bone ash fluoride were not obtained.
Conclusions: Skeletal fluorosis is uncommon in the United States, but rapid skeletal fluorosis is associated with fluorinated hydrocarbon abuse. A serum fluoride concentration and 24-hour urine fluoride and creatinine should be obtained in patients with suspected skeletal fluorosis. Bone biopsy can differentiate skeletal fluorosis from malignancies or vitamin deficiencies. Management is supportive with discontinuation of hydrocarbon use, electrolyte replacement, and correction of vitamin deficiencies.

KEYWORDS Hydrocarbon abuse; fluorosis; osteosclerosis

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146. Castor Bean Daiquiri: Unsuccessful Suicide Attempt

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Background: Categorized as a Category B biothreat agent, ricin toxicity can occur via inhalation, injection or ingestion due to endothelial cell damage, inhibition of protein synthesis and cellular death. When ingested, toxicity depends on the amount and level of mastication of the seeds. Significant gastrointestinal (GI) symptoms, including nausea, vomiting, abdominal pain and diarrhea, can present within 6 hours. Electrolyte imbalance, shock and multi-organ failure may occur. Recipes for potentially lethal preparations and purchasing sites are readily available on the internet.
Case Report: A 40-year-old male with a history of severe depression and alcohol abuse presented to an emergency department via EMS. He admitted to his ex-wife that he had attempted suicide 2-3 hours prior to arrival. A self-purported "botany student", he purchased castor beans on the internet and ingested 50 seeds ground in a blender, mixed with ethanol and strawberries. On presentation, he had severe abdominal pain and diarrhea and was given activated charcoal. His abdomen was soft but tender to palpation. Vital signs were normal. Laboratory analysis was remarkable for an anion gap of 19, lactic acid: 3.9 mEq/L and ethanol: 0.272 g%. Electrolytes, renal function and transaminases were normal. Admitted to the ICU, the patient continued to have profuse diarrhea and fluid and electrolyte abnormalities; he was anuric by day 2. Supportive care included aggressive IV fluid replacement, sodium bicarbonate, electrolyte replacement and IV hydromorphone. On day 3, the patient developed frank bloody bowel movements (BMs) with mucosal sloughing. CT scan of the abdomen and pelvis revealed diffuse bowel wall thickening of the entire small bowel with associated mesenteric venous engorgement. Pneumatosis and free intraperitoneal air were absent. At this point, 1 liter of normal saline was administered whenever the patient had a BM $>$500 cc. The patient's blood count remained stable and his bloody BMs resolved by day 5. He remained in the ICU for 7 days and was transferred to a medical floor for an additional 6 days. Repeat CT showed significant improvement in bowel wall thickening and he was eventually admitted to Psychiatry. Serial urine ricinine measurements revealed a peak of 4,030 mcg/L on day 1 declining to $<$0.3 mcg/L by day 16. The poison center provided consultation throughout his hospitalization.
Case Discussion: To our knowledge, this is the first case report of a deliberate ingestion of 50 castor bean seeds. Consistent with published data, GI symptoms predominated. It is hypothesized that the quantity as well as alcohol extraction of ricin may have contributed to early symptom onset. Early presentation and aggressive supportive care likely contributed to survival.
Conclusions: Ingestion of macerated castor bean seeds caused significant morbidity with survival, as documented by history, symptomatology and toxicological analysis. Castor beans are readily available as ornamental or native plants, and can be obtained via the internet, as are recipes for liberating ricin, a biothreat agent. Ricin should be considered in the differential diagnosis of unknown exposures in patients with excruciating abdominal pain and gastroenteritis. With aggressive supportive care, a favorable outcome is likely.

KEYWORDS Ricin; Suicide; Biothreat Agent

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147. Hydrogen cyanamide poisoning; rare but serious, case study

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Background: Although suicidal poisoning by pesticides is very common, it was the first time to face this type of toxicity in our national toxicology center; it was a very serious case.Hydrogen cyanamide poisoning is one of the agricultural pesticides that may be used in our country with no reported cases of acute or chronic toxicities. Hydrogen cyanamide causes severe irritation the eyes, skin, and respiratory tract. Also it causes severe vomiting, dyspnea, hypotension, and confusion .Some studies reported that chronic toxicity of hydrogen cyanamide in rodents causes cellular degeneration in the testes and tissue changes in the ovaries, urinary bladder, kidneys and liver.
Case Report: 34 years male patient presented to our national toxicology center in 31 Jan. 2015 at 6:30pm with history of suicidal ingestion of unknown amount of DORMEX (hydrogen cyanamide) since 2 hours.There was history of repeated vomiting and diarrhea.On examination, the patient wasn't conscious with Glasgow Coma Scale GCS 8, blood pressure was 100/60, pulse 110bpm regular, respiratory rate 30/min, oxygen saturation was 86% (97% on T-tube), pupils was bilaterally constricted (looks like pin point). CVP was average chest examination shows bilateral coarse crackles. The remaining physical examination did not reveal any significant findings. His laboratory parameters on admission were;random blood sugar: 183mg/dl, SGOT: 22U/L, SGPT: 28U/L, serum urea: 42mg/dl, serum creatinine: 1.2mg/dl, HB: 14g%, TLC: 13000, PLT: 268000, PT 13.8, INR: 1.24. ABG shows metabolic acidosis (PH 7.24, PCO2 38, HCO3-14) serum sodium 136 mmol/l, potassium 4.1mmol/l. pseudocholine esterase enzyme level was normal. As the patient was drowsy, he was intubated and connected to T-tube which was connected to oxygen source (10L), IV fluids were given to correct hypovolemia. Atropine was given.Gastric lavage was done, activated charcoal (80g)was given via ryel tube.Sodium bicarbonate (25 mEq/h intravenously) was given to correct the metabolic acidosis then ABG was repeated showed slight improvement,so bicarbonate was given accordingly and adjusted by repeated blood gas analysis.At 3 am, the patient was shocked and CVP dropped .Dopamine was started at 5 μg/kg/min, and noradrenaline 5 μg/kg/min, then he was stabilized. Suddenly at 8 am patient was arrested. Cardio pulmonary resuscitation was started, adrenaline (1 mg IV) and atropine (1mg IV) were given, followed by intravenous sodium bicarbonate (50 mEq) adrenaline and atropine were repeated again twice. In spite of all these measures, the patient died.
Case Discussion: Although insecticide poisoning is frequent in our country, it's the first time to admit a case of hydrogen cyanamide poisoning. the clinical presentation of the patient was similar to organophosphate poisoning, so atropine was given on admission till pseudo-choline esterase level was done. although these cases are rare, this poison is very dangerous and the patient deteriorated very rapidly in spite of supportive measures.
Conclusions: This case may be the first reporting hydrogen cyanamide ingestion with a suicidal intent, leading to persistent metabolic acidosis, refractory shock and death. So more studies are needed to know the cause of rapid deterioration and how to save life as hydrogen cyanamide has no antidote yet.

KEYWORDS Hydrogen cyanamide; agricultural pesticides; suicidal poisoning

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148. Don't cut it short: NAC therapy for acetaminophen toxicity in the setting of penetrating trauma

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Background: Despite the availability of an effective antidote, acetaminophen (APAP) toxicity continues to be implicated in severe morbidity and mortality. A 21-hour course of n-acetylcysteine (NAC) usually prevents APAP related hepatotoxicity, although the infusion may need to be prolonged due to delayed absorption – such as with opioid and anticholinergic co-ingestions. We present a case of delayed absorption of APAP following penetrating abdominal trauma.
Case Report: A 25 year old female arrived to the emergency department with multiple stab wounds. She had reportedly been forced to ingest an unknown amount of APAP and then was stabbed by her now deceased boyfriend as part of a suicide pact. On arrival, her exam revealed a wound to the upper abdomen. Initial labs revealed an APAP concentration of 211.7 mcg/mL, normal AST, creatinine, pH, and INR. Two hours after arrival, NAC was started and she was taken to the operating room for repair of her perforated viscus. A 21-hour NAC protocol, 300mg/kg total in three different bags, was continued in the ICU. An APAP concentration 7 hours after ingestion was 209.1 mcg/mL, followed by 16, 30, and 38-hour levels of 93.8, 15.3, and 13.9 mcg/mL, respectively. Following the initial 21-hour NAC, it was continued at 6.25 mg/kg/hr. No additional APAP was given to the patient, and she had nothing by mouth. 58 hours post presentation, the APAP level rose to 60.9 mcg/mL. With ongoing NAC, 80 and 97 hour APAP levels were 18.4 and <10 mcg/mL, respectively. NAC was discontinued. AST, INR, creatinine all remained normal.
Case Discussion: This case describes delayed absorption of APAP following a period of down-trending levels in the setting of penetrating abdominal trauma. Given that the patient was in a closely monitored setting it is unlikely that there was surreptitious ingestion of APAP. More likely the delay was due to decreased gut motility, which was due to a combination of ileus from intestinal trauma with subsequent repair along with intravenous (IV) opioids; the patient was receiving opioids until 61 hours post ingestion. Presumably, gut recovery and withdrawing the opioid led to more normal motility; resumption of bowel sounds was noted at hour 75. Alternatively, the patient could have taken a sustained release product or co-ingested an opioid or anticholinergic agent along with the APAP.
Conclusions: This case report suggests that APAP absorption may be delayed in the setting of penetrating abdominal trauma due to trauma-induced and post-operative ileus along with high-dose IV opioids. Relying on down-trending levels to discontinue NAC therapy in these patients rather than awaiting an undetectable level may lead to failures of NAC therapy.

KEYWORDS Acetaminophen; n-acetylcysteine; delayed absorption

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149. Death from Salt and Baking Soda Ingestion

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Background: Table salt and baking soda ingestions are described in the literature independently. Reports include gastrointestinal side effects, metabolic derangements, and rarely fatality. We report a unique clinical scenario of a patient presenting after coingestion of sodium chloride and sodium bicarbonate resulting in fatality.
Case Report: A 57-year-old female with an acute tramadol overdose presented to the Emergency Department after ingesting large quantities of table salt and baking soda dissolved in water to induce emesis. On arrival she had stable vital signs, normal mentation, and a benign physical exam. The patient's mental status deteriorated and she experienced a tonic-clonic generalized seizure, which resolved spontaneously. At that time her serum sodium was noted to be 168 mEq/L and her bicarbonate was 43 mmol/L. Seizure activity resumed shortly after and she was intubated, intravenous lorazepam, phenobarbital, and propofol were administered without cessation of seizure activity. Her serum sodium at that time was determined to be >175 mEq/L and bicarbonate was >48 mmol/L. No dilutions were performed. She received 3 hours of hemodialysis and demonstrated a transient decline in her serum sodium concentration, however no further hemodialysis was performed. The primary team opted for slow correction of serum sodium. The patient developed cerebral edema and diabetes insipidus. A repeat CTA was performed 48 hours after ingestion and revealed progression of cerebral edema with tonsillar herniation and lack of significant blood flow in the intracranial vessels. The patient was declared brain dead on hospital day 4, care was withdrawn and the patient expired.
Case Discussion: This patient developed cerebral edema with intracerebral hemorrhage resulting in brain death secondary to hypernatremia after ingestion of sodium chloride and sodium bicarbonate. Review of the literature suggests that acute hypernatremia should be treated with aggressive correction of electrolytes. Therapeutic options include hemodialysis and aggressive free water administration.

	HD O:		HD 1:				HD2:	
	1222	1745	2113	0142	0638	1416	0344	1008
Sodium mmol/L	168	>175	>175 Dialysis	161	170 DDAVP	167	153	148
Potassium mmol/L	2.6	2.3	2.0	2.4	1.8	1.9	2.3	2.8
Chloride mmol/L	119	130	130	107	113	110	112	111
Bicarbonate mmol/L	43	>48	48	46	>48	>48	37	30
Anion Gap	9	–	–	10	7	4	6	10
Calcium mg/dl	9.3	8.8	8.7	8.4	7.7	7.4	7.1	7.4
Blood Urea Nitrogen mg/dL	10	14	16	8	11	11	10	12
Creatinine mg/dL	<0.5	.9	1.2	.8	1.1	1.0	.7	.7
Glucose mg/dL	156	219	172	177	192	164	157	113

	HD 0		HD 1		HD 2
	1733	1826	0114	0608	0845
PH (7.35-7.55)	7.23	7.35	7.47	7.49	7.38
PAC02 (35.0-45.0)	147.0	131.0	70 2	74.0	60.3
PA02 (SO* 001	219	219	67	86	64
HC03 (22.0-24.0)	59.4	70.3	50.3	55.2	34.9
BE	24 6	35.7	21.5	8.5	5.5

Conclusions: Optimal therapy for acute hypernatremia from exogenous sodium ingestion requires rapid correction. It seems the most practical approach would involve aggressive free water administration with serial electrolyte assessment.

KEYWORDS sodium chloride; sodium bicarbonate; hypernatremia

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150. Death 10 months after long-acting anticoagulant rodenticide ingestion

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Background: Long-acting anticoagulant rodenticides (LAARs) are available in large quantities from commercial stores. Intentional exposures may result in significant morbidity or mortality if not properly diagnosed, treated, and followed. We report a case of a 48-year-old female with schizophrenia who ingested a LAAR and died from a head injury that occurred 10 months after the fact.
Case Report: A 48-year-old female with a history of schizophrenia and LAAR ingestion 10 months prior was brought to the emergency department (ED) by ambulance due to worsening mental status after hitting her head. Three hours earlier, the patient smoked marijuana and subsequently developed severe dizziness that resulted in a fall. She became lethargic and had an episode of bloody emesis, so the family called emergency medical services. The patient was able to climb onto the stretcher at home but was unresponsive on arrival to the ED. Her pupils were unequal and nonreactive. Due to her altered mental status and concern for airway protection, rapid sequence intubation with succinylcholine and etomidate was performed. After intubation, she had a heart rate of 49 beats per minute, blood pressure of 183/96 mmHg, and temperature of 94.4 °F. Laboratory studies were notable for PT >100, INR >10, and PTT >200. The patient received IV vitamin K 5mg but no fresh frozen plasma (FFP). Computed tomography of the head revealed a large intraparenchymal hemorrhage involving the left temporoparietal lobe with brainstem compression and diffuse cerebral edema. She was transferred to our facility for neurosurgery consultation. On arrival, she received IV vitamin K 10mg and 2 units of FFP; repeat laboratories were PT 33, INR 3.4, and PTT 39.4. The neurological examination demonstrated a complete loss of cranial nerve reflexes. She suddenly became hypotensive (systolic to the 40s), so 2 units of trauma blood and 2,500 units of 4-factor prothrombin complex concentrate were administered for a suspected gastrointestinal bleed. The patient stabilized after this and was admitted to the unit. Further examination revealed brain death and life support was withdrawn the next day.
Case Discussion: Outcomes after LAAR overdose are generally favorable, but must often be followed by long-term therapy with large doses of oral vitamin K. Our patient had a history of LAAR ingestion that required multiple hospitalizations over a 10 month period. She was finally stabilized on oral vitamin K 50mg daily. However, the family reported she was noncompliant due to Medicaid's refusal to cover the medication. The average cost is approximately $24,000 for 300 vitamin K 5mg tablets.
Conclusions: Long-term treatment with vitamin K is expensive and may influence medication compliance.

KEYWORDS Compliance; Antidote; Rodenticide

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151. Persistently Elevated Serum Salicylate Concentration (SS) in an Adult Patient Treated with Renal Replacement Therapy

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Background: Salicylates have erratic absorption from the gastrointestinal tract. This case report describes persistent elevations in SS despite multiple six-hour sessions of intermittent hemodialysis (IHD) and 24 hours of continuous renal replacement therapy (CRRT).
Case Report: A 59-year-old man was taken to the emergency department (ED) after being found down in a park. The patient endorsed ingesting ethanol and alprazolam. He has a past medical history of depression and ileocecectomy. In the ED, the patient would not follow commands. Initial vital signs were BP 147/88, HR 80, T 38.1 C, and RR 26. Serum creatinine was 1.6 mg/dL, glucose 62 mg/dL, and potassium 4.3 mmol/L. Venous blood gas was pH 7.36, PaCO2 47.6 mmHg, PaO2 15 mmHg, HCO3 26.3 mmol/L, and lactate 2.1 mmol/L. Chest x-ray showed left-sided lung consolidation consistent with presumed aspiration pneumonia. Additional labs revealed positive benzodiazepines in the urine, a negative serum ethanol, and a SS of 57 mg/dL. The patient received naloxone 0.4 mg, a saline bolus, one ampule of dextrose, and sodium bicarbonate infusion in D10, as well as vancomycin, azithromycin, and ceftriaxone. The patient's clinical decompensation prompted intubation with high minute ventilation requirements. The patient received 6 hours of IHD without improvement of SS (55 mg/dL). Testing the dialysate effluent verified proper functioning of the IHD machine. The patient's mental status improved after dialysis. He was given activated charcoal by orogastric tube to decrease further absorption, which he later vomited. A repeat SS was 66 mg/dL, 6 hours post the initial dialysis session, and an additional 6 hour IHD session was performed. The patient was extubated on the 3rd day, and SS decreased from 40 mg/dL to 28 mg/dL with the third session of IHD. IHD was discontinued and CRRT started on day 4 for 24 hours. After CRRT was discontinued, SS was 8 mg/dL. SS increased to a peak of 22 mg/dL, 21 hours after CRRT had been stopped, but declined to 16 mg/dL without any further therapy on day 5. SS was undetectable on day 7. The patient later admitted to ingesting two bottles of aspirin (unknown strength) and a bottle of cough syrup in a self-harm attempt.
Case Discussion: This patient was treated with three 6-hour IHD sessions and 24 hours of CRRT. The persistent elevation in SS is representative of erratic absorption and may indicate a potential bezoar. The patient's ileocecectomy may have played an unknown role in variable salicylate absorption.
Conclusions: This case illustrates an example of erratic salicylate absorption, which persisted despite – and following – multiple courses of dialysis and continuous renal replacement therapy.

KEYWORDS Salicylate; Hemodialysis; Erratic absorption

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152. Coma and Respiratory Arrest following Self Poisoning with Veterinary Pentobarbital

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Background: Although uncommon, patients may obtain veterinary medications for the purpose of self poisoning. We present a case of self poisoning with veterinary pentobarbital.
Case Report: 32 yo female with a history of depression was brought in from home after she admitted taking an animal tranquilizer to her spouse. En route to the Emergency Department (ED) by private vehicle, she became unresponsive. On ED arrival, HR was 91 beats per minute, BP was 80/55 mm Hg, temperature was 100.0 F and she had agonal respirations. On exam, she was deeply comatose. Pupils were 4mm and reactive. Her tone was flaccid without response to pain. She was emergently intubated for airway protection. Her initial laboratory results were remarkable for positive barbiturates on urine drug screen, bicarbonate 17 mmol/L with an other wise normal comprehensive metabolic panel, and lactate 2.2 mmol/L. Toxicology testing showed negative acetaminophen, salicylate, ethanol, phenobarbital, isopropranol, methanol, and ethylene glycol levels. In the patient's belongings, the spouse found an empty bottle of veterinary pentobarbital, 100 ml of 6.3% liquid containing 6 grams of pentobarbital. A serum pentobarbital level sent the day of admission returned at 21.6 mcg/mL (therapeutic 1-5 mcg/mL).
The patient's clinical course was complicated by aspiration pneumonia. Her mental status gradually improved and she was extubated on hospital day #4 and discharged to psychiatry on hospital day #7. She reported that she had researched suicide online and had purchased the veterinary pentobarbital from an internet source.
Case Discussion: Veterinary pentobarbital is obtainable over the counter in Mexico, where it is sold inexpensively for the purpose of euthanizing a sick pet. "Suicide tourism" has been publicized and refers to travel to Mexico in order to purchase this product for intentional self poisoning. Less well publicized is the internet availability of this agent.
Conclusions: We present a case pf coma and respiratory arrest following poisoning with veterinary pentobarbital obtained from a mail order website. Practitioners should be aware of the availability of veterinary pentobarbital which may be used for self poisoning.

KEYWORDS Pentobarbital; Veterinary Agent; Suicide Attempt

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153. Lamotrigine Toxidrome of Rapidly Alternating Somnolence and Agitated Delirium

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Background: Lamotrigine is a relatively new antiepileptic. There is limited data on lamotrigine toxicity. Few published cases report sedation, respiratory depression, cardiac conduction disturbances, ataxia, tachycardia, hypotension or paradoxical increased risk for seizures. This is the first case series to describe lamotrigine toxicity.
Case Reports: This is a case series of nine patients who presented after supratherapeutic lamotrigine ingestions. All were treated at a tertiary care hospital with bedside management by medical toxicologists. Ages ranged from 20 months - 45 years old and 66.7% were female. All patients had confirmed lamotrigine ingestion with either a quantitative serum or qualitative urine testing. Three patients had isolated lamotrigine ingestions and the remaining patients had coingestants. All patients exhibited a similar toxidrome characterized by somnolence with intermittent brief episodes of agitation. Agitated episodes were described as "thrashing" with myoclonus. Sinus tachycardia was the most common vital sign abnormality occurring in 66.7% of patients on arrival or early in the hospital course. One patient required a vasopressor briefly for hypotension after being started on fentanyl and propofol for sedation after intubation. Two patients had associated rhabdomyolysis with total CPK elevations in the 3000s IU/L which was attributed to increased muscle activity. Lamotrigine toxicity persisted for 30-108 hours from estimated time of ingestion. Larger ingestions correlated with longer periods of toxicity. One patient ingested 6-8 grams of lamotrigine sustained release formulation and had alternating somnolence and agitation for 108 hours. Lamotrigine serum level peaked at 74.6 mcg/mL at 54 hour post-ingestion which was likely due to the sustained release formulation and continued gastric absorption. All cases of agitation were controlled by benzodiazepines alone. Two patients required intubation for airway protection.
Case Discussion: This case series describes a toxidrome characteristic of lamotrigine exposures with alternating somnolence and agitated delirium. The myoclonus during agitation was unique and can easily be confused with seizures, but the patients were conscious and distractible making seizures unlikely. An EEG was performed for one patient and confirmed no seizure activity. A limitation of this study is that most patients had ingested additional medications. However, despite various coingestants, the syndrome in all cases was similar and lamotrigine remained the predominant ingested drug.
Conclusions: Supratherapeutic lamotrigine ingestions are associated with a characteristic toxidrome of rapidly alternating periods of somnolence and agitated delirium that is effectively managed with benzodiazepines alone.

KEYWORDS lamotrigine; toxidrome; agitation

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154. Carbon Monoxide Poisoning from Detergent Suicide Attempt

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Background: Detergent suicide is a term that describes the process of combining chemicals to produce toxic gases in order to commit suicide. This report describes a unique case of attempted detergent suicide resulting in carbon monoxide poisoning.
Case Report: A previously healthy 21-year-old woman was found unresponsive inside a running car in an open area. There were no tubes piping exhaust into the car. However, there were multiple bottles containing a yellowish fluid, some labeled "formic acid." The officer who initially found the patient slammed the door closed after smelling a strong odor. The patient was removed from the car by EMS and transported to a nearby emergency

room while being bagged to support respiration. The ED physician was preparing for intubation when the patient became more responsive and alert. The patient was moderately tachycardic but normotensive with normal pulse oximetry. There was no evidence of oropharyngeal burns or caustic effects to suggest oral acid ingestion or inhalation.
Initial lab work, including workup for possible drug overdose, was remarkable for a carboxyhemoglobin level of 38%, ABG with elevated lactate at 6.3 mmol/L and decreased bicarbonate level at 17 mmol/L. The patient was placed on a non-rebreather mask, later receiving hyperbaric oxygen therapy prior to admission to the ICU. Overnight the patient did well and had no apparent medical sequelae from the suicide attempt. The next day, she was transferred to the floor for psychiatric evaluation.
Case Discussion: Detergent suicide is a recent phenomenon in the United States. Originating in Japan, the earliest cases of detergent suicide were reported in the U.S. in 2008. Generally this involves mixing an acid with sulfur-containing compounds to produce hydrogen sulfide gas. A much less common technique of detergent suicide, as described in this report, involves mixing formic acid and sulfuric acid. This causes a dehydration reaction that converts formic acid into carbon monoxide (CO). When performed in enclosed spaces, this can result in severe CO poisoning. Odorless CO can also pose potential serious danger to first responders, though the strong odor of reagents can raise suspicion. Treatment follows standard algorithms for CO poisoning, primarily oxygen and supportive care with possible hyperbaric oxygen therapy in severe cases.
Conclusions: We report an unusual attempted suicide resulting in CO poisoning using a combination of acids in a closed vehicle. Medical providers should recognize this technique in order to treat it safely and effectively.

KEYWORDS Detergent Suicide; Carbon Monoxide Poisoning; Formic Acid

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155. Take your consults with a grain of salt

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Background: Hypernatremia is generally caused by a lack of total body water in relation to total body solute or an excessive intake of sodium. When dealing with the latter cause, especially in debilitated individuals, one has to consider accidental versus deliberate administration. A rapid rise in sodium can cause severe health effects such as seizures, intracranial hemorrhage, movement disorders, and death. A review of the literature reveals a multitude of case reports of confirmed salt poisoning in pediatric patients all with a similar presentation including a greater than 2% fractional excretion of sodium in conjunction with a high urine osmolality in excess of 400 mOsm/L and weight gain.
Case Report: A toddler presented to the hospital for persistent diarrhea and a sodium of 152 mmol/L with a complicated past medical history. Toxicology was consulted on hospital day 10 for concerns of serotonin syndrome. A review of the patient's chart revealed a rise in sodium from 139 to 206 mmol/L, a chloride level of 188 mmol/L, and a pH of 7.16 over 26 hours. The child's symptomology included staring episodes, acute mental status changes, and lower extremity stiffness. Prior to the sharp sodium rise, continuous electroencephalography (EEG) was performed and no obvious epileptiform discharges were found which lowered the suspicion for seizure at that time. Despite the patient being on serotonergic medications (ondansetron, sertraline, and trazodone), serotonin syndrome was ruled out. A free water deficit calculation determined that a deficit of 4.64 liters would be needed to account for a sodium of 206 mmol/L which was unlikely given his estimated total body water was 9.8 L; thus, an exogenous source of sodium became the primary explanation of his rise in sodium and movement disorders. A urine osmolality of 610 mOsm/kg, random urine sodium level of 280 mmol/L, and a fractional excretion of sodium above 18% confirmed an exogenous source. A follow-up EEG and MRI endorsed massive edema and brain death two days after the rapid spike in sodium. Withdrawal of care followed and the patient was pronounced dead.
Case Discussion: A 67 point rise in sodium over the span of 26 hours is highly suspicious of exogenous supplementation. A fractional excretion of sodium greater than 2% and a urine osmolality greater 400 mOsm/L are indicative of exogenous sources of sodium in presence of intact renal free water conservation.
Conclusions: This case highlights the findings of sodium chloride toxicity and the utility of laboratory values to distinguish between an endogenous versus exogenous culprit.

KEYWORDS Hypernatremia; Salt; Sodium Chloride

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156. Toxicology Results for Alcohol and Drugs of Abuse in Suicide and Motor Vehicle Crash Decedents Ages 18-54

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Background: Research has often considered the presence of alcohol in major causes of injury death including suicide and motor vehicle collisions (MVC), along with occasional examination of drugs of abuse, but the co-presence of alcohol and drugs of abuse has rarely been considered in injury research, providing the focus of the current analyses.
Methods: The sample consists of individuals in New Mexico ages 18 to 54 that died in 2012 by suicide (excluding poisoning) or MVC (including operators of motor vehicles and passengers but excluding pedestrians and bicyclists). All decedents were examined at the state's centralized medical examiner office based in Albuquerque. Data are based on information abstracted from the medical examiner files including toxicology results and demographic variables. Toxicology results were used to code the presence of alcohol based on BAC result >0.001 g/dl, and the presence of one or more commonly abused drugs including cocaine, opiates (morphine, oxycodone, etc.), or amphetamine/methamphetamine, yielding a 4-category alcohol/drug abuse variable of primary interest: alcohol plus drug, alcohol without drug, drug without alcohol, no alcohol or drug (reference).
Results: There were 264 suicides and 195 MVCs during the study period, with 34 suicides and 34 MVCs excluded due to missing or out-of-range data. An additional 45 poisoning suicides were removed due to the potential that illicit drugs were deliberately taken to hasten death, yielding 185 suicides and 161 MVCs for analysis. After adjustment for age, sex, and race/ethnicity, the combination of alcohol plus drug was more likely among suicides than MVCs: adjusted odds ratio, AOR (95% Confidence Interval, 95% CI) = 4.33 (1.70, 11.03). Suicides did not differ from MVCs in

the presence of alcohol without drug, AOR (95% CI) = 1.22 (0.74, 2.00), or drug without alcohol, AOR (95% CI) = 1.03 (0.37, 2.88). Exploration of specific drug-alcohol combinations showed that all 17 (100%) suicide decedents with cocaine present were also positive for alcohol, a pattern of co-ingestion that was not observed with either amphetamine/methamphetamine or opiates.
Conclusions: The results suggest that the combination of alcohol plus one or more drugs of abuse may be especially likely in suicide compared to MVC, and that co-ingestion of cocaine and alcohol may be particularly important. Potential explanations include that when cocaine is metabolized in the presence of ethanol it forms cocaethylene which is psychoactive, and therefore co-ingestion of cocaine and alcohol may be an effort to potentiate euphoria; and that the depressed state produced by withdrawal from cocaine may lead to an effort to self-medicate with alcohol, a scenario that may be especially important in suicide in light of the central role of depression.

KEYWORDS Suicide; death; substance

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157. Severe serotonin syndrome after citalopram overdose in a slow metabolizing patient

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Background: Citalopram is a selective serotonin reuptake inhibitor used for depression. Metabolism is primarily through CYP3A4 and CYP2C19; activity of the latter can vary depending on genetics. Although rare after single agent exposure, large citalopram ingestions can lead to serotonin syndrome with mental status changes, autonomic hyperactivity, and neuromuscular abnormalities. We report a case of citalopram overdose in an intermediate CYP2C19 metabolizer complicated by severe, protracted serotonin syndrome.
Case Report: 25 year old female presented to emergency department (ED) 7 hours after ingesting 760 mg of citalopram. Patient was noted to be anxious and tremulous with tachycardia to 160 beats/minute which progressed to generalized tonic-clonic seizure activity shortly after arrival. Patient was intubated and started on midazolam and fentanyl infusions. EKG showed QT prolongation and IV magnesium sulfate was given. Initial labs were significant for leukocytosis (18.7 x 103/μL), low serum bicarbonate (18 mmol/L), and elevated anion gap (17). Comprehensive urine drug testing was positive only for citalopram. Toxicology was consulted and she was admitted to intensive care unit (ICU) for serotonin syndrome. On evaluation in the ICU, she was hyperthermic to 38.5 °C with severe rigidity and difficulty eliciting clonus. Recommendations included discontinue fentanyl due to serotonergic effects, increase benzodiazepines for neuromuscular control, start cyproheptadine and phenobarbital in escalating doses for additional control. Due to persistent rigidity, pentobarbital was initiated on hospital day 3 and continued until day 7 with improvement in symptoms. CK peaked approximately 42 hours post ingestion at 7671 U/L. Patient was extubated on hospital day 9. Citalopram levels (normal 9 – 200 ng/mL) were ordered on initial labs (2900 ng/mL; 7 hours post ingestion), hospital day 1 (730 ng/mL; 53 hours post ingestion), and hospital day 2 (340 ng/ml; 65 hours post ingestion). Pharmacogenomic studies were obtained and she was found to be an intermediate CYP2C19 metabolizer which reduces citalopram inactivation and may cause increased levels and toxicity.
Case Discussion: We report a case of severe serotonin syndrome in a patient with impaired citalopram metabolism due to CYP2C19 enzyme genotype. In the majority of serotonin syndrome cases, symptoms resolve rapidly after initiation of treatment with severe cases typically associated with ingestion of multiple serotonergic agents. Our patient had a severe case after single agent ingestion that we believe was related to an enzyme genotype slowing citalopram metabolism.
Conclusions: Single citalopram overdose may be associated with severe serotonin syndrome in patients with slowed metabolism due to enzyme genotype.

KEYWORDS Serotonin syndrome; citalopram; overdose

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158. Use of Dexmedetomidine to Successfully Treat Bupropion-Induced Sympathomimetic Toxicity

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Background: Bupropion is a commonly prescribed medication, which, in overdose can produce a sympathomimetic toxidrome including seizures. Treatment currently centers on aggressive sedation with benzodiazepines.
Case Report: This is a single case report. A 16 year old girl presented to a tertiary children's center after ingesting bupropion, clonidine, hydroxyzine, and citalopram. The patient had two seizures in the pre-hospital setting and was initially confused, tremulous, normotensive (108/79 mm Hg) and had a heart rate of 90/min. She initially demonstrated signs of both anticholinergic and sympathomimetic toxicity and was admitted to the floor due to her stable clinical condition. Over the course of 32 hours, the patient required progressively increased amounts of intravenous benzodiazepines, totaling 32 mg of lorazepam and 70 mg of diazepam, despite which severe agitation continued. In addition, her vital signs worsened, with a heart rate of 122/min and a blood pressure of 137/67 mm Hg. The patient was transferred to the ICU and dexmedetomidine was initiated at a rate of 0.2 mcg/kg/hr which was rapidly titrated to 0.6 mcg/kg/hr. While she remained on dexmedetomidine for 50 hours, the patient received 20 mg of lorazepam for break-through agitation and seizure prevention. Over this time period, the patient's hypertension and tachycardia resolved, the patient's benzodiazepine requirements were reduced, and she never developed any respiratory depression.
Case Discussion: Dexmedetomidine is a centrally acting alpha2-agonist which reduces central sympathetic outflow. It has previously been safely used in children. It has excellent sedative effects, and does not cause significant respiratory depression. The adult literature has already described its use in the treatment of acute cocaine intoxication. In this case, we successfully applied the use of dexmedetomidine to the sympathomimetic toxicity of bupropion, reducing benzodiazepine use and possibly preventing intubation.
Conclusions: Dexmedetomidine should be considered as a valuable adjunct for the treatment of acute sympathomimetic toxicity induced by bupropion.

KEYWORDS Dexmedetomidine; bupropion; overdose

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159. Massive Ibuprofen Ingestion with Multi Organ Failure Treated with Extracorporeal Membrane Oxygenation (ECMO)

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Background: Ibuprofen is a non-steroidal analgesic with a favorable safety profile that has been available over the counter since 1984. Massive exposures (>400 mg/kg) can result in metabolic acidosis, bleeding, shock, acute renal failure, hepatotoxicity and death. We describe a case of a massive ibuprofen ingestion successfully treated with ECMO
Case Report: A 16 year old female presented to the emergency department (ED) 2.5 hrs after ingesting 1,142 mg/kg of ibuprofen. She was vomiting with decreased mental status, and was intubated for airway protection. Vitals were: Afebrile, heart rate 101 bpm, blood pressure 84/34 mmHg, respiratory rate 18 bmp and oxygen saturation 100%. Charcoal was given via nasogastric tube and whole bowel irrigation was administered. She received 3 liters of normal saline and dopamine infusions, but developed refractory hypotension that ultimately required norepinephrine, vasopressin, and epinephrine for blood pressure support. Initial pH was 7.02 and worsened to 6.85 with a lactate of 11.9 mmol/L. Given her worsening hemodynamics and metabolic acidosis, nephrology and surgery were consulted for hemodialysis (HD) and possible ECMO, respectively. ECMO was instituted in parallel with HD, but was discontinued less than 24 hours later due to a retroperitoneal hematoma and increasing abdominal distention. She developed intraabdominal hypertension requiring laparotomy and hematoma evacuation. She had a prolonged hospital course complicated by femoral artery repair, elevated transaminases, massive blood product transfusion, disseminated intravascular coagulation, pulmonary effusion requiring tube thoracostomy, and renal failure requiring prolonged HD. Four hours post ingestion her serum ibuprofen level was 570 mcg/ml. Despite these complications, she made a remarkable recovery and was discharged to inpatient psychiatry on hospital day 22. Follow up 2 months later showed a normal creatinine and full neurological recovery.
Case Discussion: Hemodialysis does not remove propionic acid metabolites, but can be used to correct severe metabolic derangements. ECMO is an attractive therapeutic modality in cases of poor oxygenation or decreased cardiac output. Its use has been described only once in the literature for this exposure. Our case highlights the challenge of managing a massive ibuprofen overdose, as well as the potential morbidity associated with utilizing ECMO to treat it.
Conclusions: ECMO can be considered for the treatment of potentially life threatening ibuprofen overdoses, but its use can be associated with significant complications.

KEYWORDS ECMO; Ibuprofen; Overdose

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160. Profound Acidosis and Hyperkalemia without Renal Failure in a Pediatric Patient after Ethylene Glycol Ingestion

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Background: Ethylene glycol, a primary ingredient in anti-freeze preparations, is a highly toxic liquid. Ethylene glycol is metabolized to glycolic and oxalic acids which are responsible for the metabolic acidosis often seen with ethylene glycol toxicity. The acidosis that accompanies these metabolic products is also typically associated with renal failure. We present a case of profound metabolic acidosis following ethylene glycol ingestion without renal failure.
Case Report: 16 yo female presented to the Emergency Department with altered mental status. She was somnolent, seemed intoxicated, and was uncooperative. She was last seen at her baseline mental status approximately 12 hours prior. She had one episode of vomiting at home but was unable to provide any further history. Her presenting vitals: HR- 102 bpm, BP- 138/98 mmHg, RR- 16/minute, O2 sat- 100%, Temp- 96.0 °F orally. On exam she appeared intoxicated and was answering questions intermittently. Laboratory analysis revealed a metabolic acidosis with an ABG- 7.11/20/141/6/-22.7/2.2, an anion gap of 26, HCO3- 6mmol/L, Creatinine- 0.78mg/dL Potassium- of 6.1mmol/L, Calcium- 7.8mg/dL and Lactate- 2.2mmol/L. Acetone and ethanol were undetectable. Serum osmolality was unavailable. Repeat labs drawn 2 hours later demonstrated a pH of 6.87, HCO3- 4mmol/L, potassium of 7.1mmol/L, Creatinine – 0.89mg/dL. Urinalysis showed crystals in the urine. Her EKG demonstrated peaked T waves. The patient was given sodium bicarbonate, calcium gluconate, insulin, and dextrose. The patient was given a dose of fomepizole (15mg/kg) and then transferred to a tertiary care pediatric center. Upon arrival to the tertiary care center, she admitted to drinking anti-freeze in a suicide attempt. She underwent 4 hours of emergent dialysis and fomepizole was continued for 2 more doses. The post dialysis ethylene glycol level was 46mg/dL. The patient had full recovery following dialysis and never developed renal failure. She was discharged to an inpatient psychiatric facility on hospital day 4.
Case Discussion: We present the only known documented case of profound metabolic acidosis with accompanying hyperkalemia and normal renal function in a pediatric patient following ethylene glycol ingestion. In the setting of a limited history, clinicians often rely on the presence of metabolic acidosis, osmolar gap, and elevated creatinine in the setting of hypocalcemia to raise suspicion of an ethylene glycol ingestion.
Conclusions: Clinicians should be aware that the diagnosis of ethylene glycol ingestions must be made while taking all values into account and not relying on a normal creatinine to exclude the possibility of an ingestion.

KEYWORDS Ethylene Glycol; Metabolic Acidosis; Pediatric

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161. Takotsubo's Cardiomyopathy following massive diphenhydramine ingestion

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Background: Diphenhydramine is a potent H1 antagonist. In overdose, diphenhydramine can cause anticholinergic symptoms and sodium channel blockade. Large exposures can result in hypotension, seizures, and ventricular dysrhythmias. We present a case of Takotsubo Cardiomyopathy following a diphenhydramine overdose.
Case Report: 40 year old female with a history of depression presents to the ED after ingesting an "entire bottle" of diphenhydramine. Neither the size nor time of ingestion were clear. Her

prescription medications included lorazepam, fluoxetine, acetaminophen-oxycodone, and tramadol. All of her prescription medications were accounted for. Initial vitals were: HR 143, BP 170/120, RR 24, Temp 98.1, O2 98%. EKG showed sinus tachycardia at a rate of 136, QRS 122, QTc 463, and ST-depressions in the inferior leads. Physical exam was significant for dry, flushed skin, urinary retention, and mydriasis. Four hours after presentation the patient experienced a self-limited seizure. Repeat ECG was grossly unchanged except for a QTc of 521. She received a bolus of 2meq/kg of sodium bicarbonate after the seizure. Basic labs, including CBC, chemistry, and troponin were all unremarkable. Approximately 8 hours post-admission a repeat troponin was 0.46 ng/ml. Transthoracic echo (TTE) revealed left ventricular apical hypokinesis with a sparing of the base, and an EF of 20%. Her coronary angiogram showed no luminal disease, and confirmed the TEE findings. A TEE from 2 years prior was normal; repeat TEE on hospital day 10 showed resolution of the left ventricular akinesis, and an EF of 60-65%. Together, these findings were consistent with Takotsubo Cardiomyopathy. At discharge she had developed a new RBBB with a QRS of 122 and QTc of 509.

Case Discussion: We present the first documented case of Takotsubo Cardiomyopathy associated with a diphenhydramine ingestion. The patient had complete resolution of cardiac dysfunction and a normal echocardiogram prior to the overdose.

Conclusions: Significant diphenhydramine overdoses can induce transient ischemic ECG abnormalities, troponin leak, and myocardial dysfunction consistent with Takotsubo cardiomyopathy.

KEYWORDS Diphenhydramine; Takotsubo Cardiomyopathy; Overdose

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162. The X Factor: Lack of Bleeding After an Acute Apixaban Overdose

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Background: Apixaban, a selective Factor Xa inhibitor, is an oral anticoagulant that has recently become available in the United States. In acute overdose, the primary effect of concern is bleeding. We present an acute apixaban overdose without coingestants; it is the first such case reported.

Case Report: A 62 year old female presented to an emergency department 2 hours after ingesting sixty 5 mg tablets (5mg/kg) of her spouse's apixaban. She denied coingestants, and did not take her prescribed medications that day. She has a history of Bipolar Disorder (Type I) and hepatitis C. Her vital signs were normal and she denied symptoms. Chemistry and hematology labs were unremarkable. Factor Assays II, and VII were mildly low, at 64 (73-141%), 56 (68-164%), respectively. The coagulation labs and plasma apixaban levels are described in Table 1. No activated charcoal was given, nor did she receive blood products or factor replacement. During the course of her hospitalization, there was no obvious bleeding, and her hemoglobin did not decrease. After two days of observation, she was transferred to psychiatry.

Case Discussion: This is the first reported case of a one-time acute overdose of apixaban. According to Mayo Medical Laboratories, peak plasma apixaban levels after a 50 mg dose range from 663 to 707 ng/ml. The package insert reports linear pharmacokinetics with oral doses up to 10 mg, and half life of 12 hours. This case suggests higher dose acute ingestions may not follow linear kinetics. Table 1 shows a potential decrease in half life early in the toxic exposure – it took less than 6 hours to decrease by half initially, then 15 hours. At doses greater than 25 mg, the rate of absorption is limited, leading to decreased bioavailability; this is a potential explanation for the non-linear kinetics represented by levels. This patient had no toxicological effects despite peak plasma apixaban level of 2765.6ng/dl. PT and INR were mildly elevated with a peak 6 hours post ingestion. The greatest risk of bleeding was during the first 6 hours in this case.

Parameter (Units/Normal Range)	2 hrs (post ingestion)	8 hrs	14 hrs	16 hrs	22 hrs	24 hrs	37 hrs	62 hrs
Prothrombin time (PT) 9.4 -11.8 sec	15.7	17.2	13.3	12.9	12.0		10.7	10.5
INR	1.6	1.8	1.4	1.3	1.2		1.1	1.0
Factor X Assay 59-102%)			68	75		102	119	
Apixaban Plasma levels (ng/ml)			2765.6		1100		560	

Conclusions: This is a case of a one-time acute overdose of apixaban with elevated levels, demonstrating non-linear kinetics. Despite confirmed ingestion, there were no bleeding events.

KEYWORDS Apixaban; Overdose; Toxicokinetics

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163. Changing epidemiology and severity of opioid poisoning: from street drugs to prescription medications

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Objectives: Opioid overdoses have been a major problem for decades, both prescription and illicit use, and is associated with significant out of hospital and in hospital mortality. We aimed to investigate the epidemiology of opioid overdoses and relative toxicity over a 28 year period.

Methods: All opioid overdose presentations to a tertiary toxicology service for 1987-2015 were reviewed. Data are recorded prospectively for all admissions, including demographics, complications (coma [GCS <9], hypotension [systolic BP <90mmHg], seizure), treatments (intubation, naloxone) and outcomes (length of stay [LOS], intensive care [ICU] admission, death).

Results: There were 1488 opioid overdoses over the period including heroin (446), oxycodone (360), methadone (206), morphine (174), tramadol (135), codeine (73) and dextropropoxyphene (29), with smaller numbers of fentanyl, buprenorphine and hydromorphone. There was a peak incidence in 1999 associated with a peak in heroin overdoses, and then increasing cases in the last 5 years associated with mainly increasing oxycodone overdoses, and relative constant numbers of methadone and morphine since 2000. The median age was 35y (14-92) and 731 (49%) were female. Of 1313 with an ingestion reason, 860 were deliberate self-harm (65%) and 407 (31%) recreational poisoning. Coma occurred in 204 (14%), hypotension in 114 (8%) and seizures in 42 (3%). The median LOS was 16.5h (interquartile range:14-29h), 238 (16%) were admitted to ICU, 132 (9%) were intubated and 21 (1.4%) died. Naloxone was administered in 629 (42%) of patients. Morphine and methadone caused more severe toxicity with more ICU admissions, naloxone use, intubations and deaths (Table). Heroin had the shortest LOS, less ICU admissions and intubations, but still 2% in hospital mortality. Oxycodone and tramadol caused less complications, lower rates of ICU, intubation and no deaths (Table).

Drug	N	Coma (GCS <9)	%	LOS (Hours)	ICU	%	Naloxone	%	Ventilation	%	Death	%
Heroin	446	82	18%	12 (4-21)	62	14%	253	57%	41	9%	10	2.2%
Oxycodone	360	34	9%	18 (12-29)	42	12%	116	32%	23	6%	0	
Methadone	206	37	18%	20 (12-41)	47	23%	112	54%	25	12%	2	1.0%
Morphine	174	23	13%	24 (13-54)	55	32%	89	51%	24	14%	8	4.6%
Tramadol	135	11	8%	16 (11-30)	14	10%	13	10%	10	7%	0	
Codeine	73	6	8%	16 (12-30)	6	8%	12	16%	3	4%	0	
Dextropropoxyphene	29	1	3%	17 (9-19)	1	3%	4	14%	0		0	
Fentanyl	ie	5	31%	17 (6-30)	2	13%	9	56%	2	13%	0	
Buprenorphine	14	2	14%	17 (2-27)	2	14%	2	14%	2	14%	1	7.1%
All Opioids	1488	204	14%	17 (14-29)	231	16%	629	42%	130	9%	21	1.4%

Conclusions: Opioid overdoses remain a frequent overdose with significant toxicity from morphine and methadone, although less severe effects with oxycodone and tramadol. Although heroin is now uncommon it once caused severe but short-lived effects.

KEYWORDS opioid; poisoning; epidemiology

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164. Holy Bezoar Batman, that's a lot of Bupropion

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Background: Bupropion bezoar formation has been speculated to cause delayed peak effects, but documentation of an esophageal bezoar could not be found in the literature. Additionally, it is known to cause QRS widening, thought to be due to reducing cardiac intercellular coupling rather than sodium channel blockade.
Case Report: A 71 year old male presented minimally responsive after suspected ingestion of 120 mg clonazepam and 90 tablets of bupropion, based on empty pill bottles. Initially, the patient was somnolent, but then developed hypoxic respiratory failure requiring intubation. Early vital signs were essentially normal, with a heart rate of 90, bp 120/50, and an initial QRS of 93 ms.
His post-intubation CXR demonstrated a widened mediastinum and a follow-up CT chest showed a massive esophageal pill impaction. EGD was performed but could not remove all of the tablets, which were breaking apart during removal, and a repeat EGD was planned for the following morning. Overnight, however, the patient became hypotensive to 70/30s and developed QRS prolongation to 117 ms with a QTc of 410 ms. The patient was treated with levophed and 4 boluses of sodium bicarbonate, during which his QRS continued to increase to 141 ms and eventually peaked at 204 ms. His QTc also increased to 537 ms. With continued supportive care, his heart rate and blood pressure eventually normalized, but he continued to have difficulty oxygenating. Bronchoscopy demonstrated egg white secretions and pill fragments, suggesting aspiration of his esophageal contents. Due to his severe illness, he was transitioned to comfort measures per his family's wishes.
Case Discussion: Formation of bezoars due to bupropion has been theorized as a mechanism for delayed peak toxicokinetic effects and long $^1/_2$-life in the setting of overdose. This case demonstrates evidence that such a bezoar is certainly possible. Management may be difficult. Removal of the pills seems logical to minimize the exposure, but may be met with difficulty due to further fragmentation of the pills. The large esophageal burden also likely contributed to aspiration of pill fragments. Whether pulmonary absorption may have contributed to this patient's course is unknown. The treatment of QRS prolongation in the setting of bupropion intoxication may also be difficult. Unlike many xenobiotics that widen the QRS, bupropion does not work by sodium channel blockade, but rather by reducing cardiac intercellular conduction. Whether the sodium bicarbonate had an effect on the QRS in this patient is unknown.
Conclusions: We present a case demonstrating severe toxicity in the setting of a bupropion esophageal impaction with resultant cardiovascular instability and severe QRS widening.

KEYWORDS Esophageal impaction; Bupropion; QRS

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165. Cyclic or Acyclic? Single Ingestion Cyclobenzaprine Trends

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Background: Cyclobenzaprine is a popular muscle relaxer prescribed primarily for the relief of musculoskeletal pain. Reported poisoning may result in both central nervous system effects (obtundation, hallucinations, lethargy, confusion) and cardiovascular toxicity (tachycardia, dysrhythmias, QRS widening). The aim of this study is to report trends in morbidity and mortality associated with single ingestion cyclobenzaprine over a 10 year period from one regional poison center (RPC).
Methods: Electronic RPC records coded for cyclobenzaprine (January 1, 2006 through December 31, 2015; single ingestion; intentional; age 12 years and greater) were queried. Numbers and trends were compared for 1) total cases; 2) intubations; 3) tachycardia (>100 BPM); 4) QRS >120 ms; 5) major effects; and 6) mortality.
Results: A total of 2378 cyclobenzaprine cases were reported to the RPC during the study period. Single ingestion cases meeting inclusion criteria were 567. A consistent upward trend in cases reported per year occurred over time revealing a 135% increase between 2006 (38 cases) and 2015 (87 cases). Each year included intubated patients (range; 2-5) with a total of 30 during the study period. Tachycardia was present in 44% of cases and QRS widening (longest 142 ms) was reported in 5 patients (all of whom had a past medical history of a bundle branch block). Five cases were coded as major effects and no deaths were reported.
Conclusions: Intentional single ingestion cyclobenzaprine cases reported to this RPC have increased dramatically. Potential reasons for this are speculative and require further study. Cyclobenzaprine cases remained benign for the most part and resulted in only a few major effects and no mortalities.

KEYWORDS Cyclobenzaprine; Single; Trend

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166. How PHAT is Fat for Bupropion Poisoning? One Poison Center's Experience

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Background: Intravenous Lipid Emulsion (ILE) has been advocated as a therapy for severe bupropion-induced cardiotoxicity after a landmark case report in 2008. That patient achieved near-normal neurologic function after the use of ILE during a prolonged cardiopulmonary resuscitation. The aim of this study is to report one regional poison center's (RPC) trends in bupropion poisoning in addition to outcomes as they relate to ILE therapy.
Methods: Electronic RPC records coded for bupropion and an intentional reason for ingestion from January 1, 2009 to December 31, 2015 were retrospectively reviewed. Information collected included total number of cases and outcomes coded as "major effect" and "death". Use of ILE, indications for its administration, and outcomes for these patients were further investigated.
Results: Results for the 7-year study period showing number of cases, major effects, use of ILE, and deaths are reported in Table 1. A total of 9 cases were identified in which ILE was utilized. Of these, 5 patients survived and 4 died. Indications for ILE included hypotension (6 patients; 2 died), cardiac arrest after return of spontaneous circulation (2 patients; 1 died), and intractable seizures (2 patients; 1 died). One of the patients who received ILE for intractable seizures subsequently had cardiac arrest within 12 hours. All patients received bolus dosing of ILE with variable lengths of infusion. Two patients developed pulmonary edema within 3 hours of ILE and both were among the patients who died.
Conclusion: In this 7-year review of RPC data, 44% of patients that received ILE died. Of patients who were hypotensive without cardiac arrest, 33% died. Further research to describe efficacy, indications, and dosing regimens for the use of ILE in bupropion poisonings is warranted.

KEYWORDS Bupropion; Intravenous Lipid Emulsion; Overdose

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Year	2009	2010	2011	2012	2013	2014	2015
Cases	175	178	187	195	137	146	256
Major Effects	8	13	12	18	14	13	21
HE	0	1	1	0	4	1	2
Deaths	1	3	4	2	0	1	3

167. Primum non nocere or primum nihil? On the ethical and legal conundrum of non-intervention

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Background: Patient autonomy among those with capacity is sacrosanct, including the right to refuse care. Capacity to make one's decisions is, however, a judgment call, the consequences of which may weigh heavily on the patient's well-being. We review a case of intentional ethylene glycol ingestion in a patient who refused care. In spite of an emergency detention order and reported intent at self-harm, hospital counsel advised against treatment, due to the patient's intact orientation. Unfortunately, the advice of an ethics panel, a subject matter expert in toxicology or the courts was not sought in time to prevent substantial clinical deterioration.
Case Report: A 61-year-old woman was brought by police to the emergency department at 2039 for suicidal ideation. Her brother reported to police she had drunk an unknown quantity of antifreeze. He did not, however, come to the hospital, so his report could not be corroborated by physicians. On arrival, the patient was awake, alert, and oriented, exhibited no evidence of psychosis and vehemently denied any suicidal ideation, specifically denying antifreeze ingestion. Vital signs were BP 155/73, pulse 92, respirations 16, and temperature 36.6. The patient refused all blood draws. At 2140, the hospital attorney was contacted. She advised physicians that "if the patient was not displaying signs of inability to make medical decisions," that she had the right to refuse a lab draw. At 0200, the patient became unresponsive, with a Glasgow Coma Scale of 3. She was intubated and sedated. At 0211, poison control was called, advising physicians to send out an ethylene glycol concentration, determine the osmol gap, and to begin fomepizole. The on-call medical toxicologist was not notified until 1544. Initial laboratory revealed a measured serum osmolality of 353 mOsm/kg, serum potassium of 7.3 mmol/L, creatinine 0.75 mg/dL, total CO2 of 5 mmol/L and anion gap of 18 mmol/L. The patient underwent hemodialysis for six hours. Fomepizole was continued for about 48 hours. She remained intubated for approximately 40 hours, suffered atrial fibrillation requiring metoprolol, and pneumonia requiring antibiotics. On hospital day 6, she began following commands. The initial ethylene glycol level returned on day six, as well: 179 mg/dL. On hospital day 19 the patient was transferred to a psychiatric center.
Case Discussion: This patient suffered potentially preventable acute kidney injury, mental status changes, pneumonia, and prolonged hospitalization due to failure to institute early fomepizole treatment. While the hospital attorney was consulted, the poison center and medical toxicologist were consulted late and neither the ethics committee nor the courts were consulted.
Conclusions: Refusal of care by patients with suicidal intent poses complex ethical and legal dilemmas for physicians. Emergency consultation with ethics and subject matter professionals, as well as the courts may be not only necessary, but lifesaving.

KEYWORDS Ethics; Consent; Suicide

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168. Is All Phosgene Created Equal? A Case of Intentional Ingestion of Triphosgene Crystals

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Background: Triphosgene [bis(trichloromethyl) carbonate], C3Cl6O3, is used as a less hazardous substitute for phosgene in phosgenation reactions. According to the American Chemical Society, the toxicity of triphosgene is "exactly the same" as phosgene . While phosgene is a gas at room temperature and exerts its toxicity via inhalational exposure, triphosgene exists as a solid at room temperature, making ingestion a more likely route of exposure. A paucity of literature exists regarding triphosgene toxicity.
Case Report: A 55 year-old-male with a history of depression and alcohol abuse presented following intentional ingestion of triphosgene crystals and an unknown liquid, both obtained from a college campus laboratory, in a suicide attempt. Initial symptoms were abdominal pain, hematemesis, burning of the mouth and throat, difficulty swallowing, and white patches visible on the tongue. Physical findings included facial erythema of the cheeks and frontal area, and garbled speech. Laryngoscopy was significant for oropharyngeal erythema with sloughing of the epidermal layer and an edematous posterior pharynx. Vital signs upon presentation were as follows: heart rate 110 bpm, blood pressure 150/110 mmHg, oxygen saturation 91%. He had no significant laboratory abnormalities. On day three of admission, endoscopy revealed hemorrhagic esophagitis with ulcers, hemorrhagic gastritis, and an inflamed upper palate, tongue base, and posterior pharynx. Repeat chest X-rays throughout admission were all unremarkable. The patient's respiratory status was stable through the course of his hospitalization with no oxygen saturations $<$91%. On day ten of admission, the patient was transferred to inpatient psychiatry with a persistent throat pain.
Case Discussion: The MSDS for triphosgene recommends adhering to the same precautions as phosgene, as contact with water or heat will release phosgene gas. In this instance however, even after triphosgene was introduced to the warm, moist environment of the GI tract, no respiratory effects were observed. The presumptive mechanism of pulmonary effects after ingestion would be off-gassing of phosgene gas. Some theories explaining why this was not observed in our patient include inhibition of conversion of triphosgene to phosgene due to the acidic gastric environment, insufficient production of phosgene gas to cause toxicity via off-gassing, or presumption of inhalation as the primary route of toxicity.
Conclusions: This case report suggests that an ingestion of triphosgene does not produce phosgene-like respiratory toxicity. Corrosive effects may occur, although significant confounding arises from the coingestion of an unknown liquid.

KEYWORDS triphosgene; ingestion; phosgene

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169. Xylazine Intoxication from Oral Ingestion

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Background: Xylazine is a potent alpha 2-adrenergic agonist used as a sedative, analgesic and muscle relaxant by veterinarians since 1988. It is not FDA approved for use in humans, however, human toxicity has been reported secondary to both intentional and unintentional exposure. Previously reported cases of human toxicity have generally been limited to the intravenous route. We present an unusual case of xylazine toxicity due to oral ingestion.
Case Report: A 46-year-old woman with a past medical history of anxiety, depression, chronic back pain, and previous suicide attempts presented to the emergency department (ED) unresponsive approximately 2 hours after intentionally ingesting 120 mg of xylazine. Paramedics administered IV naloxone with no response, and IV atropine for bradycardia. In the ED, the patient was unresponsive to verbal or painful stimuli, and was promptly intubated for airway protection. Initial vital signs were blood pressure 168/99 mmHg, heart rate 54 beats/min, respiratory rate 16 breaths/min, oxygen saturation 100% on non-rebreather mask, and temperature 37.7 °C. Physical examination revealed somnolence, miosis, and bradycardia. Her initial EKG revealed sinus bradycardia with normal intervals. Laboratory results were significant for a white blood cell count of 11.1×10^3/uL, creatinine kinase 310 U/L, lactate 5.9 mmol/L, and glucose 123 mg/dL. A urine drug immunoassay was positive for cannabinoids. Ethanol was negative. The patient was admitted to the intensive care unit for further management. She was successfully extubated 17.5 hours after intubation. Her heart rate stabilized in the 60s, and she returned to her baseline mental status. The patient was discharged to psychiatric care with no apparent adverse sequelae three days following presentation.
Case Discussion: Xylazine is a potent alpha 2-agonist that is structurally and pharmacologically similar to clonidine. As its use is restricted to veterinary medicine, data on human toxicity is limited to less than 50 cases reported in the literature. However, reported symptoms of toxicity in humans are similar to those in animals, regardless of the route of exposure. Most previously reported cases of human xylazine exposure were by the intravenous route. There is no defined toxic level of xylazine in humans. The patient described here ingested only 30% of the dose ingested in a previous report, but exhibited a similar clinical course. Nearly half the reported cases of human toxicity were fatal, the majority of which were associated with xylazine use as an adulterant for drugs of abuse. There is no antidote, and management is largely supportive.
Conclusions: Xylazine is a potent xenobiotic causing significant toxicity and mortality in humans regardless of route of administration.

KEYWORDS Xylazine; alpha 2-agonist; clonidine

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170. Caution Wet Surface Ahead: A Dangerous Soak in Nicotine

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Background: Nicotine is a highly toxic compound that is easily absorbed by the lungs, skin, and intestinal tract. Several case reports of significant toxicity and even death have been reported from ingestion of nicotine products. Misuse of transdermal nicotine patches has also resulted in toxicity. The popularity of electronic cigarettes (e-cigarettes) has increased recently, as many perceive it to be less harmful than traditional tobacco use. However, the easy availability of large quantities liquid nicotine for these products has posed a serious public health threat. We report a case of toxicity from intentional dermal exposure from liquid nicotine purchased at an e -cigarette store.
Case Report: A 34-year-old woman presented to the emergency department after a suicide attempt in which she poured a 120mL bottle of 10% nicotine onto her body approximately 90 minutes prior to arrival. The patient did not notify triage of the exposure and it was not until the treating physician noted that her clothes and skin were wet was the full history elicited. The patient was immediately decontaminated with soap and water and her belongings were removed. Initial vital signs were: pulse 86 bpm, respirations 14 bpm, BP 106/67, temperature 97.6oF. The physical exam was significant for lethargy, but a gross tremor in all four extremities was noted whenever the patient was aroused. Motor and sensation were intact. The patient had several episodes of non-bilious, non-bloody emesis. A few hours post exposure, atrial fibrillation developed with a ventricular rate of 90-100 bpm. Laboratory testing was notable for a lactic acid concentration of 5.8 mmol/L and an anion gap of 21. Ethanol, acetaminophen, and salicylate concentrations were undetectable. Two liters of normal saline were administered. Diazepam 10mg IV was administered to treat the tremor and the patient was monitored in the ICU for 24 hours. A transthoracic echocardiogram showed an ejection fraction of 60% with no sign of valvular disease. The patient regained a normal sinus rhythm without intervention 18 hours post exposure. A serum nicotine concentration from the initial ED blood work was 243ng/mL. The patient was transferred to the psychiatry service for further management.
Case Discussion: This case highlights the risk of significant toxicity with dermal exposure to liquid nicotine products. A case series of toxicity from nicotine patches recorded concentrations of 5-27ng/mL. It is possible that the liquid formulation of nicotine would allow for a larger surface area to be affected as well as faster absorption, leading to the significantly elevated concentration in our report.
Conclusions: Clinicians should assess for dermal contamination in any exposure to an e-cigarette product and begin prompt decontamination when needed.

KEYWORDS nicotine; suicide; decontamination

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171. Rapid death from metal phosphide despite optimal supportive care

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Background: Metal phosphides are inorganic compounds commercially used as fumigants. Release of cytotoxic phosphine gas occurs when phosphides react with moisture in the gut. While fatal ingestions in developing countries are well documented, few cases of rapid death have been reported in North America. We report the cardiac arrest and subsequent death of an otherwise healthy adult from phosphide tablets within 2 hours of ingestion, despite rapid EMS response and access to quaternary care.
Case Report: A 49 year old, previously healthy, suicidal male fumigator was witnessed to ingest 2 "phosphorus fumigant tablets". EMS arrived 25 minutes later, and found him to be pale, diaphoretic and vomiting, with complaints of shortness of breath and chest pressure, with the following vital signs: HR 111/min, BP 82/58, RR 24/min, RA O2 saturation 90% and GCS 15. On arrival to the emergency department (ED) 27 minutes later (52 minutes post-ingestion), the patient was hypotensive, cyanotic, diaphoretic, incontinent of stool, unresponsive, and had a strong garlic-like odor; his initial vital signs and lab results were: HR 60-80/min, systolic BP 80, T 34.9C (94.8F), RR 8/min with unmeasurable O2 saturation, GCS 8-10, glucose 4.7mmol/L (84.6mg/dl), magnesium 0.49 mmol/L (1.19 mg/dl) and venous pH 7.33, pCO2 31, HCO3 16, lactate 6.2mmol/L. Within 15 minutes, he was intubated, then an orogastric tube inserted and gastric lavage attempted. Repeat arterial blood gas showed a pH of 6.93, PC02 44mmHg, HC03 9mmol/L, lactate 8.4mmol/L, and potassium 6.1mmol/L. At 1h 55mins after ingestion the patient developed a PEA arrest and CPR was started. Resuscitative efforts included 4.7L of Normal saline, Epinephrine 3mg, Magnesium Sulfate 2gm, Calcium Chloride 1gm, Atropine 2mg, and Potassium Chloride 10mmol. Subsequent infusions included Epinephrine 2 mcg/kg/min, Dobutamine 20 mcg/kg/min and Norepinephrine 8 mcg/min. ACLS and CPR efforts were discontinued after 34 minutes. A bedside cardiac ultrasound demonstrated cardiac standstill. The patient was pronounced dead at 2 hours, 29 minutes post ingestion.
Case Discussion: Our case developed tachycardia, hypotension, diaphoresis and hypoxia within 30 minutes of ingestion, and cardiac arrest within 2 hours despite timely and aggressive management. While laboratory confirmation of gastric fluid at NMS Labs is still pending, the patient's occupation, his self-reported ingestion of "phosphorous tablets", the strong garlic-like odor and rapid decline are highly suggestive of a metal phosphide ingestion.
Conclusions: The death of our patient within hours of ingesting 2 phosphide tablets despite prompt EMS transport and aggressive quaternary care demonstrates the rapid and inexorable deterioration this poison causes.

KEYWORDS phosphide; mortality; fumigant

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172. Suicide Attempt from English Yew Intoxication Successfully Managed with Adjunctive Intralipid Emulsion Therapy

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Background: Intralipid emulsion (ILE) therapy has demonstrated positive outcomes in the management of toxicity with lipid soluble agents such as intravenous local anesthetics, calcium channel antagonists and cyclic antidepressants. ILE is gaining popularity as another line of therapy for many other toxicoses. To the best of our knowledge, this is the first reported case of ILE for severe cardiac conduction delays secondary to English Yew intoxication.
Case Report: A 19 year-old female was evaluated in the ED 90 minutes post-ingestion for a suicide attempt involving English Yew. She reported ingesting half of a blender full of a smoothie that she made from berries, leaves, and seeds of English Yew, which she had self-harvested. Her initial vital signs consisted of BP 126/88, Pulse 111, Temp 97.3 (oral), RR 18, and SaO2 100% on room air. Physical exam was unremarkable except for her tachycardia. Clinically, she had vomited several times post-ingestion and reported mild nausea persistently but no chest pain, abdominal pain or palpitations. Initial ECG showed a sinus rhythm with a rate of 109, QRS of 183 ms, and a QTc of 583 ms. APAP, ASA, EtOH and a UDS were all found to be negative. Over the next 2.5 hours her cardiac activity changed to wide complex ventricular tachycardia to Torsades de Pointes, and became pulseless. Cardiac arrest and respiratory failure ensued. She was intubated, and aggressive ACLS (advanced cardiac life support) was performed which included administration of 11 amps of sodium bicarbonate, amiodarone, and 3 grams of magnesium. She was unresponsive to defibrillation, so Intralipids were administered. She converted to sinus tachycardia from ventricular tachycardia with ACLS and sodium bicarbonate, and there was a dramatic narrowing of the QRS and QTc interval soon after administration of ILE. Approximately 30 min after administration of Intralipid, the patient's EKG showed a QRS of 144 ms and QTc of 521 ms. The longest intervals recorded were a QRS 183 ms and QTc 698 ms. Approximately 36 hours post-ingestion, her QRS and QTc intervals had normalized, she was extubated, and was medically cleared.
Case Discussion: English Yew [Taxus baccata] contains the alkaloid taxine which is a cardiovascular toxin. Fatal poisoning can occur particularly in the setting of an intentional ingestion involving plant parts other than the berries. ILE is used in management of the poisoned patient both as an antidote and as a rescue therapy based on a lipid-sink model. Chemically, taxine is soluble in organic, non-polar solvents; therefore ILE is worth considering in the multi-modal approach of severe cardiotoxicity secondary to English Yew intoxication.
Conclusions: This case illustrates the potential utility of ILE as a treatment option for taxine toxicity in addition to cardiopulmonary bypass and digoxin immune Fab which have been previously reported.

KEYWORDS Yew intoxication; intralipid emulsion therapy; taxine alkaloid

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173. The trip across the ocean: Diphenidine lands in the U.S

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Background: Diphenidine (1-(1,2-diphenylethyl)piperidine) is a novel dissociative anesthetic with activity comparable to ketamine, which has emerged as a substance of abuse favored for its mind-altering effects. Both are notable for NMDA receptor antagonism, although the extent to which diphenidine also causes sympathomimetic excess is unclear. A recent observational case series identified 14 cases of analytically confirmed diphenidine abuse in Sweden (Helander et al, 2015). To date, no published reports of its use in the United States (US) exist.
Case Report: A 32-year old male with a history of traumatic brain injury, polysubstance abuse and multiple prior overdoses presented to the emergency department from a group home after he was noted to display rapid mood swings with disorganized and bizarre behavior. At the scene, a baggie labeled "NOT FOR HUMAN CONSUMPTION: Diphenidine Crystal" was found (image provided at poster). In the ED, vital signs were normal (blood pressure 138/81 mmHg, pulse 88 beats per minute, temperature 36.8 °C, breathing 13 breaths per minute on room air, with a peripheral oxygenation of 94%). He was noted to be diaphoretic, oriented only to place and self, with limited command-following and tangential thoughts. Physical examination was otherwise normal. Basic metabolic panel was entirely within normal limits; acetaminophen and breathalyzer levels were undetectable. He was treated only with olanzapine prior to transfer to a psychiatric unit. The patient admitted to purchasing 'research chemicals' online, and frequently abusing diphenidine and methoxetamine. He reported hallucinations with this substance, although less so than what he experienced with other hallucinogenic drugs. He reported effects which were magnified with insufflation compared to pyrrolization. Gas chromatography-mass spectrometry (GCMS) of the substance that he had ingested (image provide at poster) demonstrated peaks consistent with previous spectra reported from known diphenidine exposures (Kudo et al 2015).
Case Discussion: Ketamine and phencyclidine have been popular drugs of abuse for years, but it has only been with the increased availability and popularity of online designer drugs that abuse of other dissociative substances have arisen. Diphenidine was initially identified and made available in the 1920s. Although multiple cases of its use have been reported in Europe, confirmed diphenidine abuse has not been reported in the US. Adverse effects from confirmed cases of diphenidine are similar to those seen with other dissociative substances like ketamine and methoxetamine: hypertension, tachycardia, anxiety and altered mental status including confusion, disorientation, dissociation and/or hallucinations. This patient's altered mental status correlates well with other published reports of diphenidine abuse, and GCMS data confirm that this is one more drug that has indeed made its way off of the Internet and into the US recreational drug culture.
Conclusions: This is the first laboratory confirmed diphenidine intoxication in the US. Healthcare providers should be aware of the potential for abuse of novel dissociative anesthetics that won't appear on a standard drug of abuse screen. In patients who present dissociated after suspected substance abuse, diphenidine should be considered in addition to phencyclidine, dextromethorphan, ketamine, and methoxetamine.

KEYWORDS Diphenidine; ketamine; synthetic drug

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174. (Don't) DIY: YouTube, chemistry texts, and down home chloroform synthesis

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Introduction: Chloroform (trichloromethane) is a halogenated hydrocarbon used historically as an inhaled anesthetic. Hepatotoxicity has been noted with therapeutic and recreational inhalational exposures, and significant hepatic necrosis is reported following oral ingestions. Recreational inhalation for its euphoric effect is well-documented; oral ingestions are far less common.
Case Report: A 14-year old male contacted the Poison Center (PC) to report that he had just ingested 32mL of chloroform that he had synthesized at home based on reactions that he had studied in his chemistry textbook. PC immediately communicated with EMS, and an ambulance was immediately dispatched to his home. Medics arrived 10 minutes after ingestion and encountered a patient who appeared agitated, complained of nausea and was hyperventilating, unable to speak. A complex chemical "filtration" system consistent with his report of on-site chemical synthesis was noted at his residence. His level of consciousness rapidly declined. PC recommended supportive cares en route to the receiving hospital; prehospital vital signs were not available. On arrival to the receiving hospital, the patient was profoundly somnolent, with a calculated GCS of 8. He was promptly intubated for airway protection, and initial assessment demonstrated a normothermic, obtunded, flushed, diaphoretic patient with midrange & reactive pupils with tachycardia to the 130s, with reportedly "normal" respiratory rate and blood pressure. Baseline hepatic transaminases were ordered, and the PC recommended prompt gastric aspiration and initiation of n-acetylcysteine (NAC) therapy for anticipated glutathione depletion and resultant hepatotoxicity. He was transferred to a pediatric center for further cares, arriving with vital signs of 98.7 °F, pulse 77, and blood pressure 106/45 sedated with 40μg/kg/minute propofol infusion. The patient was extubated uneventfully overnight. NAC was continued for 72 hours. He remained hemodynamically stable and appropriate and was transferred to inpatient psychiatry on hospital day (HD) #4 following 72 hours of NAC therapy. Transaminases slowly peaked on hospital day #8 (Table). Chloroform, drawn on HD#5, was in process at the time of writing.
Case Discussion: Large chloroform ingestions are unusual, and despite easily accessed do-it-yourself (DIY) videos describing its home manufacture, this case is particularly unique inasmuch as it involved the home manufacture of chloroform for suicidal purposes. Despite some similarity to acetaminophen-induced hepatotoxicity (via depletion of glutathione), no clear standard of care characterizes the optimal regimen to mitigate hepatotoxicity resulting from the cytochrome P450-mediated oxidation of chloroform to chloromethanol, and the subsequent dechlorination to phosgene and hydrochloric acid. In this case, a 72-hour course of intravenous NAC was chosen noting an anticipated delay to glutathione nadir following metabolism of the parent compound to its toxic metabolites.
Conclusions: We report the large volume ingestion of trichloromethane synthesized at home for suicidal purposes. The delay in hepatotoxicity is expected in these instances is attributable to a nontoxic parent compound with toxic metabolites. Glutathione depletion must occur prior to the onset of toxicity. Early NAC, in conjunction with early gastric content aspiration, may have mitigated the hepatotoxicity associated with this large oral chloroform ingestion by providing a substrate for the repletion of glutathione.

KEYWORDS Chloroform; hepatitis; n-acetylcysteine

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175. Multistate response to an outbreak of T3 thyroid storm following 1,000-fold compounding pharmacy error

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Background: A compounding pharmacy notified the Poison Center (PC) that a batch of 15mcg liothyronine (T3) capsules incorrectly contained 15,000mcg after a 1:1000 stock solution was replaced with undiluted T3 solution. The resulting capsules reached 10-15 patients; three patients ingested them and developed thyroid storm. Two of three cases were available to report.
Case Reports: Case 1 - A 64-year old female presented to the emergency department (ED) with chest and back pain 4 days after ingesting 30,000mcg of T3, reportedly prescribed to treat "Wilson's Temperature Syndrome." Intravenous lorazepam and nitroglycerine were administered; PC was consulted. Vital signs were: pulse 120, temperature 36.9 °C, blood pressure (BP) 121/80mmHg. Free T3 was immeasurably elevated (>30pg/mL, ref: 2.0-3.5pg/mL); troponin and TSH were undetectable, and metabolic panel revealed hypokalemia (3.2mEq/L). PC recommended an esmolol infusion and transfer to a facility with plasmapheresis capabilities. At transfer the patient was awake and alert with BP 111/77mmHg, pulse 107, and respiratory rate 18. Emergent plasmapheresis was undertaken at the receiving facility on hospital day (HD)#2; free T3 levels fell sequentially (see Table). The patient discharged on HD#4, but ill-defined symptoms persisted 6 weeks later. Case 2 - A 53-year old female with hypothyroidism and history of chronic Lyme disease developed flu-like symptoms after filling a prescription for compounded T3/T4 capsules. She presented to the ED 6 days after symptom onset, confused with "word salad" speech; she was discharged with tramadol and ondansetron. Symptoms persisted, and the next day she returned to the ED and was admitted. Head CT, MRI, EEG, lumbar puncture, chest CT, MRCP and echocardiogram were normal. She discharged on HD#7 following improvement in encephalopathic symptoms. No specific diagnosis was made; thyroid hormones were resumed. She was readmitted for erratic behavior and recurrent delirium 3 days later. Total serum T3 returned elevated (>500ng/dL, ref: 80-200ng/dL). Concurrent PC investigation revealed the compounding error and 1,000-fold overdose (15,000mcg). She was intubated, transferred to a facility capable of plasmapheresis. PC advised the receiving team of the outbreak. Initial vital signs were: temperature 38.5 °C; pulse 120; BP 121/71mmHg; respiratory rate 33/minute; SpO2 91%. Atrial fibrillation with a reduced ejection fraction developed. Endocrinology initiated cholestyramine and corticosteroids; Cardiology initiated propranolol. Repeat cerebral MRI and lumbar puncture were unrevealing. Plasmapheresis on HD#2 correlated with decreasing T3 concentrations (see Table). She was extubated on HD#5. Following hormone normalization, levothyroxine was restarted. She discharged on HD#9 with endocrinology follow-up and PT/OT services. "Non-distributional" weakness, gait difficulty, and muscle stiffness persisted. Echocardiographic

Summary of laboratory parameters in two cases of iatrogenic T3 thyrotoxicosis

Patient #1												
HD	1	2*	3	4	5	6						
Free T3 (jjg/mL)	35	32.6	13.8	5.7		2.4						
Patient #2												
HD (time)	1 (2317)	2* (0455)	2 (1733)	2 (2053)	3 (0456)	3 (1108)	3 (1650)	3 (2331)	4 (0553)	4 (1309)	4 (1757)	6 (0535)
Total T3 Ing/dL]	525	525	504	331	227		181	148	124	123	102	79
Free T3 (jgg/mL)	28		12.7	9	5.9	439	4.8					
Free T4 (rig/dL)	0.4	0.4	0.4	0. 5	0.4		0.3		<0.3			<0.3

*plasmapheresis initiated

ejection fraction was normal 7 weeks later, with grade II diastolic dysfunction.
Case Discussion: Hemoperfusion, exchange transfusion, and plasmapheresis have been implemented for T3 thyrotoxicosis, with variable results. PC response to this outbreak included consultations for the critically ill and collaboration with the state board of pharmacy, multiple state health departments and neighboring PCs.
Conclusions: This iatrogenic outbreak required PC guidance and outreach to identify exposures and ongoing risk to the public. These data suggest plasmapheresis may enhance the elimination of liothryonine in massive T3 overdose.

KEYWORDS Thyrotoxicosis; compounding error; plasmapheresis

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176. Uridine triacetate is a lifesaving antidote for overdoses and severe early-onset 5-fluorouracil and capecitabine toxicities

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Background: Uridine triacetate was approved by FDA in 2015 for adult and pediatric patients who exhibit early-onset, severe or life-threatening toxicities or who receive an overdose of 5-fluorouracil or capecitabine. Uridine triacetate is an oral prodrug of uridine, a direct antagonist of 5-FU, that dilutes and competes with toxic 5-FU metabolites, particularly FUTP incorporation into RNA, reducing morbidity and mortality due to 5-FU and capecitabine.
Increased susceptibility to 5-FU/capecitabine can lead to rapid, early-onset toxicity caused by factors such as: impaired clearance; DPD deficiency (3-5% of the population); and elevated OPRT (which converts 5-FU to toxic intracellular 5-fluorouridine nucleotides). Life-threatening or lethal 5-FU overdose occurs due to infusion pump errors, dosage miscalculations, accidental and suicidal ingestion of capecitabine.
Methods: 173 patients overdosed with 5-FU or capecitabine (n = 147); or who showed early onset of severe toxicities (n = 26) were treated with uridine triacetate in 2 clinical trials. Patients were to receive uridine triacetate (10g q6h for 20 doses) starting up to 96h post-5-FU/capecitabine. Clinical endpoints included survival, time to resumption of chemotherapy, and safety.
Results: A total of 163/173 (94%) patients treated with uridine triacetate recovered fully (within 30 days), including rapid reversal of severe cardiotoxicity (e.g. multiple cardiac arrests; LVEF of 5%) and neurotoxicity (e.g. coma, encephalopathy, ataxia), in addition to recovery from mucositis and leukopenias. Historical comparators for overdose patients (n = 47) were obtained from publicly-available sources. Of those with outcome data, 38/42 (90%) died. Of the 166 uridine triacetate-treated patients with a diagnosis of cancer, 53 resumed chemotherapy in <30 days (median 19.5 days post-5-FU), indicating rapid recovery from toxicity. Adverse reactions in patients receiving uridine triacetate were vomiting (10%), nausea (5%), and diarrhea (3%).
Conclusions: In these studies, uridine triacetate was a safe and effective life-saving antidote for capecitabine and 5-FU overexposure, and facilitated rapid resumption of chemotherapy.

KEYWORDS Uridine triacetate; 5-fluorouracil; overdose

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177. *Withdrawn*

178. Anaphylactic Shock to IV Patent Blue Dye

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Background: Patent blue dye was historically used to colour lymphatic vessels for lymphangiography and is currently used mostly for sentinel lymph node biopsy. Hypersensitivity reactions occur at rates as high as 1-2%. These are likely IgE-mediated and are usually mild with erythema, hives, urticaria and angioedema. Rarely, hypotension, pulmonary edema and cardiopulmonary arrest have been reported. Intense blue staining of the tissues and urine occur for multiple days. Allergic sensitization may occur through nonmedical exposure to patent blue dye in food colouring (E131), cosmetics and textiles. Some authors suggest preoperative skin testing for patent blue hypersensitivity prior to planned administration and avoidance of in patients with known allergy to food colorant E131.
Case Report: A 25-year-old female was undergoing an elective laparoscopic hysterectomy. Due to a difficult dissection, the gynecologist administered 1 mL of intravenous (IV) patent blue dye to assess for bladder integrity. The patient immediately became profoundly hypotensive to 70/30 and tachycardic to 115. Her face became grossly edematous with tongue and eyelid swelling and was notable for a grey-blue discoloration. The anesthesiologist administered boluses of epinephrine, phenylephrine and vasopressin and initiated an epinephrine infusion. The patient was admitted to the ICU post-operatively for persistent hypotension and an elevated lactate to 8.9. Her anaphylaxis was treated with an epinephrine infusion, methylprednisolone and diphenhydramine. The provincial toxicology service was consulted and advised administration of a 20% lipid emulsion bolus, which produced some hemodynamic stabilization. The patient was gradually

weaned off of the epinephrine infusion over 14 hours with resolution of the lactic acidosis. She was noted to have blue-stained skin, particularly in the periorbital regions, blue urine, generalized pruritis and facial swelling, which all slowly resolved. She was extubated within 23 hours and discharged home in stable condition 48 hours post-operatively. On further questioning, the patient reported allergy to synthetic food dyes.
Case Discussion: IV patent blue produced severe anaphylaxis in our patient. Her prior food dye allergy may have led to sensitization. She recovered with epinephrine, steroids and anti-histamines. To our knowledge, this is the first case to suggest a role for lipid emulsion in hemodynamic stabilization for a dye allergy.
Conclusions: Patients should be screened preoperatively for allergies to non-medical dyes prior to patent blue administration. In cases of anaphylaxis, care is supportive but IV lipid emulsion can be considered.

KEYWORDS Patent blue; dye; lipid emulsion

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179. Unsuspected Clenbuterol Toxicity in a Patient Using Intramuscular Testosterone

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Background: Clenbuterol is a type 3 β-agonist that has been abused by body-builders and other fitness oriented individuals to promote muscle growth and weight loss.
Case Report: A 46 year old male presented to an Emergency Department with dizziness, nausea, and palpitations. He reported using an intramuscular injection of testosterone - Testex Elmu Prolongatum 250mg (Byk Emlu S.A.), from a 2mL sealed glass ampule that was imported from Brazil. Upon arrival the patient was found to have a heart rate of 130 and a blood pressure of 129/66. An EKG featured sinus tachycardia with PR, QRS and QTc intervals of 160, 92, and 608 ms respectively with no ischemic changes. Labs were remarkable for potassium of 2.6 mmol/L and serum glucose of 261 mg/dL. Serum troponin concentrations were undetectable. He was treated for a possible allergic reaction with1L of intravenous (IV) normal saline, 80 mEq oral potassium, 125 mg of IV methylprednisolone, and 2mg of IV lorazepam. The patient developed hypotension of 70/33 mmHg and received an additional 2.5 L of normal saline with no improvement. The patient was transferred to our tertiary care center for further evaluation and care. Upon arrival to our facility, the patient's heart rate was 120 and he remained hypotensive at 105/43 mmHg. We suspected the preparation contained clenbuterol, and he was empirically started on IV esmolol (0.5mg/kg bolus followed by a 50mcg/kg/min drip). Shortly after titration of esmolol, the patient's hypotension and tachycardia improved. He remained on the esmolol infusion for approximately 12 hours. At the time of discharge, his heart rate was 98 bpm and a blood pressure of 120/66 mmHg. Laboratory workup during the patient's hospitalization included a urine comprehensive drug screen (GCMS) positive for caffeine and diphenhydramine. A quantitative caffeine level drawn 1.75 hours after arrival was negative (<1 mcg/mL). Hence, methylxanthine toxicity was deemed unlikely. Analysis performed by NMS Laboratories showed a positive clenbuterol serum level. An unopened glass ampule provided by the patient was sent to NMS Laboratory's Crime Laboratory. It was found to contain boldenone undecylenate, clenbuterol, and vitamin E.
Casse Discussion: Current literature describes clenbuterol exposures, however we are unaware of any literature reporting unintentional exposure and toxicity with intended steroid injection. This case illustrates a novel exposure to clenbuterol β-agonist that was masked in a sealed imported ampule of steroid.
Conclusions: Clembuterol is a β-agonist newly described as an adulterant in anabolic steroid formulations. Clembuterol induced hypotension and tachycardia is successfully treated with infusion of rapid acting β receptor antagonists.

KEYWORDS Dlenbuterol; steroid; testosterone

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180. Hyperthermia with Hyperglycemic Hyperosmolar Nonketotic Syndrome Associated with Aripirazole and Exogenous Insulin

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Background: Malignant hyperthermia with rhabdomyolysis has been reported after treatment in type 2 diabetes with insulin. We describe a similar case in a an adolescent male
Case Report: 14 y.o. presents with hyperthermia, hypotension, respiratory failure, coma. He had 4 days of polydipsia polyuria, 36 hours of lethargy, sedation, abdominal pain. Presents after syncope with the vital signs: T. 105.8, P-187, BP-149/57, RR-45, oxygen saturation-89%, 98.5 kg. He was intubated. No increased muscle tone. He received fluid resuscitation, norepinephrine for hypotension, insulin at 5 U/hr for glucose of >1250 mg/dL, 20 meq of KCl for a K of 2 mmol/L. Temperature 109 F at 2 hours. Insulin discontinued, he was transferred to a tertiary care center.
Home medications: aripiprazole 15 mg daily, lisdexamfetamine 70 mg, fluoxetine 20 mg daily. Parents deny overdose. He had a 40 lb. weight gain over 4 months.
Initial data: Head CT-normal. Na-128 mmol/L, K-3.9mmol/L, HCO3-16 mmol/L, hemoglobin-14.5 GM/dL, pH 7.09.UDS-negative.
Hospital Course:
Day#1: Vitals: T. 105.8 F, P-178, BP-89/39, RR-24, 97% on 40% FiO2. No increased tone or focal neurological findings. Cooling measures led to temperature normalization at 2 hours. Norepinephrine, epinephrine, dopamine, and vasopressin were utilized for hypotension. 4.5 hours after presentation he developed ventricular tachycardia, required CPR with epinephrine, sodium bicarbonate, lidocaine, chest compressions, potassium repletion. ECMO was started for cardiogenic shock. Dantrolene 3 mg/kg was given 8 hours after presentation. Continuous veno-venous hemodialysis (CVVHD) was initiated for electrolyte management and renal failure. Data: EEG-moderate encephalopathy. Echocardiogram: EF-44%, mildly reduced left ventricular systolic function, borderline left ventricular hypertrophy. Glucose-1250 mg/dL, HCO3-14 mmol/L, creatinine-3.5 mg/dL, lactate 4.2 mmol/L, CK-1959 U/L, AST-94 U/L, ALT-52 U/L, ASA/APAP -negative. Ventilator support, CVVHD, epinephrine, norepinephrine, dopamine, vasopressin were continued day 1-10.
Day#2: ECMO continued. Troponin >73 ng/mL.
Day #3: ECMO continued. Dantrolene 2.5 mg/kg given secondary to increased lower extremity tone. No improvement in tone or hemodynamics after dantrolene. Compartment syndrome requiring fasciotomy in left lower extremity.
Day #4: ECMO discontinued. Insulin started at 0.05 U/kg/hr. CPK 80,900. Day #6: Fasciotomy of right lower extremity for compartment syndrome. Amputation of left lower extremity above the knee secondary to ischemia. Day #7-10: Worsening hypothermia, hypotension, hypoxia, metabolic acidosis develop. Day 7 data: CK-404,730 U/L, AST 6,330 U/L, ALT-1631U/L. Day 8 data: Lactate-

19.7 mmol/L. Day #9, care no longer escalated. Death from cardiac arrest on day #10.
Case Discussion: Atypical antipsychotics have been associated with hyperthermic presentations of HHNS. It is unclear if atypical antipsychotics increase risk for hyperthermia in HHNS. This patient was on a sympathomimetic agent, a serotonergic agent. He did not have symptoms of neuroleptic malignant, serotonin, or sympathomimetic syndrome. Insulin may worsen hyperthermia, possibly from the cresol additives. This patient's hyperthermia worsened after insulin . There was no clear clinical change with dantrolene.
Conclusions: Hyperthermia was a presenting sign of HHNS in this patient. It is unclear what role his home medications had. Insulin administration was associated with worsening of his hyperthermia. He died despite aggressive supportive care.

KEYWORDS Hyperthermia; Antipsychotic; Insulin

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181. Survival After Severe Rhabdomyolysis Following Monensin Ingestion

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Background: Monensin is a highly selective sodium ionophore veterinary antibiotic. Due to its narrow therapeutic window, multiple outbreaks of lethal intoxication in many animal species have been reported. Only 2 prior cases of human toxicity from monensin have been reported, both fatal exposures. We present the first case of survival after severe toxicity following monensin ingestion.
Case Report: A 58-year-old man presented for evaluation of vomiting and abdominal pain. Due to delusions that he had CNS toxoplasmosis and inability to find a physician to treat his perceived condition, he purchased monensin, a veterinary antibiotic, from an online foreign distributor. Nine days prior to presentation, he ingested 100 mg of monensin, and he ingested an additional 200 mg the following day. After the second dose, he developed vomiting and abdominal pain, which persisted until presentation. His laboratory studies revealed severe rhabdomyolysis without renal dysfunction. Total creatine kinase (CK) peaked at greater than 100,000 U/L. Echocardiogram on admission showed no wall motion abnormalities and an ejection fraction of 69%. Despite several days of severe CK elevation, his CK decreased to 5,192 U/L after 16 days of aggressive hydration and sodium bicarbonate therapy. Repeat echocardiogram 8 days after admission remained normal with an ejection fraction of 56%. He remained hemodynamically stable throughout his admission, and he was discharged to a rehabilitation facility due to severe weakness from his severe rhabdomyolysis.
Case Discussion: Reports are limited on acute clinical effects after human exposure to monensin. It is known to cause severe skeletal and cardiac muscle rhabdomyolysis and necrosis. Prior studies have shown monensin's toxicity is due to increases in intracellular sodium concentrations. The monensin ionophore complexes with sodium and carries it through myocyte lipid membranes. Monensin also upregulates the Na+/H + antiporter, increasing intracellular sodium and pH, thereby causing increased Ca2+ release. Additionally, increased intracellular Na + leads to Ca2+ mediated Ca2+ release via the Na+/Ca2+ exchanger. Toxic levels of Ca2+ lead to phospholipase and proteolytic enzyme release, resulting in myocyte necrosis. In 2 prior reported cases of human toxicity, severe rhabdomyolysis occurred approximately 5 days after ingestion and death followed within 11 days of ingestion. To date, no effective therapy or antidotal treatment has been described.

Table 1. Laboratory Values

	Initial	Peak	Discharge
Creatine Kinase (U/LO	77,010	>100,000	5,192
Serum Creatinine (mg/dL)	0.82	0.88	0.44
Troponin-I (iig/mL)	<0.03	0.16	0.05

Conclusions: We describe the first reported case of survival from severe rhabdomyolysis following monensin ingestion. Monensin is a veterinary medication, and it is not FDA approved for human use. Though poorly studied in humans, this case demonstrates the severe harm that may occur following ingestion.

KEYWORDS Monensin; Rhabdomyolysis; veterinary

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182. Compounded Topical Ketamine Ointment Causing Systemic Toxicity in a Pediatric Patient

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Background: Compounded topical medications are becoming more prevalent in the United States, and while presumably safe, health care providers need to be aware that there is the possibility of transdermal absorption significant enough to cause systemic toxicity, especially if misused or abused.
Case Report: A 16 year old boy without significant past medical history presented to the emergency department with altered mental status and bradycardia. The night prior to presentation, he was having a headache while getting ready for a hockey game and was given a teammate's prescription ointment originally prescribed for postconcussive headache containing ketamine 10%, meloxicam 1%, baclofen 2%, bupivacaine 1%, clonidine 0.2%, cyclobenzaprine 2%, gabapentin 6%, verapamil 10%, and pentoxifylline 3%. The patient applied this ointment over his forehead and scalp, played his hockey game without problems, but still had a headache afterward so reapplied the ointment. He played a second game and was noted to behave oddly. He started a fight for no reason and acted confused, so he was removed prior to the end of the second game. Upon questioning, he was disoriented to date and stated that he had just surfed at a lake. There was no head trauma. The next morning his symptoms persisted prompting parents to seek care. Initial vitals were blood pressure 120/61, heart rate 50, and an EKG showed sinus bradycardia. Blood counts and comprehensive metabolic panel were normal, and a urine drug screen was negative. The patient was admitted overnight, observed, and discharged the next day after returning to baseline.
Case Discussion: In the USA, compounded drugs (customized medication mixtures not commercially available) are not rigorously regulated and not clinically evaluated for safety or efficacy, leading to potentially dangerous side effect profiles. Ketamine, a common anesthetic that acts via NMDA receptor antagonism, has been shown to be absorbed systemically from topical formulations, and when compounded with other drugs affecting the central nervous system (e.g. clonidine, cyclobenzaprine, gabapentin), can lead to significant patient impairment as in our case.
Conclusions: We report a case not previously described in the literature of systemic toxicity after topical application of a compounded topical ointment containing ketamine, meloxicam, baclofen, bupivacaine, clonidine, cyclobenzaprine, gabapentin,

verapamil, and pentoxifylline. Health care providers need to be aware that similar formulations are readily available to athletes and may be a cause of altered mental status.

KEYWORDS Compounded medications; ketamine; clonidine

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183. Stroke Mimic: Anticholinergic Toxicity Received tPA

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Background: Anticholinergic toxicity is one of the most common toxidromes encountered. Many drugs and plants are known to cause this classic toxidrome presentation which includes hyperthermia, anhidrosis, mydriasis, and delirium. The case described is one of anticholinergic toxicity secondary to iatrogenic atropine administration which was originally mistaken for stroke-like symptoms.
Case Report: A healthy 45 yo female was brought in by ambulance after she was found confused. Per family, last known normal was approximately two hours prior to arrival. Vital signs were: Temp 36.3C, HR 130, BP 119/97, RR 20, SpO2 97%. The patient was awake but did not follow commands. Her exam was remarkable for tachycardia, aphasia and dysarthria without drift or ataxia. She had unremarkable labs and a negative head CT. Neurology assessed that she was suffering from an acute cerebrovascular accident, then received tissue plasminogen activator (tPA). She was admitted to the ICU, her symptoms resolved after 24 hours, and she was discharged three days later. Upon regaining her mental faculties, the patient revealed she had visited her primary care physician <1 hour before presentation and received an energy injection that was supposed to contain B vitamins and adenosine monophosphate. The energy preparation had been produced by a compounding pharmacy. Investigation of the injection later revealed a compounding error had occurred where atropine (92 mg/mL) was used instead of adenosine. It is estimated the patient received 46 mg of atropine via injection.
Case Discussion: Atropine is a competitive antagonist of muscarinic acetylcholine receptors and affects smooth muscle, secretory glands, as well as the heart and the central nervous system. Clinically, patients will present with anhidrosis, hyperthermia, tachycardia, and urinary retention, as well as hallucinations, dysarthria, and delirium. Treatment is revolved around supportive care with benzodiazepines and physostigmine for central symptoms. Data suggests that doses of 49.8mg (+/− 4.5mg) within 24 hours are consistent with life threatening toxicity; however, there is minimal long term sequela if toxicity is managed appropriately.
Conclusions: Due to a compounding error, this patient presented acutely with anticholinergic toxicity, however her symptoms were first misinterpreted as an early stroke. With the advent of tPA use for acute stroke, making a proper diagnosis of a patient with anticholinergic toxicity is essential. This case illustrates the importance of early recognition of potential anticholinergic syndrome given the wide differential including CVA, in which tPA treatment therapy has a high side effect profile that includes intracerebral hemorrhage and death.

KEYWORDS Atropine; anticholinergic syndrome; adenosine monophosphate

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184. Naloxone Administration in Treatment of Opioid Toxicity Leading to Pulmonary Edema

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Background: Pulmonary edema has been infrequently reported in patients receiving naloxone for reversal of opioid toxicity. The etiology of this outcome is debated and may be related to abrupt reversal of toxicity and catecholamine surge in an opioid dependent patient, constituents within the opioid product, or negative intrathoracic pressure with an obstructed airway.
Case Report: We describe a case series of six consecutive patients evaluated over a five day period for clinical features consistent with an opioid toxidrome. All six individuals received varying doses of naloxone for suspected heroin injection or insufflation with respiratory depression. One patient received intramuscular naloxone from a bystander and three patients were treated by prehospital providers. Three patients, including one who had received bystander naloxone, were treated with intravenous naloxone in the Emergency Department for respiratory depression. Prehospital chest compressions were performed on three patients prior to naloxone administration. One patient was cardioverted by EMS for a narrow complex tachydysrhythmia after receiving naloxone. All six patients developed severe pulmonary edema and, in one case, diffuse alveolar hemorrhage with hemoptysis. Four required intubation while one required noninvasive positive pressure ventilation. The sixth patient required 15L nonrebreather facemask oxygen supplementation in the ICU for three days. Urine testing by gas chromatography/mass spectrometry did not identify adulterants suspected to cause pulmonary edema.
Case Discussion: Pulmonary edema following naloxone reversal of heroin-induced respiratory depression is rarely reported in comparison to its overall use. We report multiple cases presenting to a single hospital system over five days. Each of these patients had a history of opioid dependence. Possible contributing factors could include a heightened awareness of this syndrome leading to more consistent identification or a toxic effect of unidentified adulterants. Additionally, the incidence of heroin use is continuing to rise which would be expected to be associated with a concomitant rise in morbidity. Finally, with the advent of widespread distribution of naloxone to first responders and bystanders, naloxone administration may be performed on a larger number of patients and potentially at larger doses leading to a higher prevalence of adverse effects.
Conclusions: Pulmonary edema after naloxone administration is a rarely reported event. However, due to the increasing prevalence of opioid abuse, widespread presence of adulterants in opioid preparations, and the availability of naloxone to prehospital providers and bystanders, occurrence of this complication may be seen more frequently.

KEYWORDS Naloxone; Pulmonary Edema; Heroin

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185. Olanzapine long-acting injection (OLAI) and post-injection delirium/sedation syndrome (PDSS): two case reports

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Background: Despite advances in the treatment of schizophrenia with newer atypical antipsychotic agents, a high rate of poor adherence to therapy is described and the long-acting depot injections of antipsychotic agents may mitigate that. Olanzapine long-acting injection (OLAI) is a depot formulation (pamoate salt) administered by deep intramuscular injection every 2-4 weeks. Local injection site-related AEs and post-injection delirium/sedation syndrome (PDSS) were observed. PDSS occurred in approximately 1.4% of patients. Symptoms onset ranged from immediate to 5 hours post injection, with a median of 25 minutes. We report two cases of PDSS after olanzapine long-acting injection.
Case Reports: Case 1 - A 42 year-old schizofrenic male developed initial hypotension and coma about 30 minutes after OLAI 405 mg injection. In ED the patient was drowsy but partially awakened with pain stimulation with BP 150/72 mmHg and HR 98/min. EKG showed sinus rhythm with normal QRS and QTc (462 ms). The patient, admitted to intensive care unit, showed a progressive improvement of consciousness and discharged on the sixth day. The olanzapine plasma levels at 2, 9, 21, 48, 72, 96 and 110 hours post-injection were 800 ng/mL (therapeutic up to 50 ng/mL), 948 ng/mL, 696 ng/mL, 313 ng/mL, 264 ng/mL, 148 ng/mL, and 100 ng/mL respectively. Case 2 - A 50 year-old schizofrenic male developed generalized tonic clonic seizures about 15 minutes after OLAI 405 mg injection. Sedation with propofol and midazolam and orotracheal intubation was performed. Head CT scan, EEG and urine screening for drugs of abuse were negative. The patient showed a progressive improvement of consciousness, was extubated on the second day and discharged on the eighth day. The olanzapine plasma levels at 2, 6, 48, 110 and 192 hours after the injection were, 454 ng/mL, 513 ng/mL, 400 ng/mL. 390 ng/mL, 78 ng/mL respectively.
Case Discussion: The intramuscular in-situ rate of dissolution of the olanzapine pamoate salt is slow, but the absorption of the dissociated free base olanzapine in muscle tissue is very fast. PDSS results from unintentional intravascular injection or blood vascular injury after intramuscular OLAI administration. Olanzapine plasma levels in PDSS were reported up to 650 ng/mL within 6 hours while in our first case we found a peak level of 948 ng/mL 9 hours after injection.
Conclusions: Extended release formulations is a valid alternative for reducing the rate of relapse in patients with schizophrenia. PDSS is a serious adverse event observed after approximately 0.07% of injections. An increased awareness among physicians about PDSS risks is needed, and a proper injection technique and 3-hour close monitoring after OLAI injection should be instituted. Because of there are not antidotes and depurative treatments, PDSS management is only supportive.

KEYWORDS Olanzapine long-acting injection; Post-injection delirium/sedation syndrome (PDSS); Adverse drug events

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186. Fatal Lactic Acidosis Associated With Tenofovir Therapy for Chronic Active Hepatitis B Infection

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Background: Tenofovir, a nucleotide analogue reverse transcriptase inhibitor (NRTI), is approved for the treatment of human immunodeficiency virus (HIV) infection and, more recently, chronic hepatitis B infection. Severe or fatal lactic acidosis has been rarely reported in the setting of tenofovir therapy for HIV. We report what we believe to be the first case of fatal lactic acidosis in a patient treated with tenofovir for chronic hepatitis B infection.
Case Report: A 33-year-old Chinese male with a history of chronic active hepatitis B infection and non-insulin dependent diabetes mellitus presented to an outside emergency department (ED) for altered mental status. Two weeks prior to presentation he had been started on tenofovir for hepatitis B. In the ED he was to have a blood glucose of 17 mg/dL and was treated with 2 ampules of D50. Vital signs were pulse 162, BP 153/71, RR 18, and 97% on room air. EKG showed sinus tachycardia. Laboratory studies were remarkable for a normal WBC of 7.9×10^3/uL, thrombocytopenia of 16×10^3/uL, BUN of 33 mg/dL, creatinine of 1.9 mg/dL (estimated GFR 44 mL/min), INR of 3.2, ammonia of 53 umol/L, serum lactate of 8.7 mmol/L, positive urine leukocyte esterase and nitrites, and urine WBCs too numerous to count. He was administered 4300 mL NS, Zosyn, vancomycin, and admitted to the ICU. On the day of admission, his blood pressure decreased to 90 mmHg and he was started on norepinephrine infusion, intubated for airway protection and was transferred to our ICU. Continuous venovenous hemodialysis (CVVHD) was initiated early on hospital day (HD) #2. Despite the above therapies, serial serum lactates continued uptrending and peaked on hospital day (HD) #2 at 24.9 mmol/L. Serial blood gases revealed downtrending pH reaching a nadir of 6.824 (pCO2 61.2 mmHg). The patient developed hypotension despite epinephrine and norepinephrine infusions and he expired late on HD #2.
Case Discussion: Tenofovir is primarily eliminated by the kidneys. Tenofovir may cause hepatic mitochondrial toxicity and increased lactate production, in a concentration-dependent fashion, and should be avoided in patients with renal insufficiency. The fatal lactic acidosis in this case was multifactorial, due to sepsis, chronic liver disease, and tenofovir. We believe tenofovir was a significant contributing factor due to the renal insufficiency, recent initiation of the medication, and uptrending serum lactate despite aggressive fluid resuscitation, broad spectrum antibiotic therapy and CVVHD.
Conclusions: We present the first reported case of fatal lactic acidosis associated with tenofovir therapy for hepatitis B. It is critical for clinicians treating patients that are on NRTIs to be aware of the possibility of severe lactic acidosis.

KEYWORDS Tenofovir; hepatitis B; lactic acid

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187. Severe drug induced liver injury (Hy's law) associated with teriflunomide therapy

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Background: Teriflunomide (Aubagio®) is currently approved for treatment of relapsing forms of multiple sclerosis. It is the principal active metabolite of leflunomide, which has been associated with fatal acute liver failure. In phase II and III clinical trials with teriflunomide, serious adverse events (SAE) of elevated liver function tests have been observed. In one case demonstrating Hy's law, a patient developed ALT 32 times the upper limit of normal (ULN) and jaundice 5 months after initiation of oral teriflunomide 14mg a day. Hy's law refers to severe drug-induced liver injury (DILI) and is defined by an ALT/AST $\geq$3 times the ULN (indicative of hepatocellular injury) and total bilirubin $>$2 times the ULN (clinical jaundice) with the absence of initial findings of cholestasis and no other alternative explanation for the combination of increased ALT and total bilirubin levels. Teriflunomide is eliminated mainly through direct biliary excretion with a median t1/2 of approximately 18 days. Cholestyramine, a bile acid sequestrant that prevents enterohepatic circulation and promotes biliary excretion of teriflunomide, was suggested to enhance its elimination. We report the first case of severe DILI occurring within a relatively short time after initiation of teriflunomide with resolution of signs and symptoms after drug discontinuation and concomitant treatment with cholestyramine.
Case Report: A 48 year old man with a history of relapsing-remitting multiple sclerosis presented to the emergency department with a history of malaise, fatigue, subjective chills, generalized pruritis, and scleral icterus for four days. He had been initiated on teriflunomide 7mg PO daily two weeks prior to presentation. Initial laboratory values were notable for a AST 346, ALT 478, alkaline phosphatase 236, total bilirubin 6.2, and direct bilirubin 1.9. His hospital work-up provided no alternative explanation for the laboratory abnormalities found. Teriflunomide was discontinued and cholestyramine 8mg PO three times a day was initiated for 11 days. On an outpatient visit after completion of the medication, he was asymptomatic and his liver function tests had normalized.
Case Discussion: An R value of 2 was calculated from the patient's laboratory values, suggesting a mixed hepatocellular and cholestatic picture, but hepatocellular predominant. A Roussel Uclaf Causality Assessment Method (RUCAM) of 8 was calculated, suggesting a "probable" association between teriflunomide and the observed clinical picture of severe DILI. This case was reported to the FDA Safety Information and Adverse Event Reporting Program.
Conclusions: Medical toxicologists should be acquainted with Hy's law and clinicians should be aware that severe DILI could be a rare serious side effect of teriflunomide. Cholestyramine may be helpful in the treatment of such cases.

KEYWORDS Teriflunomide; Hepatotoxicity; Adverse reaction

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188. Serotonin Syndrome Following High-Dose Dextromethorphan for the Treatment of Methotrexate-Induced Neurotoxicity

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Background: Methotrexate (MTX) increases the concentrations of homocysteine (HCY) in the cerebrospinal fluid (CSF). Although the neurotoxicity of HCY has not been well documented, CSF levels of HCY have been found to be elevated in patients with methotrexate-induced neurotoxicity (MTX-nTOX). Because HCY and its metabolites are excitatory agonists at the N-methyl-D-aspartate receptor (NMDA-r), NMDA-r antagonists may provide protection or reversal of MTX-nTOX. Dextromethorphan (DM), a noncompetitive NMDA-r antagonist, has been shown to alleviate MTX-nTOX when given in doses high enough to penetrate the CSF. However, DM also inhibits reuptake of serotonin. We present a case of neurotoxicity related to intrathecal MTX administration, treated with high dose DM, which resulted in serotonin syndrome (SS).
Case Report: A 19 year-old male presented with aphasia, altered mental status and right hemiparesis approximately one week following intrathecal MTX administration. After neuroimaging, a diagnosis of MTX-nTOX was made and he was treated with 80mg (1mg/kg) of DM. Soon after this, he became agitated, tachycardic, hypertensive (160/110), and febrile (38.8C). Rigidity in his jaw and lower extremities was noted and he had spontaneous myoclonus. Symptoms improved with lorazepam, but the uncontrollable jaw clenching, combined with decreased responsiveness after receiving benzodiazepines (BZ), ultimately resulted in intubation. Temperature increased to 40.4C when fentanyl was used for sedation. He improved with additional BZ and dexmedetomidine, cooling blankets and esmolol. Hypotension developed later that day and vasopressor support with dopamine and epinephrine was required. He was treated with cyproheptadine 4mg followed by 2mg every 2 hours for 24 hours. The patient was extubated on hospital day 2; vitals had stabilized by hospital day 3. There was no history of serotonergic medication use prior to this encounter.
Discussion: At currently recommended doses, DM is a safe antitussive agent. It is rare to see DM alone cause SS, but it has been reported. With much higher doses (1-2mg/kg) utilized to treat MTX-nTOX, the potential for adverse reactions increases. This patient had a rapid onset of agitation, tachycardia, clonus/muscle rigidity, and hyperthermia after receiving a high dose of DM, meeting both Sternbach and Hunter criteria for SS.
Conclusions: While DM is typically considered a safe over-the-counter medication, it has been implicated in causing SS when used in high doses, such as those required to reverse MTX-nTOX. Physicians, especially oncologists, should be aware of the potential for this reaction when administering DM for this indication.

KEYWORDS Serotonin syndrome; dextromethorphan; methotrexate

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189. Medical errors and adverse reactions in elderly patients: which are the major risks?

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Background: Elderly patients are particularly at risk of medication errors (MEs) and adverse drug reactions (ADRs) with implications on their health conditions and costs for public health.
Six Poison Control Centers have run a pharmacovigilance project in order to identify the most frequently involved active ingredients, possible emerging risks, and the pharmaceutical categories susceptible to preventive strategies.
The objective of this contribution is to present observations about data collected between June 2012 - May 2015.
Methods: Data about age, gender, active ingredients, dosage, cause of medical errors, and clinical outcome according to the Poisoning Severity Score (PSS) were obtained from patients' medical records. Additional information was retrieved during a follow-up phase. The only inclusion criterion was the age to be $\geq$60 years.
Results: Overall 3,408 patients met the inclusion criterion. Among these cases 3,046 (89.4%) were MEs and 362 (10.6%) ADRs. Considering MEs, 1,945 (63.8%) patients were females, 1,084 (35.6%) males, 17 (0.6%) unknown. The typology was a dosage error in 1,453 (47.7%) cases, wrong drug in 1,150 (37.7%), wrong route of administration in 288 (9.6%), expired drug in 36 (1.1%), wrong preparation in 7 (0.2%), combinations of multiple errors in 34 (1.1%), not specified in 78 (2.6%) cases.
Symptoms were severe in 26 cases (0.9%), moderate in 161 (5.3%), mild in 444 (14.6%), absent in 2,385 (78.3%), not assignable in 30 patients (0.9%). The most frequently involved drug was tiotropium bromide (n 165; 4.9%) followed by tramadol hydrochloride (n 105; 3.1%) and tosylchloramide sodium (n 90; 2.7%). Among severe cases, the most frequent errors involved boric acid (n 5) and clonazepam (n 4). The only fatal case was caused by vinorelbine.
Considering ADRs, distribution by gender was as follows: 234 (64.6%) females, 128 (35.4%) males. PSS scores for ADRs were: severe 76 (21.0%), moderate 155 (42.8%), mild 128 (35.4%), 3 (0.8%) not assignable. The drugs most frequently involved were: lithium carbonate (n 44; 10.1%), digoxin (n 36, 8.3%), metformin (n 15; 3.4%). Considering only severe cases, the majority of ADRs were due to digoxin (n 20), metformin (n 11), lithium carbonate (n 6). Fatal cases were 19, mostly due to digoxin (n 5) or to different neurological and anti-diabetic drugs.
Conclusions: While MEs appear to be more frequent and largely inconsequential, ADRs in elderly patients can be severe in a significant percentage. Digoxin, neurological and anti-diabetic drugs must be prudently prescribed after having considered pathophysiologic factors that influence their effect.
Polytherapy and the decline of metabolism can be major factors and must be considered carefully.

KEYWORDS Medical errors; Adverse reactions; Geriatric care

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190. Accidental pediatric paliperidone ingestion resulting in prolonged profound tachycardia

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Background: Paliperidone is an atypical antipsychotic that was approved by the Food and Drug Administration in 2006. It is approved for adolescents as young as 12 years of age as well as adults to treat schizophrenia. Its use in younger children is not recommended and there is scant information on clinical effects in children.
Case Report: A 7 year old, 31kg, female with a history of prematurity, patent ductus arteriosus, and attention deficit hyperactivity disorder presented with facial twitching and neck spasms, consistent with a dystonic reaction. It was found that her pharmacy had accidentally dispensed paliperidone 3mg tablets instead of guanfacine, and she had received this dose daily for 3 days. Her dystonic reaction resolved with diphenhydramine (DPH), however she had persistent tachycardia, mydriasis, and flushing all of which were present prior to DPH administration. Twenty-six hours after her last dose of paliperidone her exam was notable for: heart rate 176 (190s with stimulation), blood pressure 106/69 mmHg, temperature 37.2oC, normal mental status, no clonus, no muscular rigidity, pupils 5mm and reactive. EKG showed sinus tachycardia with normal intervals. She did not have orthostatic changes to her blood pressure and her heart rate did not decrease with a trial of benzodiazepines. She continued to have tachycardia with minimal stimulation at 64 hours after her last dose, however she remained otherwise asymptomatic and was discharged with a heart rate 136 with close primary care follow up the following day.
Case Discussion: Paliperidone is an atypical antipsychotic that is the active metabolite of risperidone, however the tablet is a trilayer core, thus resulting in a sustained release delivery system. Overdose data is limited. A case report of an intentional overdose in a teenager also reported tachycardia with a rate in the 190s over 24 hours after ingestion. The mechanism of tachycardia is not clear. Alpha blockade may play a role, however our patient had no orthostatic changes or hypotension making this unlikely to be the sole mechanism. Literature on paliperidone does not report affinity for the muscarinic acetylcholine receptor, but she showed some signs consistent with anticholinergic findings (flushing, mydriasis) which may have contributed to her tachycardia without altered mentation or urinary retention to suggest a full anticholinergic toxidrome. Guanfacine withdrawal was also considered, but her lack of hypertension made this less likely. Her tachycardia seemed out of proportion to any of the above mechanisms alone. It is possible that there is direct cardiac chronotropic effect or a yet to be elucidated mechanism.
Conclusions: Paliperidone toxicity in children may result in prolonged, delayed tachycardia from unclear, likely multifactorial mechanisms.

KEYWORDS Paliperidone; tachycardia; pediatric

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191. Epidemiology of Activated Charcoal Use in Texas from 2000-2014

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Background: Activated charcoal has been used for at least a century for the decontamination of toxins. It has been previously reported that the use of activated charcoal has been declining for various factors. We are reporting data from the Texas Poison Center Network for the past fifteen years.
Methods: We performed a retrospective chart review of all patients that were treated with activated charcoal reported to the Texas Poison Center Network (TCPN) from 2000-2014. Exposures involving multiple substances and those not followed to a final medical outcome were included. The distribution of cases by selected demographic and management factors was determined.
Results: A total of 2,536,367 cases were reported to the TCPN during this time. Of these cases, 202,266 received activated charcoal (8%). Since 2001, there has been a steady decline of activated charcoal administration with a peak of 11.0% in 2001 to 5.6% in 2014. Female comprised 60% and males 39.9%. The activated charcoal rate per 1000 population was 12.55% in rural counties and 9.09% in urban counties. Intentional exposures accounted for 70.5% and unintentional exposures were 28.4%. Single substances were reported in 62.7% of the exposures. The distribution by medical outcome was no effect (38.9%), minor effect (27.1%), moderate effect (19.7%), major effect (4.2%), death (0.1%), not followed-nontoxic (0.2%), not followed-minimal effects (2.1%), unable to follow-potentially toxic (6.8%) and unrelated effects (0.9%). The most commonly reported substance was sedatives/hypnotics/antipsychotics compromising 25.4% of total exposures and 15.1% of single substance exposures. Acetaminophen containing products were reported in 11.6% of total and 7.3% of single substance exposures. Cardiovascular drugs comprised 9.2% of total and 7% of single substance exposures. Non-steroidal anti-inflammatory agents comprised 9.2% of total and 6.1% of single substance exposures. Selective serotonin reuptake inhibitors comprised 9.1% of total and 4.1% of single substance exposures. Antihistamines comprised 8.5% of total and 6.7% of single substance exposures.
Conclusions: The limitations of this study are that there are no comparison of single dose activated charcoal vs multiple dose activated charcoal. This is a retrospective study, with reporting bias and follow up bias. The specialists in poison information may have miscoded cases and the charts may have been incomplete. The TPCN data is consistent with previously reported studies showing a decrease in utilization of activated charcoal in the poisoned patient over the last fifteen years.

KEYWORDS Epidemiology; Charcoal; Texas

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192. Pulse check on gastric lavage: Agonal rhythm

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Background: Because of limited evidence of efficacy, indications for the use of gastric lavage (GL) are exceedingly narrow and should not be routinely employed. Despite contemporary practice, occasionally acute care providers still desire to employ GL. Therefore, we designed a study to assess the practice patterns and availability of GL by surveying producers and purchasers of GL kits and the physicians who use them.
Methods: This was an IRB exempted study. Members of the Virginia College of Emergency Physicians were surveyed about their familiarity, perceived equipment availability, and practice patterns regarding GL. Hospital supply services in central Virginia were then surveyed regarding stocking and purchasing patterns of GL kits. Finally, device manufacturers were queried regarding sales data for GL kits.
Results: The survey was distributed to 1,060 members (754 attendings and 306 residents). A total of 75 (7%) emergency physicians responded to the survey. Of these, 50% stated they have used a GL kit; 19% last used a GL kit greater than five years ago; 34% last used a GL kit greater than 10 years ago; 42% described GL as never indicated or only if instructed by a toxicologist or poison center; 81% reported having never used or no longer routinely using a gastric lavage kit. Only 5% described gastric lavage as standard of care for poisoned patients and only 20% felt it changed the outcome. 7% reported being unable to perform GL due to lack of equipment and 20% reported being unable to perform GL due to lack of nursing staff knowledge, though 49% reported being comfortable enough with the technique themselves to provide instruction on GL. Of the 43 hospitals surveyed, 38 (88%) responded. Of these, only two smaller community hospitals described still stocking GL kits, though one noted it had been more than five years since the last one was purchased. Of the 36 who did not stock gastric lavage kits, five reported using a small bore nasogastric tube for GL. One publically traded device manufacturer responded to our inquiries and reported having sold approximately $10,000 of kits in 2015 in the mid-Atlantic region (DE, MD, NJ, PA, SC, and VA).
Conclusions: The majority of emergency medicine physicians surveyed have never used or no longer routinely use a GL kit. Many that have performed GL last did so 5-10 years ago. GL is not perceived as standard of care and most believe it does not change outcome. Most hospitals in our study do not actively purchase or stock GL kits, though one manufacturer reported continued sales in this geographic region. Our results reflect the local environment of GL use in one state, and may not be true nationwide.

KEYWORDS Gastric lavage; GI decontamination; Survey

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193. Adverse Events from Physostigmine: An Observational Study

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Background: Physostigmine is a short-acting acetylcholinesterase inhibitor used to reverse delirium from acute antimuscarinic toxicity. A culture of reluctance to use physostigmine has persisted in acute care medicine; likely stemming from cases of seizures or rare cardiac arrest temporally observed with physostigmine. The risk of adverse drug events from physostigmine in contemporary medical practice is unknown. The aim of this study was to evaluate adverse drug events with physostigmine.
Methods: Cases of physostigmine use from 2007 - 2014 were retrospectively identified from electronic medical records at a tertiary medical center. Patients ≥18 years old, with suspected antimuscarinic toxicity, and who received physostigmine were included. Charts were abstracted by two independent reviewers and reconciled by the principal investigator. Clinical data included: suspected antimuscarinic overdose agent, CNS stimulation score,

vital signs, electrocardiogram (ECG) results, and physostigmine dose. Outcome data included: nausea, vomiting, seizures, respiratory distress, dysrhythmia and cardiac arrest. Of 78 patients identified, 29 were excluded as they did not receive physostigmine, 9 for age, and 2 for missing data. Complete data was available for 38 patients.

Results: Of the 38 patients, 21 (55%) were male with a mean age of 35 years (range 18 to 68, SD 14.3). Antimuscarinic overdose was intentional in 23 (61%) cases. Thirty-five (92%) of the cases presented with delirium and 26 (68%) with agitation. Twenty five (66%) of the patients received benzodiazepines before receiving physostigmine with no improvement in their delirium. The initial dose of physostigmine was 2 mg in 19 (50%) of the cases. Twenty (53%) patients received additional doses of physostigmine, with a mean total dose of 2.9 mg (SD 0.6). Thirty-two (84%) patients had improvement in their CNS stimulation score after physostigmine administration. Physostigmine was associated with a 3% incidence of nausea/vomiting and 3% respiratory distress requiring bronchodilators. Physostigmine treatment was also associated with a 17 beat/minute decrease in heart rate (range 69-103, SD 18.9), but no other significant change in vital signs. No seizures or cardiac arrest occurred. No clinically significant changes in ECG parameters were identified however, only 16 (42%) patients received a post-physostigmine ECG.

Conclusions: The majority of patients in this study received 2 mg of physostigmine and more than half received additional dosing. Overall, physostigmine improved CNS stimulation scores and decreased heart rate. No seizures or cardiac arrest occurred in this series.

KEYWORDS Physostigmine; Antimuscarinic; Antidote

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194. Poison Center Recommendations for Gastrointestinal Decontamination after Oral Overdose

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Background: Use of gastrointestinal decontamination (GID) techniques has declined in recent years. From 1995 to 2014, the proportion of oral overdose cases reported to US poison centers (PCs) receiving activated charcoal (AC) and gastric lavage (GL) decreased from 7.7 to 2.1% and 3.6 to 0.9% respectively, while use of whole bowel irrigation (WBI) remained low at 0.8%. As a result, emergency department providers (EDPs) may have less experience with these techniques and rely on guidance provided by the PC. The aim of this study was to assess similarities and differences in GID recommendations among PC directors.

Methods: An electronic survey link was emailed to managing and medical directors of all 55 US PCs. They were invited to share the link with affiliated toxicologists who advise hospital caregivers on patient management. Directors were queried about their PC's written guidelines related to GID, and factors influencing their recommendations for or against these treatments.

Results: The 60 respondents included 33 medical directors, 21 managing directors, and 3 unspecified. The majority (70.0%) indicated that their PC has a written guideline addressing AC administration. The most common reason for recommending against AC (cited by 98.3% of respondents) was ingestion of a substance that was poorly adsorbed to AC, or for which AC was contraindicated (e.g., caustics). Additional reasons for recommending against AC included altered mental status (91.7%), late presentation (90.0%), and vomiting (83.3%). 50.9% indicated that their PCs sometimes recommend GL, primarily for early presentation after potentially life-threatening ingestions. Approximately half (54.4%) reported their PC had a written guideline for WBI. 14.0% reported that WBI was addressed in other PC guidelines such as those for iron or lithium. Respondents would recommend WBI for metal salt ingestion (88.1%), overdose of potentially dangerous modified release products (81.4%), and body stuffing (74.6%). Many directors described an individualized approach to formulating GID recommendations. Examples included recommending AC in severe intoxications despite relative contraindications, or WBI in iron overdose cases but only when pills were seen on x-ray. Others noted that although rarely recommending GL, they would consider it in early presenters after highly toxic ingestions without effective antidotes, such as colchicine.

Conclusions: Most PCs have written guidelines addressing GID, however survey responses suggest that PC directors rely more on clinical judgment and consider multiple factors in formulating recommendations for or against GID procedures. The complexity of these decisions underscores the importance of EDP consultation with PCs as opposed to reliance only on written guidelines.

KEYWORDS GI decontamination; Poison Center; survey

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195. Case report: Failure of Idarucizumab in The Treatment of a Patient with Life-Threatening Hemorrhage

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Background: In 2015, the FDA approved idarucizumab (Praxbind®) for life-threatening, or uncontrolled, bleeding from dabigatran. Two studies (one in healthy volunteers and one in patients requiring urgent reversal) found that idarucizumab immediately corrected coagulation values, including thrombin time, PT, aPTT, and ecarin clotting time. Clinically observed cessation of bleeding took over 11 hours.

Case Report: A 65 year-old male on dabigatran for atrial fibrillation presented to the ED for generalized weakness and shortness of breath which began 45 minutes prior. He reported 3 days of black stools. Initial vital signs were significant for a blood pressure of 74/52 mm Hg, an irregular pulse of 122 beats per minute, and a respiratory rate of 22 breaths per minute. He appeared ill, pale, and had melena on digital rectal exam.

A nasogastric tube was placed with immediate return of 300 ml of bright red blood and failed to clear after 700 ml of 0.9% normal saline. Packed red blood cells were emergently administered, along with initiation of a pantoprazole infusion. The TT was elevated at 120 seconds (Table 1). The patient received idarucizumab 5 grams IV. Repeat TT following idarucizumab administration was 20.4 seconds. The patient was intubated and underwent emergent esophagogastroduodenoscopy in the ED, which demonstrated active duodenal hemorrhage. After the EGD failed to control bleeding, Factor Eight Inhibitor Bypassing Activity (FEIBA®) was administered along with additional units of PRBCs. Emergency angiography with embolization of the gastroduodenal artery was performed. Following cessation of bleeding, he was transferred to the ICU.

Case Discussion: In our patient, the TT improved significantly in less than one hour after idarucizumab administration. Additional

Table 1. Laboratory values

Lab value	Initial	After Idarucizumab administration	Reference range
Platelet	106	76	153-367 K/mcl
Prothrombin	23.4	13.3	12.1-15.0 seconds
INR	2.0	1.0	
Partial thromboplastin time	74	70	25-38 seconds
Fibrinogen	238	–	216-438 mg/dl
Thrombin time	120	24	15-19 seconds
TEG clotting time	9.4	3.6	5.0-10.0 minutes
TEG coagulation index	−0.9	3.1	−3.0-3.0
TEG Fibrinogen activity	70.7	74.4	53.0-72.0 degrees
TEG K time	1.3	1.1	1.0-3.0 minutes
TEG platelet aggregation	66.3	65.2	50.0-70.0 minutes
TEG lyse 30	2.6	0.0	0.0-7.5%

coagulation studies normalized with the exception of aPTT, TEG, and fibrinogen activity. Despite near normalization of the TT, bleeding persisted and the patient required emergent EGD, FEIBA®, and angiography with embolization to achieve homeostasis. The patient did not have any apparent cause of coagulopathy other than dabigatran ingestion that would explain the initial elevated aPTT.

Conclusions: In our patient, idarucizumab has successfully decreased TT to slightly above upper limit of normal immediately after administration. However, this did not lead to hemostasis of the source of bleeding.

KEYWORDS Dabigatran; idarucizumab; thrombin time

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196. Assessment of the Barriers to Naloxone Kit Distribution and the Perceptions of Layperson Naloxone Use in a Single Emergency Department (ED)

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Background: Layperson administration of naloxone is essential for the prevention of opioid overdose deaths. Prior to implementing a naloxone kit program, barriers to distribution and health care provider (HCP) perceptions must be characterized. The objective of this study is to characterize the potential barriers to naloxone kit distribution and the perceptions of HCPs concerning layperson naloxone use in a single ED.

Methods: This study is an electronic, anonymous survey that included 24 multiple choice questions. It was emailed to all eligible ED HCPs at a tertiary-care teaching hospital. Questions were designed to assess many aspects of naloxone kit distribution including naloxone legislation, pre-formed perceptions of HCPs concerning layperson naloxone administration, and barriers to distribution such as lack of resources, time, and education. The Institutional Review Board approved the study.

Results: The survey was sent to 270 ED HCPs with 107 (40%) responses. The distribution of responses was nurses (n = 44, 41%), medical doctors (n = 32, 30%), EMT (n = 19, 18%), other (n = 6, 6%), midlevel providers (n = 4, 4%), and pharmacists (n = 2, 2%). Many HCPs (41%) responded as unsure if the ED already dispensed naloxone kits. The majority of HCPs (54%) stated they were unsure if naloxone was covered under third party prescribing with 59% of HCPs responding as unsure if they were protected by law from liabilities associated with layperson administration of naloxone. When asked about required patient education with naloxone kit distribution, 28% stated they were unsure if it was mandated by law. The majority of HCPs (54%) believed that patients should be required to present to the ED every time a naloxone kit was administered by a layperson. Many HCPs (64%) did not believe that naloxone would encourage drug abuse, and 63% of HCPs reported that naloxone kits would decrease the number of opioid deaths in the state. The top three barriers identified in providing patients with naloxone take home kits were absence of knowledge about naloxone laws, time required for education, and cost of the naloxone take home kit. The top three most needed resources identified for a naloxone take home kit distribution program to be successful were more health care professional education about naloxone, more patient education about naloxone, and more time for education in the ED.

Conclusions: The top three barriers identified by HCPs in this ED focus on further education and cost. The majority of HCPs had positive perceptions of layperson naloxone use. Making patient and provider education a priority is likely to increase the success of a naloxone distribution program in this ED.

KEYWORDS Naloxone; Barriers; Layperson

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197. Simple Ultrasound Guided Gallbladder Aspiration: An Emerging Alternative for Amatoxin Mushroom Poisoning when Silibinin is Unavailable

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Background: Intravenous (IV) Silibinin (SIL) combined with sustained aggressive IV hydration is currently undergoing evaluation in a prospective clinical trial in the USA and appears to be emerging as an extremely effective treatment for reversing the hepatotoxicity induced by amatoxin mushroom poisoning (AMP). However, SIL is not available in developing countries (where mortality rates of >50% are still commonplace) and therefore, a reliable alternative is desperately needed. In a 1973 publication, beagles with a surgical biliary fistula survived fatal doses of amatoxin and suffered far less severe hepatic injury than controls. An amatoxin poisoned patient (2006 publication) with severe coagulopathy undergoing biliary drainage via ERCP nasobiliary drain placement survived with >4 mg (16.67 mcg/ml) of combined alpha and beta amanitin recovered from the total 240 ml aspirate.

Methods: Six patients in rural India with AMP underwent open surgical cholecystostomy. Three with preexisting multi-system failure and coma died soon thereafter. Three with INRs >4 but preserved renal function survived. An American AMP patient survived following percutaneous cholecystostomy (PC) placement by IR. Aggregate bile samples from each of the Indians and sequential daily samples from the American were evaluated by HPLC for alpha-amanitin content.

Results: Alpha-amanitin (only) content in aggregate bile samples from the Indian patients ranged from 3.06 to 11.67 mcg/ml. 22.3 mcg/ml of alpha-amanitin was found in the Day 0 sample from the American with all subsequent samples testing negative.

Conclusions: Based on this preliminary data, drainage of the gallbladder, in combination with sustained aggressive IV hydration, appears to be a promising treatment alternative for patients with AMP in developing country settings where SIL is unavailable. Further enterohepatic circulation mediated expansion of the

evolving hepatic injury is prevented by the definitive removal of sequestered biliary amatoxin. Open surgical or percutaneous gallbladder drain placement subjects patients to the risk of bile peritonitis at removal and may actually be unnecessary as most of the amanitin appears to be removed during the procedure itself rather than over subsequent days. Simple ultrasound guided gallbladder aspiration can be performed at the bedside, is far less technically challenging than PC or ERCP, and can be repeated after 24-48 hours. The risks of infection, hemorrhage, and bile peritonitis are substantially reduced compared to a cholecystostomy drain which must be left in place for at least 14 days or more. A transhepatic approach is appropriate early in the clinical course before the development of a significant coagulopathy; otherwise, a transperitoneal approach is recommended. LC-MS is now the gold standard for quantitative measurement of amanitin content in urine and serum. Reliable extraction of amanitins from bile in anticipation of LC-MS analysis continues to be an elusive research target. Bile is an extremely challenging matrix for small peptide quantification due to the heavy salt concentration which interferes with MS ionization and chromatography. Confirmation of these promising HPLC findings by LC-MS would help to validate this alternative approach to the management of amatoxin poisoning treatment when SIL is unavailable.

KEYWORDS Amatoxin; Mushroom; Silibinin

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198. Impact of Levocarnitine on the Half-Life of Valproic Acid in Patients with Valproic-Acid Induced Hyperammonemia Secondary to Acute Ingestion

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Background: The use of levocarnitine as an antidote for valproic acid-induced hyperammonemia has been described in case series and case reports, however, no data exists comparing the pharmacokinetic impact of intravenous (IV) or oral levocarnitine on valproic acid (VPA) clearance versus supportive care. This investigation assessed the effectiveness of levocarnitine on enhancement of VPA elimination, as characterized by a decrease in half-life, for patients with VPA-induced hyperammonemia secondary to acute ingestions. The authors also characterized the use of levocarnitine and summarized baseline characteristics and outcomes for patients presenting with hyperammonemia secondary to acute VPA ingestions.
Methods: This was a non-interventional, retrospective cohort at two hospitals within one health system consisting of a tertiary referral center and a community hospital. Patients were identified through a query of the health system database and included if they were admitted between 10/15/2011 and 10/15/2015 with an acute ingestion of VPA, were ≥18 years of age, had at least two VPA levels within 24 hours, and had an ammonia level ≥35μmol/L. Patients were excluded if they had a urea cycle disorder, pre-existing liver disease, were pregnant, or received renal replacement therapy or activated charcoal secondary to the ingestion. Descriptive statistics were used to summarize the data.
Results: In total, 512 patients were screened against inclusion and exclusion criteria. Fourteen patients were included in the primary analysis. The most common reasons for exclusion included: patients continued to receive VPA between levels (n = 262) and absence of hyperammonemia (n = 127). The majority of patients were African American (57.1%) and male (78.6%) with a mean age of 33.3 years. Eleven patients presented to the tertiary care hospital and three patients presented to the community hospital. The most common indications for VPA were for treatment of bipolar disorder (n = 7, 50%) and treatment of epilepsy or another seizure disorder (n = 5, 36%). Eight patients received oral levocarnitine, one patient received IV levocarnitine, and five patients received no levocarnitine. The mean (SD) number of valproic acid levels per patient was 4.36 (1.55). The median half-life (IQR) was 15.65 (10.78 - 20.02) hours for the oral levocarnitine cohort and 11.87 (9.36 – 14.2) hours for the group that did not receive levocarnitine. The half-life for the sole patient who received IV levocarnitine was 8.43 hours. The median length of stay (IQR) was 3 (2 – 3) days for the oral levocarnitine group, 5 (5 – 8) days for the no levocarnitine group, and 2 days for the sole IV levocarnitine patient. The majority of patients were discharged to psychiatric facilities (n = 8, 57.1%), while the remainder of patients were discharged back to prison (n = 3, 21.4%) or to home (n = 3, 21.4%).
Conclusions: VPA elimination half-life was similar with and without oral levocarnitine and was shorter in one patient treated with IV levocarnitine based upon a small sample size. The dose and route of levocarnitine in the setting of VPA-induced hyperammonemia needs further investigation before a standard regimen can be established.

KEYWORDS Valproic acid; Overdose; Levocarnitine

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199. Endoscopic Removal of a Radiodense Pharmacobezoar Following Ingestion of Diphenhydramine

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Background: A bezoar is an aggregate or concretion of undigested material within the gastrointestinal tract and can be classified based upon the origin of the material. Bezoars from medications, or pharmacobezoars, may occur following large ingestions but are often difficult to diagnose if clinical suspicion is low. Some medications are recognized as radiodense and plain radiographs of the abdomen are helpful for diagnosis. We report a case of intentional diphenhydramine (DPH) ingestion with no clinical improvement more than 36 hours after symptom onset.
Case Report: A 13-year-old 45 kg female was initially reported to have ingested 4-6 Rexall brand DPH tablets (25 mg) about 7 hours prior to admission. The patient was last seen normal by the guardian about 30 minutes prior to onset of drowsiness. Suspecting an ingestion she was transported to the emergency department where she was found to be actively hallucinating and had 2 tonic-clonic seizures with intermittent severe myoclonic jerking. The second seizure lasted approximately 20 seconds and was successfully aborted with one dose of lorazepam. A single dose of 0.9 mg of physostigmine was administered with transient slight improvement in clinical status. However, at 6 hours post ingestion, she continued to have hallucinations and myoclonic jerking, prompting admission to the pediatric intensive care unit (ICU). She was observed in the ICU with no clinical improvement for 38 hours. At this time, relatives updated the history to reflect that 100 DPH tablets were missing. Based on these concerns, an abdominal radiograph was obtained that showed a clear radiodensity over the medial half of the stomach. Gastroenterology was consulted and a bedside endoscopy was performed. A tennis

ball sized round mass of "glop" with pink colored fragments resembling the DPH tablets was found in the antrum of the stomach. The mass was removed in small pieces using an expandable net. Within hours of the removal procedure, the patient became increasingly responsive. She continued to improve clinically and was transferred for inpatient psychiatric care.
Case Discussion: After a thorough review of the medical literature, there is nothing to suggest that DPH is radiodense. Also, there are no published reports of DPH pharmacobezoar formation.
Conclusions: Failure to improve from an acute ingestion should prompt consideration of a bezoar. Despite no prior evidence of radiodensity, a DPH bezoar was identified in this case on plain radiograph and provided sufficient evidence to support endoscopic evaluation and removal.

KEYWORDS Pharmacobezoar; diphenhydramine; bezoar

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200. Ivermectin and Albendazole Toxicity Treated with Intravenous Lipid Emulsion

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Background: Little data is available to guide the management of ivermectin and albendazole overdose in humans. We report a case of ivermectin and albendazole toxicity following an intentional overdose treated with intravenous lipid emulsion (ILE) therapy.
Case Report: A 46 year old male with a past medical history of hypertension presented after being found drinking from a bottle of veterinary grade albendazole at his home. He was also noted to have a bottle of veterinary grade ivermectin. The patient was markedly delirious and unable to contribute to the history. Per family, the patient was in his usual state of health until approximately 6 weeks prior to presentation when he reportedly began treating himself for scabies with ivermectin, albendazole. Exam revealed a cachectic male with diffuse alopecia. He was awake and alert but had a tangential thought process and was not orientated to person, place, time, or situation. He reported no hallucinations. Pupils were 4 mm, equal and sluggishly reactive. Reflexes were brisk diffusely without myoclonus. He had a moderate intention tremor in all extremities. Vital signs were within normal limits; notable lab results showed: white blood cell count 17.8k/mcL, hemoglobin 7.3g/dL, platelets 741 k/mcL, bicarbonate 20 mmol/L, creatinine 1.3 mg/dL, BUN 41 mg/dL, anion gap 18, creatine kinase 2769 U/L, and INR 2.2. B12, folate, and iron levels were within normal limits. Computed tomography of the head was negative.
The patient received 1.5 ml/kg of 20% ILE over the course of 30 minutes followed by an additional dose of 1.5 ml/kg of 20% ILE 18 hours later. The patient's mental status was markedly improved the next morning and he admitted to taking albendazole and ivermectin orally and applying a topical depilatory agent in an attempt to treat scabies.
Case Discussion: There is limited human overdose data for either ivermectin or albendazole. Side effects of albendazole include elevated liver enzymes, nausea, vomiting, abdominal pain, and headaches. Ivermectin toxicity is reported to cause central nervous system depression, ataxia, tremor and visual disturbance, with prolonged prothrombin times and creatine kinase elevation also reported. Multiple cases in the veterinary literature document successful use of lipid emulsion to treat ivermectin toxicity in animals, with pharmacokinetic data demonstrating enhanced elimination. However, ILE has not been previously reported for human ivermectin toxicity. In this case lipid emulsion was given two times over the course of 18 hours and was temporally associated with marked improvement in the patient's symptoms.
Conclusions: While supportive care appears to be the mainstay in therapy of albendazole and ivermectin, lipid emulsion is a viable option for the treatment of ivermectin toxicity in humans.

KEYWORDS Ivermectin; Albendazole; lipid emulsion

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201. Octreotide Recommendation Patterns in Unintentional Pediatric Sulfonylurea Ingestions at a Regional Poison Center

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Background: Sulfonylureas are known to cause delayed and recurrent hypoglycemia in children following accidental exposure. Sulfonylurea-induced hypoglycemia treatment includes oral glucose intake and parenteral dextrose. Parenteral octreotide is used to maintain euglycemia; however, there are no commonly accepted guidelines on the use of octreotide in this population. The primary objective of this study is to determine the conditions under which octreotide has been recommended.
Methods: All sulfonylurea ingestions reported to a regional poison center (RPC) over a 15 year period were reviewed. Cases of children aged $\leq$12 years with a history of acute, unintentional single ingestions of a sulfonylurea were included. Hypoglycemia was defined as a blood glucose $\leq$60 mg/dL in children 3 months to 12 years, and $\leq$50 mg/dL at less than 3 months.
Results: Out of 558 cases identified, 452 cases met inclusion criteria. Age ranged from 1 month to 11 years, with a median of 2 years. Most ingestions (85.6%) occurred in children 12 months to 5 years. Males accounted for 50.9%. A majority of patients (pt) were managed in a HCF (77.9%). Of the pt with known outcomes, 74.9% had no effect, 1.7% had minor effects, 22.3% had moderate effects (at least one episode of hypoglycemia), and 1.2% had major effects. Octreotide was given to 15 pt who developed hypoglycemia, all treated with dextrose initially. The average initial blood glucose in pt who received octreotide was 62.3 mg/dL. Average blood glucose prior to octreotide was 73.5 mg/dL, vs average blood glucose of 109.2 mg/dL following octreotide. Hypoglycemic episodes prior to octreotide occurred a mean of once per pt (range, 1 to 7). Five hypoglycemic episodes occurred following octreotide (mean 0.3 per pt); none of these pt developed worsening symptoms after octreotide. Seventy-nine pt had at least 1 episode of hypoglycemia; 16 were observed or managed with oral intake only. Twenty-seven of 63 patients treated with intravenous dextrose had at least 1 episode of rebound hypoglycemia; 4 pts developed symptoms secondary to rebound hypoglycemia.
Conclusions: Patients were initially managed with dextrose only. When hypoglycemia occurred after dextrose, most pt received additional dextrose rather than octreotide, though octreotide was sometimes selected even after only one episode of hypoglycemia. Most pt with sulfonylurea-induced hypoglycemia can be safely managed with dextrose only, even in the presence of rebound hypoglycemia.

KEYWORDS Octreotide; Sulfonylureas; Hypoglycemia

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202. Utility Of Whole Bowel Irrigation In Bupropion Overdoses In Non-Tachycardic Adults

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Objectives: Bupropion overdoses are known to induce delayed onset seizures. Whole bowel irrigation (WBI) is often recommended to minimize delayed complications. In previous studies, tachycardia has been shown to be a predictor of seizure activity. However, data does not exist that WBI prevents delayed seizures in non-tachycardic adults at presentation. Our hypothesis was that whole bowel irrigation would not be associated with a decreased rate of delayed seizures in the non-tachycardic patient with an acute bupropion ingestion.
Methods: This is a retrospective cohort of regional poison center data. All adult patients with bupropion ingestion from January 1, 2002 to December 31, 2012 were screened. Patients were excluded if they were under 18 years of age, if there was a co-ingestion, if there was a seizure prior to arrival, and if they were tachycardic at presentation. Tachycardia was used as an objective measure of drug toxicity. The primary outcome was seizure activity. Secondary outcome was any complications of WBI therapy noted in poison center chart. The groups were separated into those who received WBI, and those who did not. Fisher's exact test was used to determine if there is significant difference between groups.
Results: Out of the 2,270 screened, 518 met inclusion criteria. 62/518 (11.9%) had a seizure. Of the patients who received WBI, 9/33 (27.3%) had a seizure, 24/33 (72.7%) did not. Of the patients who did not receive WBI, 53/485 (10.9%) had a seizure, 432/485 (89.1%) did not. The patients in the WBI group were more likely to have a seizure, OR 3.04 (1.18-7.24, 95%CI). There were no reported complications for patients who received WBI.
Conclusions: There was no clear benefit found for WBI in the non-tachycardic single bupropion ingestion in this cohort of patients. WBI was safe in this study. A future randomized control trial would further elucidate whether or not this invasive intervention has therapeutic benefit in non-tachycardic patients.

KEYWORDS Bupropion; Whole Bowel Irrigation; Seizures

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203. A 10-year retrospective review of octreotide use reported to a poison center

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Background: Octreotide is a long-acting somatostatin analogue which inhibits insulin release from the pancreas. Medical toxicologists recommend octreotide to prevent recurrent hypoglycemia from secretagogues such as sulfonylureas.
Methods: We conducted a 10-year retrospective review of data from one poison center between January 1, 2005 to December 31, 2015 for calls where antidotal therapy with octreotide was recommended and/or performed. Abstracted data included: age, exposure, context, clinical effects, therapies, and outcomes.
Results: A total of 70 cases met inclusion criteria. Group I was comprised of 46 cases involving a sulfonylurea. 14 were pediatric cases (< 18 years of age). There were 13 suicidal ingestions, 4 of which were pediatric cases. 42 patients in Group I developed hypoglycemia. 17 of the patients in the group that developed hypoglycemia also used another diabetic medication. 14 also ingested metformin; 3 injected insulin; and 1 ingested rosiglitazone. One patient used a sulfonylurea, metformin, and insulin. 37 of the hypoglycemic patients received octreotide. Efficacy, defined as clearly documented cessation of hypoglycemia following therapy, was noted in 20 (54.1%) of the patients who received octreotide. However, efficacy was not documented in 9 (24.3%) cases. Group II was compromised of 14 cases involving parenteral insulin. 10 of these cases were suicide attempts. In this group, 12 patients developed hypoglycemia, and 10 (83.3%) of those patients received octreotide. Octreotide was efficacious in preventing recurrent hypoglycemia in 3 (30%) cases. It was deemed ineffective in 4 (40%) cases. Efficacy was not documented in 3 (30%) cases. Group III was comprised of 10 cases. These involved: acetaminophen overdose (2), metformin ingestion (1), Amanita phalloides poisoning (2), isopropanol and ethanol ingestion (1), or an unknown ingestion (4). Reasons for octreotide administration in this group included hypoglycemia (4), gastrointestinal bleeding (2), Amanita poisoning (2), or an unknown reason (2). Efficacy was not recorded in any of the hypoglycemia cases.
Conclusions: In medical toxicology, antidotal therapy with octreotide is traditionally used for recurrent hypoglycemia in secretagogue-induced hypoglycemia. However, review of 10 years of data from a single poison center reveals that recurrent hypoglycemia is sometimes treated with octreotide regardless of etiology. While the efficacy of preventing recurrent hypoglycemia appears to be lower in non-secretagogue induced hypoglycemia, it may play a role in decreasing the frequency of labile blood sugars. Fortunately, the side effect profile of octreotide is relatively benign, so its use in this setting favors benefit over harm.

KEYWORDS Octreotide; Non-sulfonylurea; hypoglycemia

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204. Octreotide Administration for Hypoglycemia in a Patient on Nateglinide

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Background: Meglitinides are a class of oral hypoglycemic alternatives to sulfonylureas used due to their decreased incidence of hypoglycemia. Meglitinides such as nateglinide are structurally distinct from sulfonylureas, but they do act on the beta islet potassium ATPase in a similar, though more rapidly reversible, fashion. Octreotide is a safe and effective reversal agent for sulfonylurea related hypoglycemia through its inhibition of beta islet calcium influx. Nateglinide has been associated with increased hypoglycemic events in chronic renal failure due to decreased clearance of its active metabolite and insulin. We present a patient on nateglinide with acute kidney injury (AKI) and recurrent hypoglycemia that resolved after octreotide administration.
Case Report: A 59 year old man with a past medical history of diabetes, hypertension, and bipolar disorder whose medications

included nateglinide, sitagliptin, metformin, lithium, lisinopril, and tramadol presented with several weeks of diarrhea, nausea, fatigue, and tremors. Labs revealed AKI (Cr 1-3.5 mg/dL), hypoglycemia (31 mg/dL) and elevated lithium (3.0 mmol/L), and he was transferred to the emergency department. Vital signs were heart rate 32 bpm and blood pressure 147/65 mm/Hg. His ECG revealed sinus bradycardia with sinus pauses and 1st degree AV block. Despite eating prior to arrival the patient's blood glucose was 42 mg/dL. Transient responses to oral and IV glucose administration occurred with recurrent hypoglycemia x 2 before discussion with medical toxicology consultants and administration of 50 mcg IV octreotide. No subsequent hypoglycemic events occurred overnight or during hospitalization. The patient had nothing to eat overnight and was only administered intravenous saline. The lithium concentration improved slightly (3 - 2.7 mmol/L) with IV hydration. The patient received hemodialysis twice with improvement in bradycardia one day after his lithium level normalized. He was transferred to psychiatry and had no subsequent hypoglycemia or significant bradycardia.

Discussion: We attributed this patient's hypoglycemia to his daily use of nateglinide due to the hypoglycemia's association with renal failure, refractory nature, resolution with octreotide, and lack of other cause. The patient's other medications may have had a minor contribution to his hypoglycemia. Given its safety and mechanism of action, it is reasonable to administer octreotide in cases of refractory hypoglycemia when a meglitinide may have contributed.

Conclusions: Patients on nateglinide who develop AKI may be at increased risk of hypoglycemia. Octreotide was a safe and effective therapy for refractory hypoglycemia in this patient.

KEYWORDS Nateglinide; Hypoglycemia; Octreotide

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205. A Liver's Rise and Fall with Changes in NAC Dosing

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Background: The optimal dose of n-acetylcysteine (NAC) therapy for patients poisoned by acetaminophen (APAP) remains elusive. There has been a movement among medical toxicologists toward personalized therapy. Beyond the standard "Prescott Protocol," evidence supporting intravenous (IV) regimens has been sparse. We present a case of acute APAP poisoning in which the pattern of liver transaminase and INR rise and fall temporally reflected the dosing of administered antidote.

Case Report: A 34-year-old male was found unresponsive and acidotic. Upon evaluation at the initial ED, his serum APAP was 680 mcg/ml (unknown time of ingestion), AST 387 U/L, ALT 343 U/L. He was administered a 150 mg/kg dose of IV Acetadote™ (See figure). Beginning 2.5 hrs later, he received 3 doses of 70 mg/kg of oral NAC, 4 hours apart. During this time his acidosis improved while his AST/ALT rose and peaked at 9,376/5,887 U/L, as did his INR at 2.8. All parameters declined quickly and, apparently on the road to recovery, he was placed on an infusion of the same antidote at 6.25 mg/kg/hr beginning fifteen hours after treatment was first initiated. Serum APAP concentration remained elevated at 166 mcg/ml six hours after he was switched to the low dose of IV NAC. After 12 hours at that dose, his AST/ALT/INR again climbed precipitously. Six hours after the second rise began, he was switched to a higher infusion dose (12.5 mg/kg/hr) of NAC. Ten hours later, his markers again crested and progressively improved. His APAP level normalized without any secondary peaks. He made a complete recovery and has not demonstrated evidence of liver injury on follow-up.

Case Discussion: This case highlights a potentially significant relationship between the dose of NAC and the pattern of transaminase and INR elevation and decline. This case implies a distinct relationship between the "standard" dosing of IV NAC and a precipitous rise in serum markers of hepatic injury. The reason for this relationship is likely related to stoichiometry, with the "Prescott Protocol" likely not providing sufficient antidote to reduce the amount of toxic metabolite formed after such a large overdose, particularly after delayed presentation.

KEYWORDS n-acetylcysteine; acetaminophen; transaminase

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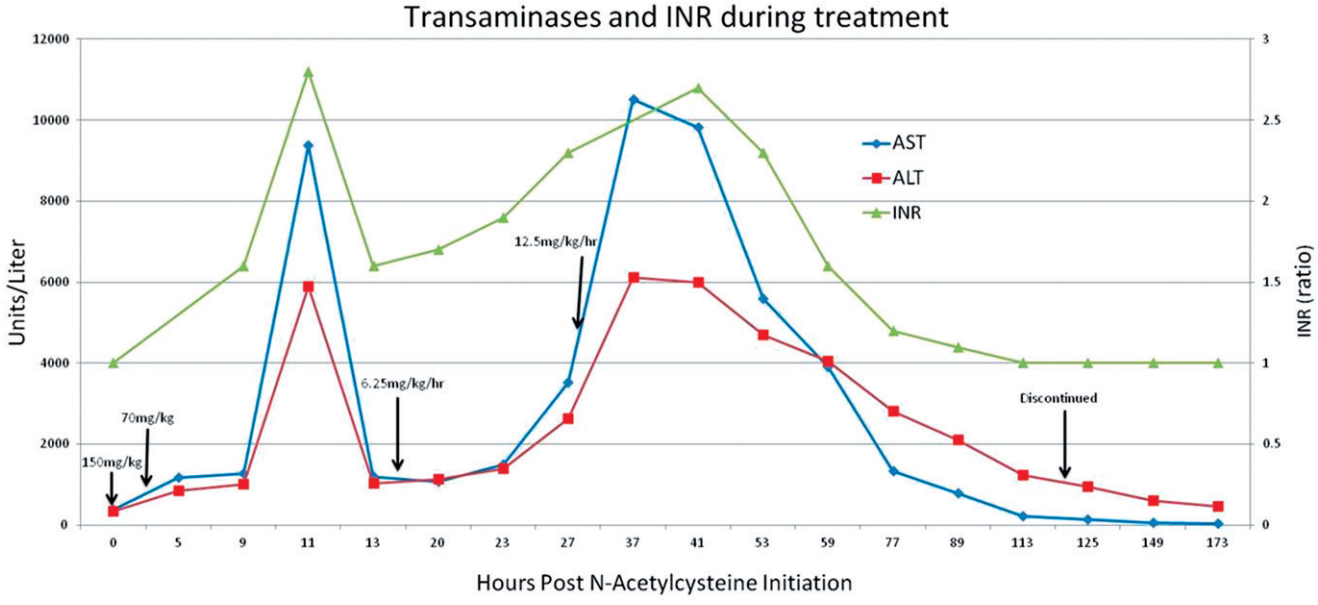

206. Gastric Decontamination in Pediatric Bupropion Ingestions

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Background: Bupropion is a commonly used antidepressant and a frequent culprit in toxin-induced seizures in pediatric patients. Gastric decontamination, including whole bowel irrigation (WBI), activated charcoal (AC), and/or gastric lavage (GL), is often performed in an attempt to prevent or ameliorate drug toxicity. The objective of this study was to determine the utility of gastric decontamination in preventing in-hospital seizures after pediatric bupropion exposure.
Methods: This is a retrospective case series of calls to a regional Poison Control system for pediatric (<18 years old) bupropion ingestions from 2002-2012. Cases of polysubstance ingestion were excluded. Logistic regression was performed using variables for patient age, gender, reported amount ingested, presence of tachycardia on ED arrival, reported seizures, performance of whole bowel irrigation, administration of activated charcoal, and performance of gastric lavage.
Results: 523 pediatric bupropion ingestions were identified. 299 patients were omitted for incomplete data. Administration of AC showed a trend towards decreased in-hospital seizures (odds ratio (OR) 0.48, 95%CI 0.23-1.03, $p = 0.058$). The use of WBI and GL had no association with seizure recurrence after administration (OR 1.5, 95%CI 0.55-4.09, $p = 0.42$; OR 0.71, 95%CI 0.08-6.32, $p = 0.78$).
Conclusions: While not statistically significant, this study suggests that activated charcoal may decrease the odds of in-hospital seizures in cases of pediatric bupropion exposure. Its use should be considered if safe administration can be ensured. Whole bowel irrigation and gastric lavage, however, do not seem to be beneficial for the treatment of pediatric bupropion exposure. Further randomized trials are warranted to determine the effects of gastric decontamination on bupropion toxicity.

KEYWORDS Bupropion; Gastric Decontamination; Antidepressant

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207. pH Manipulation of Activated Charcoal

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Background: Activated charcoal's (AC) role in acute poisonings has decreased greatly over the past decades, however, there are some acute poisonings, which may benefit from activated charcoal e.g., large aspirin ingestions. pH manipulation of blood and urine have been utilized to increase drug ionization to prevent transmembrane movement e.g., urine alkalinization. Similarly, physiologic changes in gut pH can affect ionization and decrease passive diffusion of xenobiotics and systemic absorption. We performed an in vitro experiment in an effort to manipulate the pH of activated charcoal with over-the-counter liquid antacids to assess which increases activated charcoal pH more.
Methods: Four separate EZ Char® (activated charcoal) 25 gram containers were used in each arm. Control was AC with distilled deionized water 120mL (arm A), aluminum hydroxide/magnesium hydroxide 120mL (arm B), calcium carbonate 120mL (arm C) and aluminum hydroxide/magnesium hydroxide 60mL with calcium carbonate 60mL (total 120mL) (arm D). Each container was agitated to make uniform suspension. A pH meter was used to measure each of the suspensions with re-calibration after each measurement. Each arm was performed twice. In addition, water and each antacid solution pH was measured separately without activated charcoal.
Results: Arm A (control) pH 7.15 ± 0.07, arm B (aluminum hydroxide/magnesium hydroxide) pH 7.9, arm C (calcium carbonate/magnesium hydroxide) pH 7.7 ± 0.14, arm D (combination aluminum hydroxide/magnesium hydroxide with calcium carbonate/magnesium hydroxide) pH 7.65 ± 0.07. Baseline pH of water 7, aluminum hydroxide/magnesium hydroxide pH 8 and calcium carbonate/magnesium hydroxide pH 8. Conclusions: We demonstrated in this in vitro experiment the addition of OTC antacids can increase the pH of activated charcoal suspensions over water alone. This was greatest with aluminum hydroxide/magnesium hydroxide antacid alone. As pH is base 10 logarithmic, small increases in pH can change the ionization of acidic xenobiotics. It is unclear if this would translate to a clinically significant decrease in absorption of acidic xenobiotics. As many antacids are flavored there is also a possibility the addition of these products to AC may also improve palatability and patient acceptance. In vivo volunteer studies would be needed before this technique is recommended to acutely poisoned patients.

KEYWORDS Activated charcoal; Gastrointestinal decontamination; Alkalinization

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208. The use of levetiractam in drug induced seizures

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Background/Objectives: The use of levetiracetam (LEV) in the management of drug-induced seizures has not been systematically investigated. Repetitive and continuous seizures that do not respond to benzodiazepines require second line therapy. However, phenobarbital and propofol have not been validated in randomized studies, and phenytoin is contraindicated. Levetiracetam has a unique receptor binding site, rapid absorption, no known cardiac effects at therapeutic doses, and is theoretically a good candidate for use in drug-induced seizures. We evaluate the safety of LEV and its association with seizure cessation in this retrospective chart review of patients who received LEV as a control agent in drug-induced seizures.
Methods: The medical records of patients presenting to an urban, level 1 trauma center between Jan 2010 and May 2015 were identified by ICD-9 codes based on the following: 1) a poisoning diagnosis, 2) a seizure diagnosis, and 3) an order for LEV. Patients with a drug-induced seizure based on history, electroencephalogram results, blood alcohol concentrations, urine drug screens, and adequate documentation were included. Patients with alcohol withdrawal, anoxic brain injury, subtherapeutic concentrations of other antiepileptics, hypoglycemia, and pseudoseizures were excluded. Therapeutic potential was determined by cessation of active seizures or the prevention of seizure recurrence. Safety was determined by the presence or absence of adverse drug effects (ADE) attributed to the administration of LEV.

Results: Thirty-four patients met inclusion and exclusion criteria. Half of the study cohort (17) presented with generalized tonic-clonic seizures (TCS); half (17) presented in generalized convulsive status epilepticus (GCSE). Six patients in GCSE were administered LEV during their seizures; 2 also received fosphenytoin. One improved immediately following LEV administration, and the remaining 5 were documented to have near-immediate seizure control. Twelve GCSE patients (71%) remained seizure free after LEV therapy. Patients with TCS (17) were all administered LEV after presenting seizure(s) were controlled. Sixteen (94%) were seizure free during their hospital course. There were no reported adverse drug effects. In total, 27 of 34 patients (79%) had a return to baseline neurological and physical health. Six had long term sequelae; none of which are known LEV side-effects. Forty-five toxic substances and 23 known seizurogenic agents (51%) were identified. The median length of stay was 3.7 days (0.4 – 96), and the median duration of in-hospital LEV therapy was 1.6 days (0 - 49).
Conclusions: In our cohort, LEV was associated with an 82% rate of control of drug-induced seizures—a success rate that approximates LEV's efficacy in non-drug-induced seizures. There were no ADEs attributed to LEV. LEV appears to be associated with the control of drug-induced seizures without ADEs in this retrospective study. A prospective study is needed to confirm these results.

KEYWORDS Drug-induced seizure; levetiracetam; status epilepticus

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209. Pediatric single methotrexate pill ingestion: no assessment = no risk?

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Background: Methotrexate is a commonly prescribed folate analogue anti-metabolite used to treat malignancy, rheumatologic, and dermatologic diseases. Pediatric oral exposures have rarely been reported to produce acute toxicity. We present the case of a two-year-old girl with a single pill, low-dose methotrexate ingestion resulting in a concentration above the threshold to cause toxicity.
Case Report: A 12.1kg girl presented to the emergency department approximately one hour after ingestion of her grandfather's methotrexate 2.5mg and levothyroxine 88μg. Initial VS were: HR, 118/min; BP, 96/58mmHg; RR, 28/min; room air oximetry, 98%. She was asymptomatic with a normal physical exam. Activated charcoal 1g/kg was administered on arrival. Leucovorin rescue therapy was initiated ten hours after ingestion. Renal function was normal (BUN, 5mg/dL; Cr, 0.2mg/dL). A 10-hour methotrexate concentration was 0.180micromol/L and below the limits of detection (<0.02micromol/L) at 23 hours. The patient received an initial dose of 10 mg leucovorin IV based on quadruple the equimolar dosing of presumed methotrexate ingestion, followed by repeated six-hourly dosing of 100mg/m2 until the methotrexate level was undetectable, for a total leucovorin dose of 110mg.
Case Discussion: As methotrexate indications expand, so does the potential for unintentional pediatric ingestion. In patients receiving chemotherapy, 24-hour methotrexate concentrations >10 micromol/L are considered high risk for toxicity; 0.01 micromol/L is the threshold for potential toxicity in patients receiving methotrexate for other indications. Our patient's post-distribution, 10-hour concentration exceeded the threshold for inhibition of DNA synthesis (0.01 micromol/L). In unintentional pediatric methotrexate ingestion, overt clinical signs of toxicity will rarely be present initially, if ever. However, impaired DNA replication and RNA synthesis risk future carcinogenesis or damaged gametogenesis; thus, empiric management with leucovorin rescue should be initiated even for low dose exposures. Obtaining a methotrexate concentration can assist in establishing ingestion and guide duration of leucovorin therapy.
Conclusions: Single pill pediatric methotrexate oral ingestion can lead to methotrexate concentrations capable of inhibiting DNA replication. Leucovorin therapy should be considered.

KEYWORDS Methotrexate; pediatric ingestion; leucovorin

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210. Uridine triacetate is a life-saving antidote to capecitabine toxicity

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Background: Uridine triacetate was approved by FDA in 2015 for adult and pediatric patients who receive an overdose of 5-fluorouracil or capecitabine or who exhibit early-onset, severe or life-threatening toxicities. Early-onset toxicities from capecitabine or 5-FU treatment are due to various metabolic causes, including DPD deficiency, and can be fatal.
Uridine triacetate is an oral prodrug of uridine, a direct antagonist of 5-FU, that dilutes and competes with toxic 5-FU metabolites, particularly FUTP incorporation into RNA, reducing morbidity and mortality due to 5-FU and capecitabine.
Methods: Uridine triacetate has been studied in 2 trials (173 patients) to date, including 13 who received an overdose of capecitabine or developed early-onset, severe toxicities. The Sponsor (Wellstat Therapeutics) was contacted upon recognition of an overdose or early-onset toxicity and evaluated patient eligibility. Adults (n = 6 overdose and 4 early-onset) were to receive 10 g of uridine triacetate orally every 6 hr for 20 doses. Pediatric patients (n = 3) were to receive 6.2 g/m2 orally every 6 hr for 20 doses. Uridine triacetate was to start within 96 hr following the end of capecitabine. The primary endpoint was survival at 30 days following uridine triacetate. All patients were monitored for safety.
Results: All 6 adult capecitabine suicidal overdose cases (up to 28,000 mg at once) and all 3 pediatric patients who accidentally took capecitabine recovered fully within 30 days post-treatment with uridine triacetate.
Both patients who presented with multiple severe post-capecitabine toxicities but initiated treatment with uridine triacetate >96 hr died. In contrast, both patients who presented with similar multiple severe early-onset toxicities but who started uridine triacetate within the recommended 96 hr post capecitabine fully recovered.
Only one AE (vomiting) was attributed to uridine triacetate.
Conclusions: Uridine triacetate was a safe and effective life-saving treatment for patients following potentially lethal capecitabine overexposure or early-onset toxicities when administered within 96 hr post capecitabine dosing.

KEYWORDS capecitabine; uridine triacetate; 5-fluorouracil

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211. Therapeutic Medication Errors Administered at Schools

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Background: Medication errors occurring at schools are poorly characterized. In 2000, a survey of school nurses revealed almost 50% of nurses had reported errors in administration of medications. The purpose of the study is to describe medication errors reported to one state's poison centers that occur during administration at school.
Methods: This was a retrospective chart review of children who had medication errors as reported to Poison Centers from one state from 1/1/06 to 12/31/2013. Inclusion criteria included any patient less than 19 years old with report of an exposure coded "therapeutic error", where the site was coded as "school" and where school personnel administered the medication error. Cases were double abstracted and any discrepancies were resolved by a 3rd reviewer. Data collected included age, gender, intended medication, administered medication, disposition, scenario, and outcome. Case narratives were reviewed to verify the circumstances of each exposure.
Results: 228 cases met inclusion criteria. The median age was 8 years, 71% were males. 89% of exposures occurred at a school and 11% at a daycare facility. 202 cases were not managed at a health care facility. 22 cases were treated and released from an emergency department (ED). 1 case was admitted to a critical care unit, 1 case was admitted to a non-critical care unit, and 2 cases were lost to follow up. The scenarios included repeated dose (95), administration of someone else's medication (54), other medication error (42), doses given too close together (18), received the wrong medication (their own) (11) and incorrect route (8). There were no reported major outcome cases. 4 cases reported a moderate effect (substances included metoclopramide, guanfacine, methylphenidate and lacosamide). 21 cases reported a minor effect, 198 had no effect or no significant effects anticipated. 2 were unable to be followed and 3 reported an unrelated effect. The most common class of medications involved was amphetamines (119). Errors in administering vaccines were identified in 22 cases. Clonidine or guanfacine were implicated in 17 cases. Antipsycotics were identified in 15 cases and anticonvulsants in 9 cases. The remaining cases involved a variety of substances including antibiotics, antidepressants, OTC pain relievers and cold medications.
Conclusions: Amphetamines account for the majority of medication administration errors at the school level. Approximately 11% of the errors resulted in an ED evaluation.

KEYWORDS Therapeutic error; Medication error; School

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212. Significant Gastrointestinal Injury Following Ingestions of Topical Keratinolytics

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Background: Keratinolytics are compounds that dissolve keratin in the skin and are used as topical corn, callus, and wart removers. Calls to poison control centers (PCC) regarding exposures to these products are common. From 2010 to 2014, the National Poison Data System (NPDS) reported 6,855 human exposures to keratinolytics. 2,753 cases were followed to a known outcome. Of these, 195 cases (7%) had moderate effects, while 8 cases (0.3%) had major effects with no deaths. Few references describe gastrointestinal (GI) burns following pediatric ingestions of salicylic acid, the most common ingredient. We present a case series resulting in significant GI injury following keratinolytic exposures containing other or unknown ingredients.
Case Reports: Case 1, a 22 month old male presented to the emergency department (ED) after dermal and oral exposure to unlabeled wart remover purchased at a flea market. The child developed burns to back/nape of neck, and erythema on face and lips. An esophagogastroduodenoscopy (EGD) revealed multiple 2nd degree esophageal burns. The product had a pH of 5.5, but chemical analysis for ingredient identification was not obtained. Case 2, an 18 month old female presented to the ED after ingesting a wart remover from Mexico called Ca-llosol. An internet search showed an unknown concentration of ethanoic acid (acetic acid) with an unknown pH. An EGD revealed circumferential exudate and superficial ulcers consistent with grade 2B esophageal burns. Case 3, a 2 year old female presented to the ED after ingesting Gena Callus Off the night before. The product contains potassium hydroxide (KOH), glycerin, and acrylates. She had vomited several times that evening. In the morning, she was febrile with dysphagia. An EGD revealed the entire esophagus to be indurated and erythematous with spots of ulcerations. Case 4, a 15 month old female presented to the ED following possible ingestion of callus remover. The label listed KOH, propylene glycol, glycerin, and acrylate. The pH was 14. Oral examination showed no burns or swelling. EGD was advised, however, the family left against medical advice.
Case Discussion: Most over-the-counter (OTC) keratinolytics are made with 17% salicylic acid with a pH of 3-5 and are not sold in child resistant packaging. Although 93% of exposure calls recorded in NPDS result in no or minor effects, the potential for greater injury exists, especially in products containing alkali or ethanoic acid. Products with unknown ingredients should be managed with extreme caution.
Conclusions: Unintentional pediatric exposures to keratinolytics can result in significant caustic injury to the eyes, skin, and GI tract. Vigilance by PCC staff in identifying ingredients and triaging these exposure calls is vital.

KEYWORDS corn/callus/wart remover; caustic ingestion; esophageal burns

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213. Pediatric Buprenorphine Exposure: Poison Center Documented Dose, Onset, and Duration

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Background: Buprenorphine (BUP) is a partial μ-opioid agonist typically used for the treatment of opioid addiction or chronic pain. It is not currently recommended for use in pediatric patients. Questions surrounding pediatric BUP ingestion often involve required length of observation and symptom duration in the event of clinical manifestations. In answer, this poison center (PC) documentation study reviews the symptom timeline of BUP exposures.
Methods: Human pediatric (≤6 years old) BUP ingestion from 2008-2015 were sorted into 2 groups: PC observed ≥8 hours

(Group I) or PC observed <8 hours (Group II). Group I and Group II were assessed by reviewers A & B and B & C respectively for the following: age, management site, medical outcome (Table 1), BUP dose, treatment with naloxone, oxygen, or assisted ventilation (Table 2). Symptom onset time and duration was determined with reviewers selecting and modifying as needed the appropriate documentation time-stamp. Where reviewers disagreed, consensus was attempted. The remaining disparity for onset and duration was plotted with linear regression and described with correlation coefficients (r). Finally, all values were averaged (Table 3).

Results: 225 BUP exposures were reported to the PC during the study period with 68 excluded, leaving 157 for review. Exclusion was primarily for lack of follow-up to confirm presence of any symptoms. Group I was comprised of 107 patients and Group II was comprised of 50 patients. The results are shown in Table 1 (Demographics), Table 2 (BUP Dose and Treatment), and Table 3 (Symptom Onset and Duration). Symptoms after pediatric BUP ingestion were likely rapid, on average 127 minutes and 83 minutes for Group I & II respectively (overall range: 1-480 minutes). Naloxone use was frequent (32% Group I, 12% Group II) but did not appear to shorten clinical effect duration. Reviewer assessment of BUP dose was often conflicted and did not predict symptom severity. Study limitations included difficulty in distinguishing BUP clinical effects from normal behaviors, delays in reporting, inability to confirm BUP ingestion, infrequent PC sampling (call-backs), and reporting bias.

Conclusions: This evidence supports an observation period of at least 8 hours after BUP ingestion for children ≤6 years old. A healthcare facility is probably the best management site due to persistent and sometimes severe symptoms. Further efforts are recommended in primary prevention of these exposures.

Table 1.

Demographics	Group 1	Group II
N	107	50
Average age in years (SD)	21 (0.9)	2 (0.9)
Management site:		
Home	4	12
Healthcare facility (HCF)	103	34
Unknown/Against medical advice	0	4
No effect	19 (18 96)	3 (6%)
Mnor	52 (49 96)	23 (46%)
Moderate	33 (3196)	5 (10%)
Major	1 (0.9%)	0 (0%)
Not foil owed	1 (0.9%)	19 (38%)
Unrelated	1 (0.9%)	0 (0%)
Average follow-up hours (SD)	24 (19)	5 (4)

Table 2.

BUP dose and treatment	Group 1	Group II
BUP Dose evaluable (%)	78 (73%)	27 (54%)
Evaluable average BUP dose (SD)	4.8 mg (3.1)	3.8 mg (3.2)
Any naloxone used	34 (32%)	6 (12%)
Multiple dose naloxone	19 (18%)	1 (2%)
Naloxone infusion	8 (7. 5%)	2 (4%)
Oxygen	11 (10.3%)	2 (4%)
Ventilator	1 (0.93%)	0 (0%)

Table 3.

Clinical Effect Onset and Duration (combined reviewer results)	Group 1* (Reviewer A& B)	Group II* (Reviewer B & C)
Onset (minutes) post-exposure r:	0.96	0.99
Average (SD):	127 (101)	83 (60)
Median:	110	60
90th percentile:	270	180
Duration (hours) post-exposure r:	0.96	X
Average (SD):	18 (16)	X
Median:	14	X
90th percentile:	31	X
Naloxone versus symptom duration average hours (SD)	21 (21)	X
Naloxone:		
No Naloxone:	17 (14)	X
Excluded cases	*4 cases onset time uncertainty[1] >8 hours, 20 asymptomatic	* lease onset time uncertainty >8 hours X = not evaluable

KEYWORDS Buprenorphine; Pediatric; Timeline

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214. A Sinking Feeling... Lead Toxicity in a 9 year old Child

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Background: Lead poisoning in children can occur from different sources such as paint, food and water. Childhood lead poisoning can cause damage to the brain and nervous system, slowed growth and development and hearing and speech problems. Children with blood lead levels of 10 mcg/dL or greater are more likely to have learning and behavioral effects than children with levels of less than 10 mcg/dL (CDC, 1997).

Case Report: A 9 yr old male with a history of attention deficit/hyperactivity disorder reports increasing intermittent abdominal pain increasingly over the last 3 months. The patient admitted to possibly swallowing some of the lead fishing weights after he took up the hobby around the same time as the abdominal pain started. He had undergone lead screening 6 months prior at which time his BLL was 9 mcg/dL. He was again seen by his pediatrician at which time the BLL was 58 mcg/dL with a hemaglobin 12 mg/dL. He was sent to the emergency department where an abdominal x-ray showed 5 large foreign bodies throughout the gastrointestinal tract with additional fragments in the rectum. Whole bowel irrigation (WBI) was initiated as the recommendation of the poison control center (PCC) with minimal effect. Patient was admitted to the hospital and started on oral succimer. Lead levels gradually decreased from 45 mcg/dL on hospital day (HD) 1 to 33 mcg/dL on HD 3. WBI was continued without significant effect so colonoscopy was performed with removal of 4 weights from the large intestine. Following colonscopy, a repeat x-ray showed a retained weight in the right lower quadrant. Abdominal computed tomography confirmed that he weight was lodged in the appendix. A surgical consult was obtained and the patient underwent appendectomy without complication. Post removal lead levels were sent but not recorded and the patient was lost to PCC follow up. Child was discharged for follow up with pediatrician.

Case Discussion: Pediatric ingestion of lead fishing weights is an uncommon but serious problem. Prior experience from our center has shown success with whole bowel irrigation. However this approach does raise some questions such as the role of endoscopic removal early in a hospital stay. Additionally, lead weight ingestions pose a unique challenge. Due to their density, lead weights may have slow transit through the gastrointestinal tract which may lead to increased absorption. They are also at risk for getting entrapped in the appendix or colon diverticula. Finally, there is a question of the role of chelation when a foreign body is still present due to concern for increased absorption.

Conclusions: We present a case of lead fishing weight ingestion complicated by entrapment in the appendix requiring surgical intervention. This case highlights the risks and possible complications which can occur with pediatric lead ingestions.

KEYWORDS Lead; Toxicity; Pediatrics

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215. Acute respiratory failure due to pediatric melaleuca oil ingestion

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Background: Melaleuca oil is a mixture of approximately 15 different terpene hydrocarbons derived from the Melaleuca species. We report a case of a 19-month-old male who presented with altered mental status and acute respiratory failure after ingesting a teaspoonful of 100% melaleuca oil.
Case Report: A 19-month-old, previously healthy male presented to the emergency department (ED) obtunded and in acute respiratory failure. Two hours earlier, his father had found him with a 15mL vial of tea tree oil (100% melaleuca oil) that had been left out on the bedside table. The father estimated that the child had ingested no more than a teaspoonful of the tea tree oil. The child was asymptomatic and otherwise acting normal for his age. Over the next hour, he became increasingly lethargic and was taken to a local fire station then subsequently transported by paramedics to the ED. IV naloxone 1.25mg was administered during transport without a significant effect. On arrival, the patient was ventilated with a bag-valve-mask due to respiratory rate of 6-8 breaths per minute and shallow inspirations with poor effort. He would only open his eyes to sternal rub with minimal movement of extremities. He was noted to have a strong odor of tea tree oil. Due to his altered mental status and concern for airway protection, rapid sequence intubation with ketamine, atropine, and rocuronium was performed and a bolus of normal saline was administered for a blood pressure of 80/30 mmHg. After intubation, he had a heart rate of 131 beats per minute, blood pressure of 106/33 mmHg, and oxygen saturation of 100%. His chest was clear to auscultation, and the chest radiograph demonstrated satisfactory placement of the endotracheal tube with clear lung fields. Results of serum and urine toxicology screens were negative. The child was admitted to the pediatric intensive care unit for further management. He was easily weaned from the ventilator over the next few hours and extubated without complications.
Case Discussion: Literature on melaleuca oil toxicity is sparse. Three cases of pediatric ingestion noted somnolence, confusion, and ataxia after ingestion of 10mL or less of 100% melaleuca oil; one patient was intubated due to unresponsiveness but did not demonstrate respiratory symptoms. An 18-month-old exhibited severe altered mental status and aspiration pneumonitis after ingestion. This is the first published case report of respiratory depression due to melaleuca oil without evidence of pulmonary aspiration. The mechanism responsible for this effect has not been fully elucidated.
Conclusions: Melaleuca oil toxicity may cause significant respiratory depression in the absence of pulmonary aspiration.

KEYWORDS Melaleuca Oil; Respiratory Failure; Pediatric

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216. Prostaglandin E1 Overdose in a Term Neonate with Congenital Heart Disease

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Background: Prostaglandin E1 (PGE1) is commonly used in neonates to maintain a patent ductus arteriosus in cases of congenital heart disease. We report a medication error in a neonate resulting in PGE1 overdose.
Case Report: A full term baby girl with known large ventricular septal defect and severe aortic arch obstruction was born via uncomplicated spontaneous vaginal delivery. After delivery, PGE1 was ordered to be held at bedside but was inadvertently started and set to infuse over 30 minutes. After 15 minutes of administration, the patient had received 250 mcg of PGE1 (5 mcg/kg/min), representing 200 times the intended dose of 0.025 mcg/kg/min. The neonate became apneic with desaturation to 11%, bradycardic with a nadir in the 50s, hypotensive with a MAP of 24 mmHg, and unresponsive despite stimulation. The medication error was identified and the infusion was stopped. The neonate was immediately intubated for respiratory support and given a bolus of intravenous fluids (IVF) with positive response on blood pressure. Toxicology was called for bedside consultation. Given the pharmacokinetics of PGE1, it was felt that toxicity would be relatively short-lived and could be managed with supportive care alone. Within an hour of administration, the neonate received a total of 30 mL/kg crystalloid IVF before becoming hemodynamically stable and consistently breathing over the mechanical ventilator. The patient was subsequently restarted on PGE1 for her congenital heart disease four hours after the error without further complications.
Case Discussion: We report a case of neonatal PGE1 overdose associated with apnea, bradycardia, and hypotension. To our knowledge, this is the first such case reported in the literature. While it is unclear if pharmacokinetics of PGE1 change significantly in overdose, the expected elimination half-life of PGE1 in a therapeutic dose is very short - approximately 5-10 minutes. As such, in our patient the duration of toxicity was less than one hour despite a significant overdose and was managed with supportive therapy.
Conclusions: Supportive therapy with IV crystalloid boluses and respiratory support either through bag mask ventilation and/or endotracheal intubation may be sufficient for significant PGE1 overdoses in neonates.

KEYWORDS Prostaglandin E1; pediatric; medication error

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217. Clinical Toxicity following Suvorexant Ingestion

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Background: Suvorexant is a new sleep aid that received FDA approval in August 2014. It is the first medication in a class of orexin receptor antagonists. Currently there are no published studies on suvorexant toxicity. Adverse effects, monitoring, and treatment recommendations for overdose listed in the suvorexant

monograph are theoretical and as yet unsubstantiated. Suvorexant is a highly selective orexin receptor antagonist. Orexin neuropeptides from the hypothalamus promote wakefulness. Suvorexant is a dual orexin receptor antagonist (DORA), affecting both the OX1R and OX2R receptors. Antagonism results in shortened time to fall asleep and enhanced persistent, continuous sleep. This is the first FDA-approved drug with this unique mechanism of action. Due to its novel mechanism of action and lack of data on clinical toxicity, a review of toxicologic events was deemed relevant.

Methods: We reviewed data from the National Poison Data System (NPDS) that was compiled from calls to U.S. poison centers. The NPDS database is the most comprehensive available, with over 2.1 million human exposure calls in 2014. It contains data on 60 million human exposures since 1983 when data collection started. Inclusion criteria were: human exposure to suvorexant with exposure date from 8/1/2014 to 12/31/2015.

Results: There were 86 human exposures to suvorexant (32 male, 54 female); 43 entailed single ingestion of suvorexant only (20 male, 23 female). Patient age ranged from 18 months to 84 years, with the majority falling between 30-65 years of age. Outcomes attributed were no effect (11.6%), minor effect (26.7%), or moderate effect (19.8%); there was one death. Of the exposures, 57% were acute in nature. Of suvorexant single medication ingestions, the most common outcomes were no effect (18.6%), not followed - minimal effect (34.9%), minor effect (18.6%), or moderate effect (14%). There were no deaths or major effects in the single substance subgroup. Of single medication exposures, 74.4% were acute ingestions. Clinical effects were primarily CNS in nature and included: drowsiness/lethargy (18.6%), dizziness/vertigo (4.7%), nausea (4.7%), abdominal pain (2.3%), agitation (2.3%), confusion (2.3%), headache (2.3%), hypotension (2.3%), slurred speech (2.3%), vomiting (2.3%), miosis (2.3%), and other (2.3%).

Conclusions: This is the first retrospective case series detailing suvorexant clinical toxicity. In patients who ingested suvorexant alone, there were no deaths or major effects. The most frequent clinical effect noted was drowsiness/lethargy. This study is limited by its retrospective nature and by passive collection of reported exposures. Additional study is needed to determine degree of toxicity by dose and by serum concentration, as well as determine the most appropriate therapies.

KEYWORDS Orexin; Poisoning; Toxicity

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218. Unintentional Adult Exposures to Single-Ingredient Acetaminophen Before and After Regulatory Changes

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Background: Between 2011 and 2013, a series of regulatory events led to the implementation of interventions targeted to improve the safety of acetaminophen use in adults, including FDA advisory committee meetings, the launch of several educational campaigns, and the lowering of the recommended over-the-counter total daily dose for 325 mg and 500 mg single-ingredient (SI) acetaminophen products. The objective of this study is to compare the frequency of adult unintentional exposures (UEs) reported to US Poison Centers before and after this period of increased regulatory activity to evaluate the impact.

Methods: Data were extracted from the National Poison Data System (NPDS) database. UEs were categorized to the following periods in relation to the regulatory events: 'Before' (01 January 2007 to 31 December 2010), 'During' (01January 2011 to 31 December 2013), and 'After' (01January 2014 to 30 June 2015). Case criteria included adult (age 12-89 years) human exposures to one or more oral SI acetaminophen product. Only cases with UEs (unintentional general, unintentional therapeutic error, or unintentional misuse) were included. A general linear model with time period as a fixed effect was used to determine if the average monthly case exposure counts in the 'Before' period differed from the 'After' period.

	Before Average (95% CI)	After Average (95% CI)
All Concentrations	751.3 (725.3, 777.3)	573.9 (531.4, 616.3)
500 mg	538.9 (520.0, 557.7)	398.6 (367.8, 429.3)
325 mg	47.5 (44.9, 50.2)	46.9 (42.6, 51.3)

Results: Within the study period, 67,891 UEs were reported, with 36,062 (53%) during the 'Before' period, 21,499 (32%) in the 'During' period and 10,330 (15%) during the 'After' period. Average monthly adult UEs reported in the 'Before' period was significantly higher than in the 'After' period ($p < .0001$). Among 500 mg SI acetaminophen products, the average monthly UEs reported in the 'Before' period was significantly higher than the 'After' period ($p < .0001$). When restricted to 325 mg SI acetaminophen products fewer average monthly UEs were reported in the 'Before' period compared to the 'After' period with no significant difference detected ($p = 0.82$).

Conclusions: Analysis of NPDS exposures showed that fewer adult UEs involving SI acetaminophen were reported monthly after the 2011 to 2013 period of increased regulatory activity, particularly among 500 mg product exposures. This suggests that product safety activities that occurred during this time may have improved the safe use of 500 mg SI acetaminophen in adults. Continued surveillance of NPDS data may provide insight into SI acetaminophen safety and the long term impact of industry-wide drug safety efforts.

KEYWORDS Acetaminophen; unintentional exposures; US Poison Centers

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219. A Retrospective Review of Antipsychotic Medications Administered to Psychiatric Patients in a Tertiary Care Pediatric Emergency Department

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Background: Increasing number of pediatric patients with psychiatric chief complaints are presenting to hospital emergency departments (EDs) nationwide. Some of these patients arrive to the ED acutely agitated or become agitated during their stay, and many of them require treatment with antipsychotic medications to treat their agitation. We reviewed the use of antipsychotic medications in pediatric patients presenting to a single tertiary-care pediatric emergency department.

Methods: We performed a retrospective review of the electronic medical record from Jan 2009-Feb 2016 to identify all patients presenting to the ED with a psychiatric chief complaint who were administered at least one antipsychotic medication during their ED stay. Data collected included age, sex, whether the patient was already on antipsychotic medications prior to their ED visit,

vital signs, chief complaints, medications and dosages administered, route of administration, length of ED stay, and patient disposition.
Results: 229 patients were identified, 54.1% of whom were male. Mean patient age was 14.6 ± 2.3 years (median 15 years; range, 4-19 years). 85.2% were already on antipsychotic medications. 18.3% were tachycardic and 5.7% were hypertensive on presentation. Presenting complaints included suicidal ideation (47.6%), aggression (27.5%), homicidal ideation (10.5%), agitation (7%), psychosis (2.6%), and hallucinations (2.2%). Medications administered included olanzapine (51.1%), aripiprazole (26.6%), haloperidol (24.0%), risperidone (11.4%), quetiapine (10.0%), lurasidone (3.9%), and ziprasidone (2.2%). Eighty patients (34.9%) were given at least 1 intravenous or intramuscular dose of antipsychotic. A total of 173 patients (75.5%) were administered 1 antipsychotic medication, 43 (18.8%) were administered 2 antipsychotics, and 13 (5.7%) were administered 3 antipsychotics during their ED stay. A total of 191 patients (83.4%) were admitted to an inpatient mental health facility; 38 (16.6%) were discharged. Median length of stay was 683 min (IQR 410-1239) for patients given 1 antipsychotic, 888.5 min (IQR 549-1496) for patients given 2 antipsychotics, and 1239 min (IQR 874-1652) for patients given 3 antipsychotics (Kruksal-Wallis $\chi 2 = 7.48$; $p = 0.024$). Length of stay was shorter in patients given only oral medications (617 min, IQR 418-1194) compared to those given at least 1 parenteral dose of antipsychotic (979 min, IQR 458-1671) ($z = -2.47$; $p = 0.014$).
Conclusions: In this retrospective series, the majority of patients were treated with newer oral antipsychotics; a substantial number required treatment with 2 or more medications. Most patients did not have objective signs of agitation (e.g., hypertension or tachycardia) prior to medication administration. Administration of multiple medications was associated with a longer length of stay in the ED, as was parenteral administration of antipsychotics.

KEYWORDS Pediatric; antipsychotic; psychiatric

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220. Treatment of salicylate-induced respiratory failure in a 12-month old infant using extracorporeal membrane oxygenation

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Background: Severe salicylate toxicity can result in pulmonary edema and occurs more commonly in adults. We report the successful use of extracorporeal membrane oxygenation (ECMO) for treatment of salicylate induced respiratory failure in a 12-month old child.
Case Report: a 12-month old previously healthy boy presented to the emergency department (ED) after being found with increased work of breathing and a single episode of emesis by his mother. On arrival to the ED the patient was awake, in moderate distress, with a HR 190 and RR 48-55, oxygen saturation 94% on 8L O2 by NC. Lung exam revealed severe retractions, increased work of breathing, and crackles throughout. His venous blood gas showed a pH 7.31, pCO2 19, HCO3 9.7; serum salicylate level peaked at time of ED presentation at 80.5 mg/dl. Initial chest x-ray demonstrated mild hazy opacities bilaterally, with subsequent CXR on admission to the pediatric ICU demonstrating pulmonary edema in bilateral lung fields. Patient was administered IV bicarbonate therapy and noninvasive positive pressure ventilation (NIPPV), however he failed NIPPV due to hypoxia and fatigue and was subsequently intubated and ventilated. Following intubation frothy oral fluid was noted and there were difficulties maintaining his oxygen saturation, requiring continuous bag valve masking with high positive end-expiratory pressures; despite these measures the patient became more acidotic (pH 7.43), hypoxic (pO2 54mmHg, FiO2 100%, PEEP 18), and hypotensive (BP 72/33, HR 185) refractory to norepinephrine infusion. The patient was then placed on veno-venous ECMO (flow 95-105 ml/kg/min, sweep 1.2 goal CO2 30-35 mmHg, oxygen 100%) with improvement in oxygenation (pO2 68, FiO2 40%, PEEP 12), acid-base status (pH 7.54), and hemodynamics (BP 105/51). CRRT was performed concomitantly to aid in clearance of salicylate. After eight days of ECMO therapy, the patient was weaned from ECMO treatment and decannulated. He was discharged from the hospital without sequelae on hospital day 21.
Conclusions: ECMO was a successful adjunct for the treatment of pulmonary edema and severe respiratory failure secondary to salicylate toxicity in an infant.

KEYWORDS salicylate; extracorporeal membrane oxygenation; pediatric

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221. Inadvertent Methylergonovine Administration to a Neonate

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Background: Methylergonovine is an ergot alkaloid used to treat post-partum hemorrhage secondary to uterine atony. Reports of accidental neonatal administration secondary to confusion with vitamin K are rare in the United States. Complications of ergot alkaloids in neonates include respiratory depression, seizures, and death.
Case Report: Baby boy W (3520 grams) was born at 37 + 5/7 weeks gestation to a 31 year old gravida 2 para 1 woman with an unremarkable pre-natal history. The delivery was uncomplicated and initial APGAR scores were 9 and 9. Two hours after birth, while still in the delivery room, the neonate was inadvertently given 0.1 mg of methylergonovine intramuscularly in the right thigh. The error was only noted when the vial of medication was scanned, after administration, identifying it as methylergonovine rather than vitamin K. The vials of methylergonovine and vitamin K are of similar size and shape single use glass vials. The local poison center was notified and the baby was transferred to the neonatal intensive care unit for observation. Two after transfer, the neonate was noted to de-saturate to the low 80's requiring 2 liters oxygen via nasal cannula. Supplemental oxygen was continued for 4 hours until the neonate was able to maintain oxygen saturations on room air. Feeding was started by 10 hours of life and the baby was discharged in good condition after a 72 hour stay without further complications.
Case Discussion: Confusion of methylergonovine for vitamin K with accidental administration to the neonate is a rare iatrogenic illness occurring almost exclusively in the delivery room setting. Both drugs are dispensed in similarly sized, colored, and shaped single use glass vials. This similarity in the medications' packaging likely contributes to its' occasional erroneous administration. While the current practice of maintaining maternal and neonatal contact after birth is important to the bonding process, it does provide the opportunity for a potentially significant medication error. It is important to highlight this risk so that future errors can be prevented.
Conclusions: Vigilance is required to prevent accidental administration of methylergonovine to the neonate because of possible

confusion with vitamin K in the early post-partum period and the potential for serious adverse events.

KEYWORDS Methylergonovine; Neonate; Ergot

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222. Prolonged Somnolence and Hypoglycemia in a Pediatric Opioid Overdose

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Background: Pediatric opioid exposures continue to increase. The physiology and metabolism of pediatric patients poses a challenge for toxicologists in terms of treatment and management. The opioid toxidrome and its duration may vary in children. We present a case of a pediatric opioid ingestion with prolonged somnolence.
Case Report: An 11 month old girl presented to the Emergency Department (ED) overnight for nasal congestion. The patient had a normal exam and was discharged home. She was seen at her doctor's office 12 hours later and was lethargic with oxygen saturation of 80%. Upon EMS arrival blood glucose was <20 mg/dL. She received intramuscular glucagon and was transported to the ED. In the ED her vitals were: blood pressure 80/35mmHg, temperature 38.7 °C, respiratory rate 57/minute, oxygen saturation 100% on NRB. On examination she was lethargic with a rash on her extremities. A 10% dextrose bolus was given with subsequent normal blood glucose. Laboratory analysis revealed a WBC count of 14,750, potassium 7.3 mmol/L, glucose 347 mg/dL, ALT 1342, AST 1752, AlkPhos 178 IU/L. Venous blood gas showed pH of 7.17, pCO2 55.6 mmHg, bicarbonate 20.3 mmol/L, and lactic acid 2.9 mmol/L. CK was 640. Her urinalysis and chest x-ray were unremarkable. Her mental status improved after glucose administration and she was admitted to the PICU. Upon arrival to the PICU, providers noted pinpoint pupils, facial swelling and pressure sores to the forehead and bilateral knees. She remained lethargic with a respiratory rate of 12 and sonorous respirations. Intravenous naloxone 0.8 mg (0.1 mg/kg) was administered with improvement in her alertness and respiratory drive. Providers then became aware that there were opioids in the home. CT of the brain was normal. She had one additional episode of hypoglycemia 3 hours after the dextrose bolus but subsequently maintained euglycemia. N-acetylcysteine was started and 4 more doses of naloxone were needed during hospital day 1. She remained somnolent with miosis until hospital day 3. She continued to have rising AST/ALT which peaked at 2429/1273 IU/L and resolved. Gas chromatography/mass spectrometry yielded morphine in her urine.
Case Discussion: Prolonged somnolence in pediatric patients following an opioid ingestion may persist well outside the expected period of metabolism. If pediatric patients can have prolonged somnolence with opioid exposure, then it is reasonable to expect delayed respiratory depression. Additionally, children have decreased glycogen stores and may have hypoglycemia following prolonged somnolence.
Conclusions: Clinicians should be aware that children may have somnolence that is prolonged beyond the time period expected from adult metabolism. Caution should be used when observing these patients following an opioid exposure.

KEYWORDS Pediatric; opioid; prolonged somnolence

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223. Delayed Stridor after a Pediatric Laundry Pod Ingestion and Aspiration

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Background: Laundry detergent pod ingestion and aspiration have been associated with an array of clinical manifestations in pediatric patients. These range from asymptomatic to severe respiratory distress or ARDS requiring intubation and typically manifest within 6 hours of exposure. We present a case of delayed stridor in a pediatric patient following a laundry pod ingestion and likely aspiration.
Case Report: A 16-month-old girl with no significant medical history presented to the Emergency Department after ingestion of a detergent pod. Just prior to arrival, the patient's mother discovered her with a detergent pod in her mouth and promptly removed it noting that approximately half the pod was ingested. The patient had 3 episodes of non-bloody emesis at home. In the Emergency Department, no initial RR was recorded but her oxygen saturation was documented as 92% on room air and repeated shortly afterwards with no intervention and was 99%. The patient was noted to be crying but consolable in moderate respiratory distress. Her lung exam revealed scattered rhonchi in the lung bases without stridor but some increased work of breathing. No oropharyngeal lesions were visualized. CXR displayed no acute findings. The patient's respiratory symptoms improved with nebulized albuterol while in the Emergency Department and she was admitted to the Pediatric ICU for observation. Upon admission her respiratory rate was documented at 28 breaths per minute. Approximately 12 hours post ingestion the PICU team noted the development of stridor. There was no associated hypoxia but continued coarse breath sounds and a respiratory rate of 38. At this point dexamethasone was administered. Within 6 hours the stridor resolved. The patient was discharged home the next day.
Case Discussion: Pediatric ingestions of household items is a common occurrence. In recent years as laundry detergent pods have become increasingly popular, we have seen a rise in pediatric exposures. Typically respiratory distress and signs of caustic injury such as vomiting, drooling, and stridor occur within the first 6 hours of exposure. Our patient had initial vomiting and lower lung findings, but had a delayed presentation of stridor.
Conclusions: As laundry detergent pod exposures continue to occur, it is important for clinicians to be aware that delayed upper airway edema causing stridor may occur. It is unclear whether this is a manifestation which may occur in the initially asymptomatic patient.

KEYWORDS Pediatric; Laundry Pod; Delayed Stridor

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224. Unintentional ingestion of methylcyclopentadienyl manganese tricarbonyl due to label misperception

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Background: Household items are frequent sources of unintentional poisonings, despite improvements in product labeling.

Packaging similarities between toxic and nontoxic products may lead to confusion over a product's intended use. We report a case of unintentional methylcyclopentadienyl manganese tricarbonyl ingestion due to label misperception.
Case Report: A 54 year-old male with history of methamphetamine use presented to the Emergency Department two hours after drinking 12 ounces of NOS Octane Booster Racing Formula containing 5% methylcyclopentadienyl manganese tricarbonyl (MMT). The patient's girlfriend reported that the patient obtained the product from a food bank and thought it was an energy drink due to labeling similarities with NOS High Performance Energy Drink. Shortly after arrival, the patient had two generalized seizures and was subsequently intubated. He was transferred to the intensive care unit on a midazolam infusion for seizure control. Two hours following admission, the patient had an additional seizure, and he was transitioned to propofol. Two days following ingestion, the patient had another seizure during an attempt to wean sedation. The patient was extubated four days post-ingestion, but he remained confused and ataxic. His symptoms resolved five days after the ingestion. After reviewing the respective product containers, the local poison center reported this case to the Food and Drug Administration (FDA) and cited the potential for consumer confusion and unintentional exposure due to labeling similarities between NOS Octane Booster Racing Formula and NOS High Performance Energy Drink.
Case Discussion: MMT is an organometallic additive used to increase the octane level of gasoline and to enhance its anti-knock properties. Exposure to MMT has been previously described in environmental and occupational settings through chronic inhalation of combustion fumes. Clinical manifestations include pneumonitis, neuropsychiatric symptoms, and Parkinsonism. To our knowledge, we report the first case of MMT ingestion characterized by severe, reversible neurotoxicity. Additionally, this case highlights the potential dangers of similar product labeling among toxic and nontoxic products. Poison centers and clinicians should report such cases to the FDA.
Conclusions: Acute MMT toxicity may be characterized by acute, reversible neurotoxicity. Manufacturers should clearly label toxic products and choose packaging that is unlikely to be confused for consumable products. Efforts should be made to report misleading or similarly marketed toxic and nontoxic products to prevent unintentional exposures.

KEYWORDS MMT; product labeling; unintentional overdose

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225. Exploratory Rivaroxaban Ingestion in a Child Associated with Elevated Coagulation Studies

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Background: Rivaroxaban is one of several novel oral anticoagulants (NOACs) developed for prevention and treatment of thromboembolic disease. It is a direct factor Xa inhibitor with predictable pharmacokinetics and pharmacodynamics, and does not commonly cause alteration in coagulation studies in normal use, allowing use without routine monitoring as in vitamin K antagonists (VKA). However, there is a paucity of data on its effects in children. We present a case of a 3 year old presenting after an exploratory ingestion of rivaroxaban.
Case Report: A previously healthy 3-year-old girl was discovered by family to have ingested 60mg (4.4mg/kg) of her aunt's prescribed rivaroxaban from an open pill box. She was taken to a nearby ED where she was found to be asymptomatic with no signs of active bleeding and normal vital signs. Basic metabolic profile, liver function tests, and complete blood count were within normal limits. Coagulation studies were notable for prothrombin time (PT) 47.6 seconds (normal range 11.5-16.1 seconds), International Normalized Ratio (INR) 4.4 (normal range 0.9-1.3), and Partial Thromboplastin Time (PTT) 87.4 seconds (normal range 27-39.4 seconds). Activated charcoal was administered 90 minutes after ingestion and the patient was transferred to a tertiary children's hospital. The patient was admitted for observation. A plasma rivaroxaban level drawn 9.5 hours after ingestion was 78 ng/mL. The following morning, coagulation studies had normalized with PT 16.7 seconds, INR 1.3, and PTT 40 seconds. She continued to have no signs of bleeding and was discharged home.
Case Discussion: We present a case of an exploratory pediatric rivaroxaban ingestion. Rivaroxaban and other NOACs are being prescribed more frequently, making accidental pediatric ingestions increasingly likely. While coagulation studies are not used for routine monitoring, transient abnormalities in both PT/INR and PTT have been described in adults. This case shows that pediatric ingestions can also cause derangement in coagulation studies. The half-life of rivaroxaban is 5-9 hours, significantly shorter than VKAs such as warfarin, thus normalization of coagulation studies is seen relatively quickly. The patient's plasma rivaroxaban level was ultimately found to be elevated, although likely the peak concentration was much greater than the level observed due to delay to obtaining a serum level and administration of activated charcoal. No evidence of derangement of hepatic function was observed, making interference with laboratory .
Conclusions: Pediatric ingestions of novel oral anticoagulants have been rarely reported. Effects in this population are largely unknown but thought to mirror those in adults, but transient elevations in coagulation studies can be noted in rivaroxaban ingestions, and generally resolve quickly given the medication's short half-life.

KEYWORDS Rivaroxaban; coagulation; pediatric

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226. Unintentional pediatric liquid nicotine ingestion necessitating intubation, with accompanying mass spectrometry analysis

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Background: Exposures to electronic cigarettes and their refillable liquid nicotine are increasing, according to the Centers for Disease Control and Prevention and the American Association of Poison Control Centers. An average conventional cigarette delivers between 0.2-2.4 mg of nicotine, of which 80-100% is absorbed during deep inhalation. In studies of habitual smokers, peak plasma nicotine concentrations range between 12-54 ng/mL. The "lethal" dose of nicotine (in an adult) has been estimated at 60 mg, and although countless textbooks and reference materials consistently cite this idiopathic figure, a recent exhaustive literature review supports an increased lower-limit fatal nicotine dose of at least 500-1000 mg in adults, which corresponds to an oral LD50 of 6.5-13 mg/kg. We report here a case of severe toxicity in

a child who ingested a well-documented amount of liquid nicotine, with confirmatory serum and product analysis.
Case Report: A 6 year-old girl was given a measured 10mL dose of liquid from an ibuprofen bottle by her father, which caused an immediate burning oral sensation. The patient's mother had purchased liquid nicotine online, diluted it 50:50 with propylene glycol/vegetable glycerine, and unbeknownst to her husband had stored it in a used ibuprofen bottle, which she labeled "NIC" (Figure 1). In the emergency department, the patient's vital signs were HR 99, BP 93/70 mmHg, 35.2oC (rectal), SpO2 95%, weight 20 kg. Her heart rate decreased to 60 bpm and she developed vomiting, diaphoresis, disconjugate gaze, fasciculations, obtundation, and inability to control her copious secretions. She was intubated. Labs were notable for hyperlactatemia (4.4 mmol/L), hypokalemia (2.7 mmol/L), leukocytosis (15 000/mm3), and mildly elevated serum creatinine (0.7 mg/dL). The patient was admitted to the pediatric intensive care unit, extubated the following day, and discharged in stable condition. A serum sample obtained 30 minutes after the ingestion was notable for detectable nicotine (348 ng/mL), cotinine (742 ng/mL), and 3-hydroxy-cotinine (32 ng/mL). A comprehensive urine drug screen obtained 18 hours post-ingestion detected nicotine (140 ng/mL), cotinine (>1600 ng/mL), and metabolites of medications administered during hospitalization (midazolam, lorazepam, and fentanyl), but was otherwise negative for >300 analytes. The contents of the ibuprofen bottle were analyzed with liquid chromatography–quadrupole time-of-flight mass spectrometry (LC-QTOF/MS) and found to contain 70.3 mg/mL nicotine with no other analytes.
Case Discussion: Exposures to electronic cigarettes and their refill fluid are increasing, with potentially significant clinical effects. With supportive care, this patient survived an ingestion (703 mg; 35 mg/kg) several times higher than the previously reported "lethal" dose of nicotine, supporting previous arguments to reconsider this "lethal" dose.4 Furthermore, after two-fold dilution, the final nicotine concentration of the ingested liquid (70.3 mg/mL) was greater than that of the original product label ("60 mg/mL"; Figure 2).
Conclusions: Refill products may have unreliable commercial labeling. This case demonstrates the use of technologies such as LC-QTOF/MS to set a higher standard for establishing drug toxicity and collecting and reporting comprehensive and quantitative data.

KEYWORDS Liquid nicotine; pediatric; electronic cigarette

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227. Prolonged sedation and QTc prolongation after unintentional overdose of ondansetron by a toddler

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Background: Serotonin receptor (5-HT3) antagonists such as ondansetron are commonly used off-label for nausea in emergency and acute care settings. Unintentional pediatric ingestions, while common, rarely result in significant clinical effects. Adverse effects have been reported both in therapeutic use and in overdosed pediatric patients, but there is limited data on duration of toxicity. We describe a case of prolonged sedation and QTc prolongation in a 2-year-old child after ingesting 56 mg of orally disintegrating (OD) ondansetron.
Case Report: A 33-month-old African American male, weighing 13.2 kg, reportedly ingested seven OD tablets of ondansetron, 8 mg. Ingestion history was deemed reliable and time of ingestion was approximately 11:00 a.m. The child did not have any other medication or toxicant available to him. He was brought to an emergency department (ED) for somnolence after vomiting once at home. On arrival, he was sleepy but arousable and protecting his airway. Initial vital signs and lab workup were normal. Urine toxicology screen was negative. Electrocardiogram (ECG) obtained 90 minutes after ingestion showed sinus rhythm at 111 bpm. The Bazett-corrected QTc was 492 msec, while the T peak-to-end interval (Tp-Te) was 212 msec in lead V5. At 5 hours post-ingestion, the child was deemed more alert and was about to be discharged from the ED. Upon discharge, he appeared more sedated and was transferred to a pediatric specialty hospital for further care. He was monitored through 9 hours post-ingestion, without demonstrating arrhythmia or clinical findings of serotonin syndrome (SS). His mentation gradually improved, and he was discharged home in baseline condition. Repeat ECG prior to discharge again showed sinus rhythm at 116 bpm with improved QTc at 464 msec. The Tp-Te had decreased to 84 msec.
Case Discussion: Serious adverse effects are rarely reported from pediatric ingestions of ondansetron. Obtundation, seizures, hepatotoxicity, QTc prolongation, loss of airway reflexes requiring endotracheal intubation, and SS were reported in an infant. Hypotension was reported in an 8-year-old after ingesting 60 mg of ondansetron. Death was reported after a therapeutic dose to a child with known susceptibility to malignant hyperthermia. There is little information available on the incidence or duration of sedation after pediatric ingestions. Significant QT prolongation and arrhythmias are unlikely with therapeutic dosing.
Conclusions: We describe a case of prolonged sedation and QTc prolongation from unintentional OD ondansetron ingestion by a 2-year-old child. Further work is needed to clarify the mechanism and duration of effects after overdose with such agents.

KEYWORDS Ondansetron; QTc prolongation; prolonged sedation

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228. Pediatric Hair Relaxer Ingestion with Delayed Severe Respiratory Burns

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Background: Hair relaxer creams traditionally contain sodium hydroxide but the no-lye formulations often contain calcium or guanidine hydroxide. These agents are alkaline caustics (pH 12-13). Due to their high viscosity, burns from ingestions are limited to the lips and mouth. Respiratory burns are rare and have not been reported without lip, mouth, or esophageal involvement. We report a case of delayed respiratory symptoms requiring prolonged intubation and tracheostomy after a hair relaxer ingestion.
Case Report: A healthy 10-month-old boy was found with cream on his hands and mouth from "Africa's Best Hair Relaxer" no-lye formulation. His mother cleaned him then gave him milk. Twenty minutes later, he began drooling and was taken to an emergency department (ED) where he vomited once. He was observed for 6 hours, tolerated clear liquids, and discharged without abnormal drooling or stridor. Three hours later, he developed sudden breathing difficulty and represented to the ED. He was treated with nebulized epinephrine and intravenous steroids for stridor before transfer to a tertiary care facility.
After transfer, he continued to have increased work of breathing and stridor. Urgent endoscopy showed an estimated 30%

subglottic stenosis, mild erythema and necrosis above the cricopharyngeus but normal epiglottis, arytenoids, vocal cords, esophagus, stomach, and duodenum. Endotracheal and nasoduodenal tubes were placed.
His course was complicated by three unsuccessful extubation trials. Repeat laryngoscopy showed edema of the arytenoids and vocal cords with persistent subglottic stenosis. A tracheostomy was placed on hospital day (HD) 14.
On HD 20, he was weaned off the ventilator and tolerated pureed food and liquids by mouth. The tracheostomy collar was maintained to treat the subglottic stenosis.
Case Discussion: Children with caustic ingestions are assessed for stridor or a combination of vomiting and drooling to determine the need for endoscopy. Since hair relaxers are usually creams, children may present with drooling and mouth lesions; endoscopy or intubation are seldom required. Babl, et al. reported an 11-month-old boy who was observed for 2 hours then developed stridor after discharge from the ED. He had mild airway erythema and subglottic stenosis but did not require intubation. Endoscopy showed mild erythema of the upper esophageal sphincter. Our patient developed severe respiratory symptoms 10 hours after ingestion requiring intubation and tracheostomy placement despite appropriate early management.
Conclusions: This case highlights a rare but serious complication that occurred from hair relaxer exposure. Delayed respiratory symptoms should be considered when managing these children.

KEYWORDS Hair relaxer; pediatric caustic; airway burns

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229. No Skin in the Game: Characterization of Human Oclacitinib Exposures Called to Regional Poison Control Center

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Background: The veterinary pharmaceutical oclacitinib, marketed under the brand name Apoquel® in the USA, was approved by the FDA in May 2013 for treatment of allergic dermatitis in canines greater than 12 months of age. Oclacitinib is in the class of medications known as Janus kinase (JAK) inhibitors, and they serve to interfere in the JAK-STAT signaling pathway by inhibition of one or more of the Janus kinase family of enzymes. Inhibitors of Janus kinase have therapeutic application in human medicine for treatment of cancer, and inflammatory conditions such as rheumatoid arthritis and psoriasis. We report what we believe to be the first poison center analysis of human exposures to the canine drug oclacitinib.
Methods: All human exposure cases called into the poison control center from the FDA approval date, May 2013, through February 2016 were evaluated if they involved the veterinary drug oclacitinib. This is a retrospective review of those cases. Data was broken down for gender, age, dose ingested, management site, clinical effects, and outcome.
Results: A total of 20 human exposures were identified in the database involving ingestion of the veterinary pharmaceutical oclacitinib. The first case was called in September, 2014, more than a year following the FDA approval. The age range involved persons 2 years of age up to 73 years, and three quarters of the cases involved females. Only 10% of cases involved children under the age of 12 years. All doses were known amounts, with a range of 2.7milligrams to 16 milligrams. In 90% of the cases, the canine's owner ingested oclacitinib instead of their own medication as both were placed in close proximity. Clinical effects reported included nausea (1), diarrhea (1), and chest discomfort (1). Treatment recommended and/or performed included oral fluids and Maalox. Nearly all the cases were managed at home, and of the cases followed to outcome, there were no serious adverse effects reported.
Conclusions: All of the human exposures to oclacitinib in our study were acute unintentional exposures. The two children obtained access just prior to when the canine was to get medicated, and the adults inadvertently ingested the medication when they were simultaneously retrieving their own medication. None of the cases followed to outcome involved a serious adverse event. Dog owners would benefit from additional education to separate their medication from their own and to keep all of them out of reach of children.

KEYWORDS Oclacitinib; Therapeutic misadventure; Janus kinase inhibitor

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230. Venlafaxine: Effex-your baby's blood sugar?

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Background: Venlafaxine and its active metabolite, O-desmethylvenlafaxine, inhibit neuronal uptake of serotonin, norepinephrine, and dopamine. In overdose, it can cause serotonergic toxicity with agitation, tremor, and autonomic instability. Hypoglycemia has also been observed in case reports of large overdoses in adults. We present a case of hypoglycemia after confirmed exposure to venlafaxine in an infant.
Case Report: A 9-month-old female infant was suspected to have ingested a parent's venlafaxine XR 150 mg tablet, a 20.3 mg/kg dose. She presented to the emergency department with tremor, flushing, dilated pupils, hypertension, and altered mental status. Glucose was 70 mg/dL. She was treated with lorazepam with improvement in symptoms. However, 15 hours after suspected ingestion time, she had dilated pupils and altered mental status. Blood glucose was checked and was 35 mg/dL. She was treated with intravenous dextrose and had subsequent low blood sugars between 50-60mg/dL. Patient was able to wean off dextrose infusion 24 hours later without recurrent hypoglycemia. She was discharged 60 hours post-ingestion when she remained asymptomatic. Serum comprehensive drug screen collected at the time of hypoglycemia was positive for venlafaxine 103 ng/ml and desmethylvenlafaxine 448 ng/ml, with no other drugs detected. There were no oral hypoglycemics available for exposure in the infant's home.
Case Discussion: This infant had a confirmed exposure to venlafaxine and subsequent hypoglycemia. The mechanism for this is unknown, but has been proposed to be part of serotonin toxicity. This child had symptoms of serotonin toxicity with tremor, agitation, and hypertension. Although her initial symptoms appeared to be resolving, hypoglycemia developed approximately 15 hours post-ingestion. The delay may be due to the peak of desmethylvenlafaxine, which is later than the parent compound and delayed further in overdose, although a mechanism for this difference in presentation is not apparent. She was taking less of her regular diet prior to admission and starvation hypoglycemia cannot be excluded, but this is less likely with the degree of decreased intake that was observed. Clinicians should be aware of the potential for hypoglycemia with venlafaxine exposures, especially in young children and infants who have lower glycogen stores. The onset may be delayed, especially with the extended-

release formulation, and extended observation periods should be considered.
Conclusions: Hypoglycemia is a possible sequela of venlafaxine overdose in children. Because the onset may be delayed, extended lengths of observation should be considered.

KEYWORDS Pediatrics; Hypoglycemia; Serotonin syndrome

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231. Unintentional Ingestion of Extended Release Oral Suspension of Methylphenidate With Serial Serum Levels

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Background: Methylphenidate is a stimulant widely used in the treatment of attention deficit hyperactivity disorders (ADHD). Recently, an extended release oral suspension of methylphenidate (Quillivant XR™) was introduced which potentially could cause delayed or prolonged toxicity in either intentional or unintentional ingestions. We report a case of unintentional ingestion of methylphenidate extended-release oral suspension (MEROS) in a pediatric patients confirmed with serial serum levels.
Case Report: A 5-year-old, 29.6 kg male with history of ADHD and possible post-traumatic stress disorder was brought to the emergency department (ED) after ingesting of an unknown amount of his prescribed MEROS 2 hours prior. His initial vital signs were temperature of 36.7 °C, pulse of 84 bpm, blood pressure of 120/89 mmHg and respiratory rate 16 with an oxygen saturation of 100% on room air. In the ED he was not agitated. Due to the concern regarding his ingestion, he received a prophylactic dose of 0.5 mg lorazepam and was admitted to the intensive care unit for observation. Approximately 14 hours from the reported ingestion he developed agitation and hallucinations and his pulse increased to 118 bpm. He also continued to be hypertensive with systolic blood pressures in the 120-130's mmHg range. He received 5 doses of lorazepam totaling 4mg over the next 20 hours. His agitation and hypertension resolved approximately 36 hours from the reported ingestion. Serum methylphenidate levels were obtained and are listed in Table 1.
Case Discussion: The extended release oral suspension of methylphenidate was introduced in 2013. The technology with allows extended release of an oral suspension is proprietary but reported to involved particles of a cationic polymer matrix that bind methylphenidate via an ion exchange mechanism and then are coated to various thicknesses. Literature suggests that MEROS is a blend of uncoated and coated particles that is ~20% immediate release and 80% extended release methylphenidate. This case demonstrated a peak methylphenidate level 6 hours from ingestion while symptom onset was 14 hours from ingestion. There are no other prior reports of MEROS ingestions which to compare our case with.

Table 1. Serial serum methylphenidate levels.

Time from Ingestion (hours)	Methylphenidate level (ng/ml) (Reference range: 5.0–20.0 ng/ml)
3	68.4
6	84.5
10	58 9
16	28
24	11

Conclusions: Peak serum level and symptom onset were delayed in this unintentional ingestion of MEROS. Medical toxicologists and other health care providers should be aware of the potential for delayed and prolonged toxicity with this ingestion.

KEYWORDS Methylphenidate; Extended Release; Pediatric

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232. Too much NRG-3 for a 3 year old? Ataxia associated with unintentional synthetic cathinone exposure

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Background: Synthetic cathinones are a structurally diverse group of novel psychoactive substances (NPS) which are reported to cause a wide range of toxicity. However, unintentional exposures to synthetic cathinones are rarely reported. We describe a case of 3 year old with new onset ataxia found to have high urine levels of 2-(methylamino)-1-(naphthalen-2-yl)pentan-1-one (NRG-3) by liquid chromatography- quadrupole time-of-flight mass spectrometry (LC-QTOF/MS).
Case Report: A 3 year old girl with a history of bronchopulmonary dysplasia, seizure disorder and developmental delay was brought to the emergency department for sudden inability to stand or walk. The patient had a witnessed ground level fall 5.5 hours prior to arrival without loss of consciousness or seizure-like activity. Two hours later the patient was noted to be unable to walk or sit up and to be less verbal and interactive. Initial vital signs were: heart rate 135 bpm, temperature 37.5 °C, and respiratory rate 24/min with a 100% O2sat on room air. A blood pressure was not obtained. Her exam was notable for her being non-verbal but able to follow commands. She was ataxic on sitting and could not ambulate. No nystagmus was noted. No ingestion was reported. She had not been on seizure medication for over 1 year but was prescribed albuterol and sildenafil for her pulmonary disease. Per mother the child had received a diphenhydramine containing cold product that morning. There were no other medications in the home. CT scan of her head was normal. Laboratory results were normal including a negative urine drug immunoassay and serum ethanol level. She was admitted for observation and here ataxia continued. An MRI of the brain, cervical and thoracic spine was normal, as was a magnetic resonance angiogram/venogram of the brain and neck. An initial electroencephalogram suggested possible epileptiform activity and the child was started on levetiracetam without improvement. A repeat electroencephalogram 3 days later was normal. The patient had two normal lumbar punctures done 6 days apart. In total, she had a 21 day hospital course, including an inpatient rehabilitation stay. Her ataxia slowly resolved and was attributed to cerebellitis. Ten days after discharge she was noted to be ambulating without assistance. Urine from admission was sent for analysis by LC-QTOF/MS (QTOF/MS 6550, LC 1260, Agilent) and demonstrated 4.9 ug/mL NRG-3 along with a formula match to its reduced metabolite, C16H21NO. Investigation, including a home visit, could not identify the source of the exposure.

Case Discussion: NRG-3 is a synthetic cathinone which has not previously been identified in biological specimens. While the toxicity of NRG-3 is unknown, ataxia was noted in animal studies of other synthetic cathinones. Other phenyethylamine based stimulants, such as methylenedioxy-methamphetamine (MDMA) and methamphetamine have been described to cause ataxia in humans. The prolonged symptoms seen in this case have not previously been described with synthetic cathinones but have been associated with MDMA.
Conclusions: Unintentional exposure to NRG-3 was associated with profound and prolonged ataxia. Health care providers should be aware of this association.

KEYWORDS Synthetic Cathinone; NRG-3; Ataxia

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233. Death from liquid nicotine ingestion in a pediatric patient

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Background: Reports of liquid nicotine exposures to the American Association of Poison Control Centers increased by 145% from 2013 to 2014. A majority of these exposures were unintentional and occurred in pediatric patients. The introduction of refillable electronic cigarettes spurred the production of liquid nicotine preparations with varying concentrations, some as high as 100%. We report the first case of an accidental ingestion of a liquid nicotine preparation resulting in a pediatric death.
Case Report: 18 month old male with no medical history was found by his family with vomiting, altered mental status, and seizure activity. He was alone for a brief moment and drank from an uncapped container of "Heartland Vapes 100 mg" liquid nicotine. Symptoms developed within minutes. On emergency medical services arrival, he was pulseless. He was intubated and CPR was initiated. En route to the Emergency Department (ED) ongoing efforts for resuscitation were performed. On arrival to the ED he remained pulseless and the Poison Center was contacted. Twenty percent intravenous lipid emulsion was recommended, however never administered. Resuscitation efforts were unsuccessful and he expired shortly after arrival. Autopsy revealed no blunt force trauma or penetrating injury, no significant natural disease, and no anatomical abnormalities. Post-mortem toxicology revealed cardiac blood nicotine and cotinine concentrations of 4,700 ng/mL and 220 ng/mL, a gastric nicotine concentration of 620,000 ng/mL, and urine nicotine and cotinine concentrations of >800 ng/mL and 81 ng/mL. No peripheral blood samples were obtained.
Case Discussion: Nicotine toxicity has been well described in the literature. Liquid nicotine products pose a unique concern given their high concentration, lack of child resistant packaging, and desirable flavors. In our patient, the onset of symptoms was near immediate after exposure. The concentration of the product is unknown. There are no known antidotes and care is largely supportive. Varenicline, a nicotine receptor partial agonist, may have a role in the management of nicotine exposed patients. Further investigation is needed to determine if it has the ability to displace the full agonist from the nicotine receptor. Significant regulatory changes occurred after this death. The Child Nicotine Poisoning Prevention Act of 2015 now requires manufacturers of liquid nicotine preparations to comply with the Poison Prevention Packaging Act.
Conclusions: Liquid nicotine formulations are highly concentrated, easily accessible, aesthetically packaged, and flavored in a way that might be appealing to young children. A minimal exposure can result in significant morbidity and mortality.

KEYWORDS Liquid nicotine; pediatric; death

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234. Iatrogenic lamotrigine toxicity manifesting as encephalopathy with chorea

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Background: Lamotrigine is an antiepileptic with sodium channel blockade properties. Overdose can result in cardiac toxicity and neurologic symptoms including "paradoxical" seizures. Choreiform dyskinesia in the setting of lamotrigine toxicity is rarely described.
Case Report: A 16-year-old male with a past history significant for Duchene's Muscular dystrophy presented electively to a pediatric tertiary care center for initiation of nighttime non-invasive positive pressure ventilation for chronic hypercapnic respiratory failure. Confusion over the name and dose of a recently prescribed anti-epileptic medication resulted in the administration of 5 doses of 300mg lamotrigine over the course of three hospital days. The patient developed progressive mental status changes including slurred speech and ataxia. At the time of evaluation, comprehensive evaluation including CT head, MRI, and infectious workup were negative. He was found to have low grade tachycardia, minimal purposeful movement, and minimal response to noxious stimuli. Neuromuscular findings progressed into non-purposeful, low frequency choreiform movements of the face, extraocular musculature, and extremities. He developed repetitive vocalizations. Serial electrocardiograms demonstrated no appreciable QRS prolongation. Serum lamotrigine level performed by liquid chromatography and mass spectroscopy returned elevated at 24 g/ml (normal range 3-14 g/ml). After discontinuation of all his medications (lamotrigine and paroxetine), the patient's symptoms progressively improved with supportive care. He was discharged after return to his baseline neurologic status.
Case Discussion: This is a case of severe iatrogenic lamotrigine toxicity manifesting as mental status depression with choreiform movements. Choreiform dyskinesias caused by lamotrigine is rarely reported. Treatment includes supportive care and lamotrigine discontinuation.
Conclusions: Lamotrigine toxicity can result in mental status changes and neuromuscular findings including encephalopathy and choreiform dyskinesias.

KEYWORDS Lamotrigine; Chorea; Encephalopathy

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235. That doesn't belong there! Unusual pediatric Epipen® adverse effect from accidental deployment

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Background: EpiPen® auto-injector devices are designed for the emergency treatment of life-threatening allergic reactions

(anaphylaxis) and are intended for emergency therapy only. They are critical in the reversal of symptoms for those individuals experiencing these reactions. Side effects include tachycardia, arrhythmias, sweating, nausea, vomiting, dizziness, shakiness, headache and anxiety. There have also been reports of accidental or inappropriate administration resulting in local effects, most commonly in a finger or a thumb. This case describes an accidental deployment of an Epipen® device in a pediatric patient resulting in a retained device and local tissue effects.

Case Report: A 5 yr-old boy was brought by EMS to an Emergency Department immediately (16:00) after he had accidentally deployed his Epipen® device into his lower leg. He had been playing with the device but did not think he was having an allergic reaction. Exam revealed T 37.1, P 91, RR 20, BP 100/63, Wt 23.7 kg. He was nervous but in no distress. He had no signs of allergic reaction including no facial or intraoral angioedema, no wheezing and no urticaria. His right lower extremity revealed an Epipen® device projecting from the anterior tibia with surrounding blanching of 3 cm. He reported decreased sensation in the surrounding tissue. The device was not extractable with initial manual attempts by hand. The needle was ultimately separated from the plastic attachment to prevent further discomfort during his evaluation. Radiographs demonstrated the needle in superficial soft tissue. Removal with hemostats and scissors was completed and revealed that the tip of the needle had become bent (likely from striking the bone) and then wrapped in fibrous material and entrapped. There was no significant bleeding or tissue compromise. He did not require further care and follow up phone call revealed no delayed effects and return of normal sensation.

Case Discussion: This case describes a pediatric accidental deployment of an Epipen® device that resulted in the needle becoming entrapped as a subcutaneous foreign body. There have been other reports of accidental or inappropriate injections with these products, typically into a thumb or finger when demonstrating use or when attempting to use appropriately. In most of these reported cases, symptoms and effects have not been serious and include temporary numbness or tingling with occasional elevated heart rate or palpitations.

Conclusions: Epipen® device needles can become bent and entrapped in soft tissue after accidental deployments near the tibia. Patient education should be aimed at minimizing these events with added attention to potential pediatric misadventures.

KEYWORDS Pediatric; accidental deployment; epinephrine autoinjector

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236. Mandating limits on acetaminophen in combination opioid products does not decrease incidence of hepatotoxicity

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Background: According to the National Poison Data System, 22,165 calls were placed in 2005 to Poison Control Centers (PCC) in regards to hydrocodone/acetaminophen exposures; over 7,000 of those exposures were unintentional in nature. Two of these cases resulted in fatalities. Using this data, a 2011 Federal Drug Administration (FDA) advisory committee recommended limiting the amount of acetaminophen in opioid-combination products to 325 mg per dosage unit. This mandate went into effect in early January 2014. According to an FDA statement, "by limiting the maximum amount. . .patients will be less likely to overdose on acetaminophen if they mistakenly take too many acetaminophen-containing products". We analyzed the PCC data to determine if the rate of hepatotoxicity decreased after the change in formulation.

Methods: Data from two PCC systems were analyzed. The databases were queried to search for unintentional hydrocodone/acetaminophen exposures. Exclusion criteria included anyone under the age of 13 or those identified as having pre-existing liver disease. Two separate yearlong periods were used; January 2013-December 2013 and April 2014- March 2015, assuming a three-month washout period to eliminate the remaining higher potency formulation tablets. The primary endpoint was defined as initiation of N-acetylcysteine (NAC) therapy, which was used as a surrogate marker for hepatotoxicity.

Results: 172 cases were identified in the pre-mandate year of which 11 (6.4%) received NAC therapy. In the post-mandate year there were 145 identified cases of which 5 (3.4%) received NAC therapy. There was no statistical significance (p-value $=0.23$) between the two time periods in the rate of initiation of NAC therapy. There was 1 case in each cohort where the clinical effect was listed as "major" and no deaths in either group.

Conclusions: There was no statistical difference in the need for NAC therapy as a result of the 2014 FDA mandate limiting acetaminophen in combination products, although a non-significant 3% absolute risk reduction was observed. Our study was limited by the small incidence of acetaminophen-induced hepatotoxicity. This study will be extended to increase its power by evaluating data from the entire National Poison Data System.

KEYWORDS acetaminophen; hepatotoxicity; opioid

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237. Duration of Emergency Department Phone Consults to the Poison Center and to the Inpatient Admitting Service

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Background: Poison Center (PC) consultation by emergency department physicians (EDPs) is voluntary. Busy EDPs may be less likely to call the PC if they believe the process will be more time-consuming than phone calls to other consultants, or that follow-up calls from the PC will place further demands on their time.

Methods: Audio recordings of consecutive phone calls to one PC from the parent institution's ED were retrospectively reviewed. Calls were included if they were made by an EDP (resident or attending) about a patient >18 years of age who required medical admission. Length of the initial call was determined. If the specialist in poison information (SPI) placed the EDP on hold while checking references or contacting the on call toxicologist, this time was included as part of the initial call. However, if the PC requested further information or made additional recommendations at a later time, this was considered a follow up call. Initial ED-PC call length was compared with length of initial calls from EDPs to internal medicine (IM) providers for patients requiring admission. The latter had been recorded for a previous ED study. The frequency with which follow up calls initiated by the PC were received by the EDP making the initial contact was also determined.

Results: The prior ED study found that for 180 calls to IM providers the median phone time was 2.11 minutes (IQR 1.62-2.68).

102 ED-PC calls were reviewed. Most (93.1%) were made by residents. ED-PC calls were longer (median 5.41 minutes, IQR 3.85-8.48, $p < 0.0001$) than ED-IM calls. Call time distribution was skewed for both data sets. Many of the longer ED-PC calls included time when the EDP was on hold while the SPI accessed information on specific substances or contacted the on call toxicologist. There was no significant difference in ED-PC call times between patients admitted to ICU and to floor beds. For 92% of the ED-PC interactions, follow up calls initiated by the PC were received by ED nurses or providers from the admitting team rather than the EDPs who initiated contact.
Conclusions: Initial EDP calls to PCs for adult patients requiring admission are significantly longer than those to IM admitting teams, but follow up calls made by the PC typically go to other caregivers. Although ED-PC calls take longer than those to IM, the PC must obtain sufficient information to provide treatment recommendations by phone, while IM providers can access test results directly and evaluate the patient in person.

KEYWORDS Poison Center; phone interaction; emergency department

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238. Increasing Abuse and Misuse of Loperamide

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Background: With the vast amount of information on the Internet, techniques for abuse and misuse of readily available medications are becoming rapidly disseminated resulting in several new "legal highs." A simple Internet search reveals numerous websites/forums that describe various ways to use loperamide for self-treatment of withdrawal syndromes or recreational use. The goal of this study was to determine if there has been an increase in loperamide misuse/abuse cases reported to a state poison system.
Methods: We conducted a retrospective review of loperamide cases called into a statewide poison system between 1/1/02 – 11/10/15. Cases were included for patients 18 years and over where reason for exposure was coded as intentional. Patients were only included if they presented or were referred to a healthcare facility.
Results: Between 2002 and 2013, the number of loperamide cases identified each year ranged between 12 and 20 cases; average per year- 16.75, median- 17. In 2014, there was a sharp increase to 40 cases. In 2015, 27 cases have been identified thus far. For those cases with known outcomes, 4 cases resulted in death, 74 had major effects, 48 had moderate effects, and 40 had minor effects. We identified 10 cases of cardiotoxicity with 8 of them occurring after October, 2014.
Conclusions: Loperamide is readily available for purchase over the counter and via the internet. We found a spike in the number of cases of loperamide toxicity reported in the past two years that coincides with many websites providing information on how to take this drug to get high or to help ameliorate the symptoms of opioid withdrawal. Almost all of our cases developing cardiotoxicity occurred in the last 13 months. Our data suggests that loperamide may be increasing in popularity as a drug of abuse/misuse. Given the potential for significant toxicity with these types of exposures, increased control over the availability of loperamide may be warranted.

KEYWORDS Loperamide; Drug abuse; Dysrhythmias

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239. Rising Cost of Antidotes: Cost Comparison 2010 to 2015

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Background: The price of several drugs in the United States' market has increased significantly over the past few years. There are limited data that focus on price escalations on commonly used antidotes. The objective was to describe and quantify the price changes of antidotes over the past five years.
Methods: Average wholesale price (AWP) of antidotes were identified from 2010 and 2015 using Red Book Online® drug resource. Minimum stocking requirements of antidotes were determined using previously published stocking recommendations. Stocking recommendations were used to calculate the costs of stocking the minimum amount of each antidote. The average drug price of the minimum stocking requirement in 2010 was compared to that of 2015, using the same stocking recommendations for each antidote. All prices from 2010 were adjusted to compensate for inflation using the Consumer Price Index (CPI). All figures are in US dollars.
Results: Many of the antidotes incurred a substantial increase in average wholesale price from 2010 to 2015. Edetate calcium disodium had the largest price increase of 5,000%, followed by methylene blue (1,786%), digoxin immune fab (319%), and calcium gluconate (275%). The relative price increase was over 50% in 11 out of 27 antidotes and the absolute increase was greater than $1000 in seven of the antidotes. Only eight of the antidotes had a decrease in average wholesale price and those were mostly minimal decreases.
Conclusions: There has been a large increase in antidote costs over the past five years. Due to high cost and low use, many health systems may become reluctant to routinely stock necessary antidotes, especially at the recommended stocking levels. This promotes concerns regarding the timely access for antidotal therapy and potential impact on patient safety. Poison Centers may play an important role in helping healthcare providers locate regional supplies of antidotes. Antidote repositories may also need to be considered. In addition, costs of therapy may need become a larger factor in making treatment decisions supported by weak evidence.
Conclusions: Cost reduction strategies must be developed to ameliorate the untoward effects of the significant increase in antidote pricing and to ensure the proper use and proper access of antidotes for patients.

KEYWORDS Antidote; cost; therapy

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240. Review of temporal trends of multiple Poison Centers' HCF management patterns of children less than 6 years old

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Background: Poison centers (PC) in the US operate independently and determine management of patients based on the individual center's clinical and medical direction. While all centers utilize similar resources and communicate frequently, it is unknown how much, if any, practice patterns vary.
Methods: Retrospective evaluation of temporal trends of the number and percentage of human exposures (HE) in children <6 years old reported to 10 regional PC and one state PC system; the total group covered 12 states. We evaluated annual volumes/percentages for 2010 to 2015, for percentage of HE managed in non-health Care facility (non-HCF) and Health Care Facility (HCF) settings. Those managed in HCF were separated into 1) referred to HCF by a PC and 2) already in/enroute to HCF when the PC was contacted. Additionally we compared HE with serious outcomes (moderate, major and death) in HE referred to HCF by a PC and HE already in/enroute to HCF.
Results: The majority of children were safely managed at a non-HCF (>88%). There was a small but linear increase in HE managed in a HCF in 6 of the 11 centers ($R2 > 0.88$), with a mean increase in the group of 12% (range: 11.1% - 12.4%) over the 6 years. One center decreased HE managed in a HCF over this time period. Percentage of HE referred to HCF by a PC showed no significant change over time in 8 centers and a decrease in 3; with a group mean of 4.4% and range 3% to 6%. One center showed a linear decrease in HE referred to HCF by a PC from 10 to 6% over 6 years, without a change in serious outcomes. Serious outcomes remained uncommon, but increased slightly from 0.78% to 1.0%. This increase in serious outcomes occurred in HE already in a HCF. This increase was influenced by an increase >30% from four centers and was predominantly caused by an increase in moderate outcomes. Serious outcomes in HE referred to a HCF by a PC showed no change over time. (mean 0.28%, range 0.1 to 0.5%)
Conclusions: There was remarkable consistency between centers across multiple regions of the US. The small increase in HE managed in a HCF appears to be influenced by HE already in the HCF prior to PC contact. A number of questions merit further evaluation, including: can the increase be attributed to a subset of substance groups, and what percentage of "non-toxic" cases managed in a HCF were self-referred and might have benefited from PC management advice to save a HCF visit. One center's evaluation of their individual performance reduced their percentage of referral to a HCF without change in serious outcomes.A small increase in HE managed in an HCF as reported to PC was seen over 6 years in 12 states. The percentage of HE referred to a HCF by PC did not change over time. Serious outcomes were rare and increased only in HE already in a HCF with moderate outcomes.

KEYWORDS HCF management; practice variance; trends in outcome

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241. Retrospective Review of Pediatric Cetirizine Ingestions Reported to a Poison Center Network and Formulation of Evidence-Based Thresholds for Pre-Hospital Triage

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Background: Antihistamines are a commonly used class of medications due to over-the-counter (OTC) availability in multiple formulations and routes of administration. Cetirizine is a second generation antihistamine available OTC. Currently, the American Association of Poison Control Centers does not have a position statement, specific recommendations, or pre-hospital triage criteria for management of cetirizine ingestions. Reviews of safety and/or specific patient outcomes resulting from cetirizine ingestions involving significant numbers of patients are lacking. One yearlong review of five regional poison centers' data reported only 3.4% of ingestions had 'moderate clinical effects', with tachycardia being the most severe symptom. All other effects were identified as 'minor' or no effects were reported. The mean dose per ingestion was 43.4 mg, approximately four times the maximum recommended daily adult dose. In line with the authors' findings, this poison center uses four times the maximum recommended daily dose for age as a pre-hospital triage criterion to determine if a cetirizine ingestion requires medical evaluation, which may be too conservative based on the pharmacokinetic and pharmacodynamic properties of cetirizine. The purpose of this study was to develop an evidence-based dose threshold for pre-hospital triage of single-substance cetirizine ingestions in children ≤5 years.
Methods: All single substance cetirizine exposures reported to a poison center network occurring in children ≤5 years were identified. Exposures were excluded if any of the following criteria were met: outcome lost to follow up, reason for exposure attributed to adverse drug reaction, 'taste/lick/drop' code applied to amount of ingestion, or if the exposure was not an ingestion. Data collected include: demographics, reason for and amount of ingestion, clinical effects and duration, type of toxicity, medical outcome, caller and management site, and hospital length of stay.
Results: A total of 3825 exposures were reviewed and 2970 were included for analysis, 1659 (55.86%) of which were males. Ages ranged from 10 days to 5 years, with a mean age of 2.55 ± 1.16 years. Amount ingested (mg) ranged from 0.4 to 320 with a mean of 19.74 ± 26.97. Amount ingested (mg/kg) ranged from 0.03 to 30.61, with a mean amount of 1.49 ± 2.27. Of the exposures followed to known outcome, 1096 (86.64%) experienced no effects, 165 (13.04) experienced minor effects, and 4 (0.32%) experienced moderate effects. Drowsiness was the most commonly reported adverse effect, with 125 instances (4.21% of exposures). Other adverse events reported include: agitation (n = 66 instances, 2.22% of exposures), vomiting (n = 13, 0.44%), tachycardia (n = 7, 0.24%), and erythema or flushing (n = 5, 0.17%). In addition, there were 3 exposures causing hypertension or mydriasis, 2 exposures causing cough, and 1 instance of each of the following: fever, abdominal pain, diarrhea, constipation, and/or premature ventricular contractions.

Conclusions: Cetirizine is a very common exposure, but rarely results in clinical manifestations. When symptoms arise, drowsiness is the most common. Only 0.13% of included exposures experienced a moderate adverse effect from cetirizine. No patients experienced major symptoms and no deaths were reported.

KEYWORDS Cetirizine; overdose; pediatric

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242. NPDS Comparison of Novel Oral Anticoagulants and Warfarin exposures in a Regional Poison Center

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Background: Poison centers across the country have seen a rise in calls involving novel oral anticoagulants (NOAC) due to their expanded use as an alternative to warfarin therapy. The advantages of NOACs are convenient, standardized dosing and lack of routine monitoring. The lack of monitoring is the precise disadvantage of NOACs in the setting of adverse reaction or overdose. Currently they are used for a variety of conditions including the treatment and prevention of deep venous thrombosis (DVT) and pulmonary embolism (PE), to reduce risk of stroke and systemic embolism in patients with nonvalvular atrial fibrillation, and for the reduction in the risk of recurrence of DVT and of PE. Dabigatran was the first approved NOAC in the US (2010), followed by rivaroxaban (2011), apixaban (2013), and edoxaban (2015). We sought to characterize the trend of NOAC cases in our 5-state regional poison center (RPC) and also compare with warfarin case trends. Methods: 15 NPDS Product Codes involving dabigatran, Pradaxa®, Eliquis®, rivaroxaban, Xarelto®, edoxaban, Savaysa®, and warfarin were queried from 2006 through 2015. The same reports for both NOAC and warfarin were duplicated for single-substance case analysis. Descriptive statistical analyses were performed.
Results: Not surprisingly, the trend of NOAC and warfarin calls were inversely proportional. Warfarin calls peaked in 2011 followed by a steady decline to the present; whereas NOAC calls first showed up in 2011 with a bump in 2012 and dipped slightly in 2013 before rising significantly the next 2 years and continues to rise (see Table). In fact, by 2015, there were more NOAC calls (n = 60) than warfarin calls (n = 58). From 2011-2015, our RPC reported a total of 161 exposures involving single-substance NOAC ingestion. 55% were female. Most common reason for ingestion was Unintentional Therapeutic Error (68%), followed by Unintentional General (5%) and Intentional Suspected Suicide (4.3%). Most were managed on site (63%). Of the 39 patients (24%) seen in a health care facility, 26 (67%) were treated, evaluated, and released from the Emergency Department. 6 (15%) were admitted to a critical care unit. Outcomes: there was 1 death that involved dabigatran (adverse drug reaction). 66% had no effect, 10% had minor effects, 5% had moderate effects. There were no major effects. Most common clinical effect was PT prolongation (n = 6), then headache (n = 4), and ecchymosis and "other coagulopathy" (n = 3 for both). Most treatment was symptomatic and supportive; 2 patients required prothrombin complex concentrates (PCC).
Conclusions: The rise in popularity of alternative agents to warfarin is apparent within our 5-state RPC region and we suspect the trend will likely continue to rise. NOAC exposures overtook warfarin exposures in 2015. The increased prescribing of NOACs in the general population means that poison centers will receive more calls on these drugs. While there are established guidelines for managing warfarin toxicity, there are not yet standardized guidelines for treating NOAC toxicity. Future studies are needed in this area to better characterize and manage NOAC toxicity.

KEYWORDS Novel anticoagulants; warfarin; poisoning

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243. Use of the Clinical Health Information Exchange (cHIE) In Preparing Fatality Abstracts for National Data Submission

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Background: US poison centers are required to submit structured, annual fatality abstracts to the National Poison Data System (NPDS) for all reported cases ending in a fatality. Each abstract contains de-identified patient information including past medical history (PMH), home medications, clinical course, laboratory values, therapies received, and a medical examiners report when available. Poison centers do not always have access to all the requested information for a fatality abstract. The cHIE is a repository of secured patient health information accessible to healthcare professionals throughout the state. The purpose of this project is to describe the information collected from the cHIE used to supplement the NPDS fatality abstracts.
Methods: All fatalities reported to one regional poison center during 2015 were identified. For each fatality, the cHIE was searched for additional medical information to supplement the existing poison center medical record. Date of birth (DOB) is necessary to access the cHIE.
Results: There were 22 fatalities identified in 2015. We were unable to search the cHIE in 6 patients (27%) because the poison center record lacked DOB. One patient (5%) had DOB recorded, but could not be located in the cHIE. Three patients (14%) were located in the cHIE without any records available for review. Twelve patients (55%) had supplemental information that was added to the fatality abstract. Of the 12 cases that provided additional information, 8 cases (67%) provided PMH including home medications, while 6 cases (50%) provided information on clinical course including labs, therapies, and time of death. In one case, information collected from the cHIE may have changed management recommendations provided by the poison center.
Conclusions: Information on PMH and clinical course were provided by the cHIE in 67% and 50% of cases, respectively. Electronic health repositories of patient health information, like

	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015
NOAC single substance exposures	0	0	0	0	0	19	23	19	40	60
NOAC total exposures	0	0	0	0	0	22	33	25	52	93
Warfarin single substance exposures	80	78	107	101	101	131	80	62	62	58
Warfarin total exposures	120	130	153	156	154	198	123	124	114	109

the cHIE, can provide useful information in supplementing the fatality abstracts. Further studies are needed to identify the utility of the cHIE in other areas of poison center workflow.

KEYWORDS fatality; cHIE; NPDS

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244. Spectrum of acute accidental poisoning in children and adolescents: a community based survey of urban slums of Ujjain, India

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Background: According to World Health Organization poisoning is one of the most important public health threats. Most information on poisoning is based on hospital or poisoning center based reporting. In view of paucity of community based studies from India we did this study to determine prevalence and pattern of acute accidental poisoning in children and adolescents up to 18 year of age.
Method: This cross-sectional study was conducted from December 2015 to February 2016 in two contagious slums in Ujjain city in Central India, having a total of 448 households out of which 326 households were identified having children in desired age range. The heads of the family of all 326 households were approached; informed consent obtained and asked to recall any episodes of poisoning in the past one year. The data was obtained using pretested questionnaire, administered by trained research assistants. Questions related to poisoning and its symptoms, its route, first-aid at scene of poisoning and community knowledge regarding first-aid required for the poisoning were administered.
Results: In 326 households 826 children and adolescents were eligible to participate. Information was obtained for 800 children. A total of 16 children of poisoning were identified giving a prevalence of 2% (16/800) with mean age of 2 years. Both sexes were equally affected. There were no reported hospital admissions or deaths due to poisoning. Commonest route of poisoning was ingestion (n = 14; 87%) followed by skin contact and animal bite each (n = 1; 6%). Commonest type of poison ingested were detergent based cleaning products like soap and washing powder (n = 10;66%), followed by kerosene (n = 2; 13%) and left over medicines at home (n = 1; 6%). Most common symptoms reported were vomiting (n = 10) and increased salivation (n = 3). Very few (n = 3; 19%) children with poisoning received any first-aid at household. Most (n = 15;93%) of the family heads reported not having any knowledge of first-aid. Care was sought for most children with poisoning (n = 10;63%), despite children being clinically stable with 35% children taken to local traditional healers or informal health-care providers for treatment. The most common reason for not seeking any medical care was the perceived "non-serious" nature of poisoning.
Conclusions: Community based survey for poisoning gives different information compared to hospital based poisoning centers and could also provide important clues to community perception about poisoning and its first aid. In view of poor knowledge for first aid after poisoning, a household intervention to provide anticipatory guidelines to the parents could play a major role in prevention and improved poisoning management.

KEYWORDS Poisoning; Urban Slum; Children

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245. Sticky Situations: Cyanoacrylate Exposures Reported to a Poison Control System

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Introduction: Exposures to cyanoacrylate-containing products (such as Super Glue® and Krazy Glue®) are commonly reported to poison control centers, but little has been published in the medical literature regarding these exposures. We sought to characterize cyanoacrylate exposures reported to a poison control system.
Methods: We performed a retrospective review of a poison system's database for all cases of single-substance human exposure to cyanoacrylate-containing products from 2005-2015. Data collected included age, gender, route of exposure, clinical effects, treatments recommended and administered, and medical outcome.
Results: There were a total of 893 patients, 505 (56.6%) of which were female. Patient ages ranged from 6 months to 88 years with a mean of 20.2 years and median of 11 years. The vast majority of exposures (n = 871, 97.5%) were unintentional, but a small number of exposures (n = 22, 2.5%) were due to intentional misuse (such as trying to stop a bleeding cut) or malicious intent (such as purposefully gluing a person's eyes shut as a prank). Routes of exposure included: ingestion, n = 337 (37.7%); ocular, n = 322 (36.1%); dermatologic, n = 285 (31.9%); inhalation, n = 16 (1.8%); nasal, n = 1 (0.1%); otic, n = 1 (0.1%); some patients had multiple routes of exposure. Treatments recommended by the poison center included irrigation (n = 411), petroleum jelly (n = 143), mineral oil (n = 131), topical antibiotic ointment (n = 82), peanut butter (n = 6), and WD-40® (n = 2). A total of 657 patients (73.6%) were managed on-site, while 236 (26.4%) were seen in a health care facility. Among all exposures, effects were classified as none (n = 287), minor (n = 529), and moderate (n = 77). No major effects or deaths were reported.
Conclusions: In this case series, the majority of cases occurred in children and most exposures did not result in significant morbidity. Notably, there was wide variation in terms of recommended treatments; further study is needed to determine the optimal treatment method and to standardize poison center recommendations for treating patients with cyanoacrylate exposures.

KEYWORDS Cyanoacrylate; poison center; superglue

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246. 100% Participation in a New Hospital-Cost-Sharing Program

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Background: Hospital cost-sharing programs are a growing trend and over a dozen poison control centers (PCC) across the country have this type of program. It has become almost standard of care for hospital-based health care practitioners to consult with our PCC in order to provide optimal care for their poisoned and

overdosed patients. For many years our PCC explored the concept of partnering with the hospitals throughout our state to help fund the PCC, and the increased hospital utilization of PCC services prompted us to initiate a hospital cost-sharing program.
Methods: First we addressed several hospital CEOs or their designee at a meeting of our state hospital association. We provided information about our PCC and its value to the overall health care system, and described our proposed hospital subscription program. The voluntary hospital assessment was designed with a 4-tiered billing structure based on the volume of emergency department visits. Hospitals that opted out of the annual subscription plan would incur a $200 fee per call and billed quarterly. There was no extra incentive for joining and no repercussions for not joining. With the help of hospital executives and clinicians from our affiliated health systems, we mailed the initial letter to all hospitals in November of 2012 with an effective date of CY 2013. Hospitals that did not initially pay the subscription or pay per-call received a follow-up letter, a personal call, and continued to receive an invoice for our service. The hospital executives and clinicians from our affiliated health systems also played a key role in encouraging hospitals to join the program by discussing the matter with their peers. The PCC simultaneously worked on leveraging additional funding from both public and private sources.
Results: Many hospitals initially requested PCC data for the number of calls their hospital made to the PCC. This was for a cost evaluation based on the annual subscription or fee per call. By the end of year two (2014), only 4 hospitals had not joined the subscription. With continued encouragement to join the effort, every hospital in the state voluntarily contributed and joined the program by the end of year three (2015).
Conclusions: Careful planning and communication is needed when implementing a new hospital cost-sharing program given that hospitals would be charged for a service that was formerly free. Our PCC had strong support from hospital leadership as well as the state legislature to reach 100% hospital participation.

KEYWORDS Hospital Cost Sharing Program; Subscription Program; Poison Center Hospital Subscription

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247. Keep Calm and Cryo-On

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Background: Cryotherapy involves exposing the human body to nitrogen at subzero temperatures (as low as -250 F) for up to several minutes. Post nitrogen cryotherapy outcomes postulated include blood vessel expansion that promotes healing and provides pain relief. Whole body cryotherapy treatments originated in Japan in the 1970s. Today, it is increasingly popular among elite athletes, but also used by the average spa- goer to treat a wide variety of ailments including rheumatoid arthritis, migraines, insomnia, and joint pain.
Case Report: A 45-year-old cryotherapy spa owner stepped into one of his cryotherapy chambers early in the morning without the benefit of an observing technician. His wife and coworker heard a thud, and they found him unresponsive in the chamber. Emergency medical services (EMS) were called, and upon arrival found the patient in cardiac arrest. The patient was in ventricular fibrillation and he was given 2 rounds of defibrillation before he had return of spontaneous circulation. He arrived to the emergency department (ED) with a Glasgow coma scale of 3. The finding of bilateral pulmonary edema resulted in the patient being emergently intubated. His initial lab work showed no abnormalities. A methemoglobin was obtained and resulted as 0.4%. He was admitted to the intensive care unit and remained for 21 days on a ventilator and on antibiotic therapy. The hospital stay was additionally complicated by acidosis, hypothermia, hypertension, tachycardia, and pneumonia. The patient was eventually discharged to a rehab facility with persistent short-term memory loss and permanent left side weakness.
Case Discussion: Nitrogen acts as an asphyxiant- it is unclear if our patient suffered from asphyxiation from dipping his head too low into the chamber, hypotension followed by a syncopal episode, or something else. The patient was known to be noncompliant with his antihypertensive medication. A cryochamber manufacturer, consulted for discussion of any prior incidents, revealed that the institution of an oxygen sensor by the level of the mouth mitigated cases of asphyxiation reported in the 1980s. Several cases of increased parasympathetic tone by an unknown mechanism in patients with prior cardiac history have been reported. The exact cause of our patient's symptoms remain a mystery, but we suspect that our patient had an asymptomatic dysrhythmic episode prior to cryochamber entry, and the nitrogen exacerbated his cardiac rhythm, putting him in arrest. One previous death has been reported in a cryotherapy chamber worker.
Conclusions: This patient had a past medical history of hypertension, a condition considered a contraindication for use of nitrogen cryotherapy. Health care professionals should be aware that patients with a history of heart conditions should use extreme caution in cryotherapy chambers.

KEYWORDS Cryochamber; liquid nitrogen; cryotherapy

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248. The prevalence of fatal adolescent poisonings not reported to an urban poison control center and the comparison of reported versus unreported cases

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Background: Poison control centers (PCCs) provide expert consultation and collect epidemiologic data on toxic exposures, which aid in primary prevention. In some states, local health code laws require poisonings to be reported which can improve patient care and aid in toxicosurveillance. However, multiple studies demonstrate that poisonings are vastly underreported to PCCs. Possible reasons for underreporting fatalities include death occurring prior to reaching health care or poisoning was not suspected until time of autopsy. This retrospective study was designed to determine the prevalence of fatal adolescent poisoning cases that go unreported to a large urban PCC serving a population of over 12 million people.
Methods: All recorded deaths that occurred in this large urban area containing the ICD-10 codes pertaining to poisoning including; X60-84, T36-65 from 2000 to 2012 were requested from the Office of Vital Statistics (OVS) via the regional medical examiner's office. From these cases, all deaths ages 10-18 were extracted. Within this age group, poison-related deaths were identified using poison codes above and further divided into two groups: deaths that occurred in the home and deaths that occurred at a location outside of the home or at a medical facility. Toxicall was then used to obtain all adolescent poisoning deaths recorded by the

regional PCC during the same time period. PCC deaths were matched to OVS deaths by using name, date, and age to accurately identify the cases that were reported to the regional PCC.
Results: A total of 18,788 cases were initially extracted from the OVS, 374 of which were between the ages 10-18. Of these, 157 were identified as poison-related deaths. Fifty-two of the 157 died at home, while 105 died at either a medical facility or a location outside the home. Of these 105 adolescent poisoning deaths, only 17 (16%) were also found in the Toxicall database. When dead on arrival (DOA) cases were removed from analysis, only 18% of deaths were reported to the PCC. Review of each of the 17 cases reported to the PCC suggested the causes of death to be opioids in 5; acetaminophen in 4; psychiatric medications in 3, salicylate in 2 and unknown/unclear in 2.
Conclusions: These findings reinforce gross underreporting of fatal poisoning cases to PCCs. Only 18% of fatal adolescent poisonings over a 13-year period were reported to the regional PCC. Failure to report such poisonings may have a negative effect not only on individual clinical outcomes, but also on the accuracy of epidemiologic data and thus, on the long-term goal of primary prevention. Future studies should attempt to identify barriers to reporting.

KEYWORDS Poison control center; Adolescent Deaths/Poisonings; Reporting

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249. Analysis of patients presenting to a medical toxicology clinic with a focus on self or poison center referrals

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Background: There are a limited number of outpatient medical toxicology clinics (MTC) in the US, and their collaborations with local poison centers (PC) are not well documented. Data from the Toxicology Investigators Consortium (ToxIC) was used to identify patients that accessed a stand-alone MTC in the Midwestern US via routes other than physician referral, and to characterize this patient population.
Methods: All cases reported to the ToxIC Registry from our MTC from July 1, 2010 to January 27, 2016 were retrospectively analyzed. Those referred by a physician were excluded from analysis. The source of referral was identified, and basic demographics, exposure source, reason for referral and interventions performed were further investigated for PC or self referral.
Results: A total of 1048 patients were seen in the clinic during this 6.5 year period. 634 of them were referred by a physician (60%). 164 patients (16%) were referred by local PCs and 202 patients (19%) were self referred. Of the 366 non-physician referrals, 348 (95%) involved adults (including 6 pregnant women); 18 involved children under 12 years old. Approximately 60% of the (self or PC) referrals involved occupational/environmental exposure evaluations (see Table). Neurological symptoms (n = 57) and rash (n = 39) were the most common presenting complaints among self/PC referrals. Suspected substances in the self or PC patient referrals were mercury (22), other metals (43), carbon monoxide (22), other gas inhalation (39), mold (41), and pesticide (19). Specific toxicological based intervention occurred in only 4 patients (1.1%): 2 received succimer for metal toxicity, 1 received a course of steroids, and 1 received diphenhydramine.
Conclusions: A vast majority (approximately 70%) of self- or PC-referred patients either involved suspected environmental/occupational exposures or required interpretation of toxicology based laboratory data. Of the symptomatic patients, neurological or dermatological complaints were the most common. Specific toxicological intervention occurred in only 1.1% of these patients. PCs account for 16% of referrals to a stand alone, Midwestern MTC. Medical based toxicological interventions were rarely necessary for patients regardless of method of referral (self or PC).

	Environmental or Occupational exposure	Interpretation of Lab Data	Organ System Dysfunction	Other
Self Referral n = 202	127 (63%)	29 (14%)	23(11%)	23 (12%)
PC Referral n = 164	95 (58%)	14 (9%)	13 (8%)	42 (25%)
Total n = 366	222 (60%)	43 (12%)	36 (9%)	62 (19%)

KEYWORDS Outpatient; clinic; referrals

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250. Proton Pump Inhibitors in Children: A Poison Center Experience

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Background: Proton Pump Inhibitors (PPIs) are a commonly used class of medication to decrease gastric acid secretion but information regarding toxic exposures and symptoms in the pediatric population has rarely been described. The aim of this study is to report one regional poison center's (RPC) experience with PPI exposures in the pediatric population.
Methods: Electronic RPC records coded for PPIs in patients 5 years of age and younger from January 1, 2003 to December 31, 2015 were retrospectively reviewed. Information collected included age, weight, medication, route of exposure, symptoms, and outcome. Cases were excluded if they involved multiple agents.
Results: There were 1,196 cases of isolated PPI exposures in children aged 5 years or less reported to the RPC during the 13-year study period. Of these, 30 reported symptoms. Gastrointestinal symptoms included emesis (10 patients), diarrhea (2 patients), and decreased appetite (1 patient). Neurologic symptoms included drowsiness (8 patients), hyperactivity (1 patient), and headache (1 patient). A total of 5 patients had "eye redness", all related to ocular exposures with liquid preparations in infants less than 7 months of age. All symptoms resolved on follow-up and no toxicologic based interventions were noted. The parents of one 2-month old child inquired about the association of elevated hepatic transaminase levels with chronic use of lansoprazole but no actual transaminase values were available. The most commonly reported agents were lansoprazole (466 exposures; 39% of total), omeprazole (338; 28%), and esomeprazole (253; 21%). There were no hospitalizations, major outcomes, or fatalities during the study period.
Conclusions: This study indicates the relatively benign effect of PPI's in pediatric overdoses. In the nearly 1200 cases of pediatric overdoses of PPI's, there were no reported significant sequelae or fatalities in children under the age of 5 years. In virtually all of these cases, the patients were asymptomatic on six-hour follow-up after ingestions up to 60 mg/kg. Pediatric PPI overdose cases can be managed safely at home without requiring referral to a healthcare facility. This study (which is one of the first case series

of pediatric PPI overdoses) demonstrates the relatively benign course of pediatric PPI overdose.

KEYWORDS Proton Pump Inhibitors; Pediatrics; Poison Center

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251. Let's give them something to CHAT about

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Background: Oxford's Dictionary defines a call center as, "An office staffed and equipped to handle large numbers of telephone calls, using computer technology to assist in the management of calls and supply of information." Poison Control Centers (PC) have engaged in call center activities for nearly half a century. In 2010, our PC became the first in the nation to launch real-time online CHAT, allowing the general public to securely contact us directly through our website. Our telecom system routes chats much the same way phone calls are routed through our ACD. We describe here our experience to date with online CHAT.
Methods: Introduced 6 yrs ago, online CHAT became another way individuals were able to reach our PC's services. While CHAT interactions represent a small portion of our overall exposure call volume, they provide today's millennials a communications method to which they are more accustomed. To showcase our experience with CHAT, we compared PC exposure data from phone calls along with CHAT conversations for July 2010-December 2015. Data fields compared included Call Types, Caller/Management sites, Age, Reason, Substances, Medical Outcomes, and Caller location.
Results: From July 2010-December 2015, 461,917 exposure calls were documented by our PC. Of those, 2,391 were completed from our CHAT interface. The top 3 Caller Sites recorded from calls were Own Residence followed by Health Care Facilities, then by Schools. For CHAT, Own Residence, UNKNOWN, then Schools rounded out the top 3 sites. Health care providers made 36 CHAT contacts. The top 5 Substances reported by phone and by CHAT closely mirrored one another, with Analgesics ranking first, followed by Household Cleaning agents, then Cosmetics/Personal Care products; Sedatives/Hypnotics/Antipsychotics and Bites/Envenomations were 4th and 5th respectively from phone calls, while Pesticides on the other hand followed by Antihistamines were 4th and 5th in chat frequency. The top 3 Age Groups involved in calls were Birth-5 yrs, followed by 20-29 yrs, then 30-39 yrs; chat patients were Birth-5 yrs, 20-29 yrs, and Unknown aged adults. Being accessible via the web opened up a new group of consumers to our PC services. Approximately 72% of CHAT calls were from our service area, while 94% of calls were from our State.
Conclusions: Nearly 6 yrs of CHAT interactions are described. Making available a technology that has been used successfully as a means of communication for over 20 years is seriously overdue. The traditional model of the voice-only call center of yesterday, under which a majority of PCs still operate, is in need of an overhaul. To meet the demands of today's caller, we must upgrade to reach the whole and remain relevant.

KEYWORDS CHAT; Communication; Tecnhology

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252. Characterizing exposures to ionizing radiation reported to poison centers, 2012-2015

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Background: Since 2010, the Centers for Disease Control and Prevention (CDC) and the American Association of Poison Control Centers (AAPCC) have conducted surveillance on poison center (PC) data uploaded to the National Poison Data System (NPDS) for radiological exposures such as ionizing radiation. In 2011, the coding options for PC staff to classify the different types of ionizing radiation were greatly expanded. This enabled greater differentiation and classification of ionizing radiation exposures such as the type (e.g., alpha, beta, and gamma radiation), the specific non-pharmaceutical radionuclide source (e.g., uranium, cesium-137), and if a radiological weapon (e.g., radiological exposure device) was used. This nomenclature expansion was accompanied by multiple in-person and online trainings for PC staff on how to use the new codes. Our objective is to assess whether PCs are using these new codes and characterize ionizing radiation-related exposures reported to PCs since their introduction.
Methods: We analyzed all calls reporting any exposure to ionizing radiation from January 1, 2012 to December 31, 2015. We excluded reported exposures to "radon", "radon gas", "non-ionizing radiation", and "smoke detectors" along with calls about requests for information (no exposure) and confirmed non-exposures (e.g., exercises). We calculated the median age and range of callers, and analyzed exposures by sex and medical outcome as defined by AAPCC (death, major effect, moderate effect, minor effect, and no effect) and exposure type. We tabulated the three most frequently reported ionizing radiation codes. Descriptive analysis was performed using SAS 9.3.
Results: We identified 565 ionizing radiation exposures called to PCs during the study period. Most exposures were in males (n = 319, 56.5%). Age was recorded in the majority of them (n = 430; 76.1%) and the median reported age was 36 years (range, <1 – 85 years). Of those exposures with outcome data available (n = 137, 24.2%), the majority reported no effect (n = 87, 63.5%), followed by minor effect (n = 33, 24.1%) and moderate effect (n = 15, 10.9%). Two reported exposures were associated with a major effect; one of these was associated with multiple exposures. A specific radionuclide was reported in more than one-third of exposures (n = 222, 39.3%) followed by radiation type (n = 81, 14.3%). The top three reported exposure substances included "unknown type of ionizing radiation" (n = 266, 47.0%), X-ray radiation (n = 60, 10.6%), and Uranium-238 (n = 59, 10.4%).
Conclusions: PC staff are using the expanded nomenclature to better characterize reported exposures to radiation. This study provides evidence that expansion of coding paired with appropriate training opportunities for PC staff can provide more specificity of certain reported exposures of public health concern and can improve national surveillance activities.

KEYWORDS Radiation; Public Health; Surveillance

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253. Blooming Trouble: Toxic Flower Cakes

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Background: Humans, livestock and honeybees can all be poisoned by Yellow/Carolina Jessamine (Gelsemium sempervirens), an evergreen vine that produces tubular flowers in the spring. The plant is popular in southern United States (US) gardens because it produces showy fragrant blossoms, is resistant to pests and is not considered an invasive species. We report on a cluster of cases in a family newly arrived in the US who picked the flowers and consumed them.
Case Report: A Chinese family of three, two of whom recently arrived to the US misidentified Caroline Jessamine flowers as a benign edible flower commonly used in tea and cakes in Shandong Province, China. Only the 29-year-old mother spoke limited English. An attending physician, who speaks Mandarin, interviewed the grandmother who reported that 4-5 flowers and some leaves were used in the production of cakes that the family consumed. Within 1 hour all the family members began feeling ill with dizziness, lightheadedness and weakness and all three were transported to a nearby emergency department along with a sample of the flowers ingested. A consultant botanist identified the flowers as Gelsemium sempervirens. The 57-year-old grandmother was the first to arrive and demonstrated hoarse voice, ptosis, and difficulty speaking and breathing but was moving all her extremities. Initial oxygen saturation was 100%. Epinephrine, steroids and diphenhydramine were administered for possible anaphylactic reaction. The grandmother suddenly became apneic, hypoxic and required intubation. No airway edema or swelling was present. Post intubation ECG was concerning for inferior STEMI and the patient underwent urgent cardiac catheterization without evidence of obstructive coronary disease. Her 5- year-old granddaughter also arrived with normal oxygen saturations, moving all extremities and had non-fatigueable horizontal nystagmus, but developed weakness and air hunger and required intubation. The 29-year-old mother developed bulbar muscle weakness but didn't require intubation. All the patients did well and both were extubated within 48 hours. The grandmother continued to have ptosis and diplopia following extubation. Both resolved within 72 hours of exposure.
Case Discussion: Cases of botanical misidentification by new immigrants are not unusual. In the US, gardeners are well acquainted with the toxic nature of the plant, which produces gelsemine and other alkaloids that agonize glycine. Intoxication symptoms include muscular weakness, respiratory depression and paralysis of motor nerve ends. Care is supportive; patients generally survive with appropriate ventilation. Interestingly strychnine, a glycine antagonist, is produced by a plant in the same family as Yellow Jessamine.
Conclusions: Information for new immigrants should include warnings that US plants may not be the same as those in their homelands and consumption of such plants may be ill advised.

KEYWORDS Plant toxin; immigrants; critical care

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254. Getting Our Fair Share: An Innovative Approach to Poison Center Funding

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Background: Funding for Poison Center (PC) operations continues to be a major challenge across our nation. For nonprofit PCs such as the Washington Poison Center (WAPC), the challenge includes greater competition for fewer governmental and foundational funds. In an effort to create a more reliable and sustainable source of operation funding, WAPC undertook research to interview other PCs who receive community benefit funds from the hospitals in their service regions. Our intent was to discover a set of best practices we could use to create a more successful Hospital Fair Share program to help fund the WAPC. Prior to this effort, WAPC annually ran a statewide campaign asking for the support of 98 acute care hospitals. The campaign faced many barriers: public district hospitals who indicated they were barred from giving through their charters, nonprofit hospitals using their status to refuse support, decision-makers who were far removed from the Emergency Departments who utilized our services and refused to see the value of support, and state laws that barred WAPC from refusing service or charging a fee. As a result, support was inconsistent and unreliable over the past 16 years ranging from a low of $9,750 to $82,000 and thus difficult to plan on as a funding stream.
Method: WAPC staff developed a set of questions to survey nine poison centers with successful fair share type programs to identify best practices. The outcomes fell into natural categories of strategies to use with hospitals, fee structures/billing, Hospital Associations, legislation, and other opportunities. These results helped define the approach used by WAPC and included an implementation timeline, a cost structure and algorithm that remained fair to participants, and a suite of customized documents for hospital staff that included data to back up our value. Concurrently, WAPC developed a new brand suite for this campaign to set it apart from previous efforts that didn't have a defined structure. The program was launched in phases defined by hospital size so the materials could be adjusted if necessary. Hospitals received a kick-off letter announcing the upcoming formal campaign and explained the new program, the invoice process, and emphasized our high expectations for support. The letter also included a "whitepaper", which explained in detail the structure and justification of the Fair Share program; we feel this was pivotal to our success.
Results: There was a dramatic increase of 247% in funding from previous years, netting $277,340 from 57% of hospitals in Washington State.
Conclusions: Two months into the program, it was evident that the Fair Share program was a success.

KEYWORDS Funding; Management; Fundraising; Poison Control Center

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255. The Changing Face of Poison Center Call Metrics over Ten Years

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Background: For decades a primary value of poison centers (PC) was measured through triage of unintentional pediatric poisonings, and prevention of unnecessary hospital care. In recent years residential calls to PCs have decreased, and poison specialists are increasingly managing older and complex hospitalized patients. This report compares call data from a regional poison center (RPC) over a 10 year period, and the potential impact on performance or certification metrics.
Methods: Call data from 2005 was compared with 2015 data. Specific metrics included call volume (exposures, information calls, and total calls), staffing levels, caller location, patient age, management site, follow-up call prevalence and medical outcome. Comparisons are made on human exposures (HE) only unless otherwise noted.
Results: Total call volume decreased by 20% over the ten year period; information calls by 63% and HE by only 5%, yet average staffing (poison specialists) increased by 12%. Exposure call to FTE ratio and call penetrance decreased by 16% and 31%, respectively. Compared to 2005, calls from a residence decreased by 11.3% to 65.1% of HE calls in 2015; however the rate of calls from healthcare facilities (HCF) almost doubled (13.4% to 26.4%) during this same time. Children less than or equal to 6 years old accounted for 52% of HEs in 2005, and dropped to 44% in 2015. Increasing toxicity is reflected by the increase in hospital care, including a 100% increase in the percent of patients managed in intensive care units. Accordingly, the percent of patients with no effect/mild outcome decreased by 7%, but the number of patients with moderate/major outcome and death increased by 135% (from 5.4 to 12.7 percent). The decrease in incoming call volume was offset by an increase in follow-up calls. The percent of calls with at least one follow-up increased from 42% to 56% of human exposures, the average number of follow-up calls per day increased by 45% and more calls (11.2% vs 5.9%) were followed for longer than 3 days.
Conclusions: Over the last 10 years, more staff are managing less incoming calls in a sample RPC. However, PCs are now managing patients who are older, being treated in hospitals, and requiring more resources such as follow-up calls. These data suggest that call metrics developed decades ago, which centers often use to guide staffing and budgets, are likely obsolete and require updating, particularly if used as performance measures.

KEYWORDS Call volume; Metrics; Performance

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256. Adherence rate to poison center recommendations at a single regional poison center

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Background: Poison Centers (PC) play a critical role in the dissemination of poison information to healthcare providers with the ultimate goal of decreasing morbidity and mortality associated with toxic exposures. To our knowledge, there is little to no published data regarding the adherence rate of healthcare providers to PC recommendations. In addition, there is limited data regarding factors that may affect this adherence rate.
Methods: We performed a retrospective review of cases from a single regional PC made over a three-year period from 2012 to 2014. We limited our search to hospital encounters in which one of the following antidotes or interventions was recommended: fomepizole, insulin, hydroxycobalamin, naloxone, physostigmine, glucagon, n-acetylcysteine, urine alkalinization, hemodialysis, flumazenil, methylene blue, and octreotide. We excluded cases lost to followup from the final analysis. Additionally, we separately analyzed cases in which a medical toxicologist provided input and compared the adherence rate to that of cases without such direct input.
Results: We found 860 cases that were suitable for review. Of those, 58 had unknown outcomes and were excluded from the final analysis. The overall adherence rate to PC recommendations was 92.5%. The adherence rates with and without the involvement of a medical toxicologist were 91.6% and 93.0%, respectively. This difference was not statistically significant as determined by a Fisher Exact Test.
Conclusions: This retrospective review determined the adherence rate for specific PC encounters over a three-year period. Additionally, we found that involvement of a medical toxicologist did not significantly affect the adherence rate in this limited sample. The overall adherence rate of 92.5% is slightly higher than values reported in other reviews that analyzed referring physicians' adherence to consultants' recommendations, which ranged from 72% to 85%. This study is of limited size, and a more comprehensive review of multiple PCs would provide further insight. We recommend continued research into PC adherence rates and factors that affect adherence, as this could potentially lead to improvements in adherence and ultimately patient outcomes.

KEYWORDS Poison Center; Adherence; Recommendation

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257. Public health risks associated with large acute ingestions of apricot pits - an educational miss!

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Background: Recently there has been increasing interest in the consumption of amygdalin-containing apricot pits as adjuncts to or in place of modern medicine for the cure or prevention of certain diseases. In the stomach, amygdalin is hydrolyzed first to mandelonitrile, then again to hydrogen cyanide, potentially causing toxicity if systemically absorbed. Apricot kernels can easily be mistaken for almonds.
Case Report 1: A 72 yo female and a 68 yo male reported that they had consumed about 20 apricot kernels each mistaking them for almonds. The apricot kernels belonged to another resident in the home as an adjunct therapy for breast cancer. After consulting with a regional poison center (RPC) both patients were referred to a local ER and were given multi-dose charcoal at 25gm PO every 4 hours for 3 doses. The lactate levels for the male increased from 10 to 17.8 mg/dl, however, the levels for the female increased beyond the normal range from 12.9 to 23.3 mg/dl (normal: 5-19.8) Both patients remained asymptomatic with

normal ABG's and no signs of hypoxia, and were discharged the following day.
Case Report 2: A 63 yo male with a history of hypertension and arthritis read that a large ingestion of apricot kernels can be helpful for reducing arthritic pain and blood pressure. Consulting with the RPC, he was referred to a local ER after ingesting 25 apricot kernels. He was given a single dose of activated charcoal. All labs two hours post-ingestion were unremarkable. After a 6-hour observation period, the patient remained asymptomatic and was discharged home.
Case Discussion: Apricot kernels, like many fruit seeds of the Prunus genus, have been associated with cyanide toxicity when the kernel is chewed or crushed. Recommendations from packages bought at health food stores suggest consuming 3-5 apricot kernels per day as a homeopathic remedy. The amount of cyanide produced from apricot kernels range from 0.122 to 4.09 mg/g, with an average of 2.92mg/g. A lethal dose of cyanide in humans is 0.56 – 1.52mg/kg. Consumption of apricot kernels has been reported to cause mild symptoms, like headache, nausea, and vomiting, but with excessive use can lead to life-threatening symptoms like cyanosis, cardiovascular collapse, and death.
Conclusions: Foods products containing cyanogenic glycosides are currently available, yet unregulated and marketed for presumed health benefits. Consumers are unknowingly at risk for developing significant symptoms since there are no warning labels making the consumer aware of the potential risks. Due to the current popularity of amygdalin therapy of those seeking alternative therapies, consumers will continue to be at risk unless appropriate warning labels are affixed to these products.

KEYWORDS apricot; cyanide; toxicity

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258. Telephone Follow Up Confirmation (or Modification) of Poison Center Data for a 15-year Data Set

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Background: Case series and retrospective cohorts using poison control center data are common to the Medical Toxicology literature. There can be limitations to the data, including lack of case follow up and incorrect assignment of outcome. One option for attempting to temper these limitations is phone contact follow up with the patient or patient's guardian. It is not clearly established how effective or accurate this means of case verification is. We conducted a retrospective review of brake fluid exposures in toddlers using phone follow up to supplement our data.
Methods: A retrospective observational case series of pediatric brake fluid exposures reported to a single regional poison control center was conducted with IRB approval. All cases identified over a 15-year period were followed up by telephone to confirm or modify the recorded case outcome. A standardized questionnaire was used and 3 phone calls were attempted prior to a case being deemed lost to follow up.
Results: Attempts were made to contact all 121 cases initially identified in the series. Of these 121 cases, in 56 cases a phone number for callback was not available (27 disconnected numbers, 12 with only health care facility numbers, 11 with no caller information, 2 business numbers, 2 nurse lines and 2 incorrect numbers). Of the remaining 65 cases where phone numbers were available, 2 refused to answer survey questions, 2 did not recall phoning the Poison Center, and 47 resulted in no answer whatsoever. In only 14 cases were callbacks successful to completion of the standardized questionnaire. In 11 of these 14 cases a callback resulted in a change to the previously recorded final outcome. Successful callback rates for each year were as follows: 2000 (50%, 1/2 cases), 2001 (0%, 0/4 cases), 2002 (20%, 1/5 cases), 2003 (0%, 0/3 cases), 2004 (0%, 0/2 cases), 2005 (100%, 2/2 cases), 2006 (0%, 0/6 cases), 2007 (50%, 2/4 cases), 2008 (17%, 1/6), 2009 (0%, 0/3 cases), 2010 (0%, 0/4 cases), 2011 (67%, 2/3 cases), 2012 (50%, 2/4 cases), 2013 (75%, 3/4 cases), 2014 (0%, 0 cases).
Conclusions: Only 14 out of 121 cases resulted in successful callback completion (11.6%; 95%CI 5.9%-17.3%). Despite the low successful callback rate, 11 resulted in the previously documented case outcome being changed (9.1%; 95%CI 4.0%-14.2%). Successful callbacks were more common in recent years but spanned the entire 15-year set. Attempts at telephone confirmation of patients in retrospective poison center case series may reveal incorrectly documented case outcomes. In these data callback attempts to confirm outcomes had a low success rate but when callback was successful the final outcome frequently changed.

KEYWORDS brake fluid; poison center; pediatric

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259. Toxicity of Inadvertent Promethazine Exposures in the Pediatric Population

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Background: Unintentional ingestions of promethazine are frequent in children, mostly as tablets or suppositories. Although there are a few case reports of toxicity resulting from promethazine ingestion, there are no triage guidelines for referral to a healthcare facility. Our primary aim is to report the minimum dose of promethazine ingested by a child <6 years resulting in moderate or severe symptoms.
Methods: Retrospective chart study of regional poison center (RPC) cases involving children age 6 years or less of a single acute ingestion of promethazine from December 1999 to December 2015. Other inclusion criteria required a historical amount of promethazine ingested per weight (mg/kg) and a documented outcome.
Results: Out of 1179 cases identified, 172 cases met all inclusion criteria. Males made up 57%. Of the 172 cases, 43 involved exact known amounts ingested (25%), 3 estimated amounts (<2%), and 126 amounts were described as maximum possible (73%). The known exact amounts of promethazine ingested ranged from a taste to 9.8 mg/kg, with a possible maximum amount of 61.9 mg/kg. By history, 20 had ingested ≤1 mg/kg (11.6%), which is the maximum therapeutic dose used as an antiemetic; 119 had ingested >1-6 mg/kg (69.2%), which is the maximum therapeutic total daily dose used as an antiemetic; 33 had ingested >6 mg/kg (19.2%). The most common symptoms reported were drowsiness (43%, 0.1-36.5 mg/kg), agitation (6.4%, 1.1-11 mg/kg), vomiting (3.5%, 0.6-9.8 mg/kg), and mydriasis (2.3%, 1.2-9.8 mg/kg). Two case reports of dystonia (1.2%) had known amounts ingested of 0.6 mg/kg and 1.1 mg/kg, and one case report of hallucinations (0.6%) had a known amount ingested of 9.8 mg/kg. No effects were reported in 57.6% of the cases. Eighty cases (46.5%) were treated at home, averaging 2.0 mg/kg, and 92 (53.5%) were evaluated in a hospital, averaging 7.1 mg/kg. Of those treated in a hospital, 40 (23.3%) were given activated charcoal; the remaining

were observed with no further therapeutic intervention necessary. This sample size provides statistical support that this no-effect finding is predictive at >95% confidence.
Conclusions: Based on this data and the recommended max daily dose, pediatric ingestions of <6mg/kg promethazine are not likely to cause serious toxicity and can be managed outside the HCF. Any cases where dystonia or hallucinations are noted in the home setting should continue to warrant HCF referral and evaluation. Continued monitoring of promethazine ingestions in children ≤6 years of age will be needed to reconfirm this triage guideline as safe and reliable.

KEYWORDS Promethazine; Toxicity; Pediatrics

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260. Trazodone ingestion in children less than 6 years of age... When to worry?

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Background: Trazodone (Desyrel®) is an atypical antidepressant used for treating depression and insomnia. Trazodone exhibits antagonistic activity at 5-HT1a and 5-HT2c sites, with significant blockade of peripheral alpha-adrenergic receptors, moderate antihistaminic effects, and weak anticholinergic activity. Although approved by the FDA in 1991, its safety and effectiveness in children has not been established. In previous studies, trazodone ingestions of 50 to 500 mg resulted in no complications. This study seeks to determine a more accurate amount of trazodone ingested by children in this age group that could be safely managed at home.
Methods: All cases of unintentional trazodone-only ingestion by children <6 years of age reported to our regional PC between 2000 and 2015 were retrospectively analyzed for amount of drug ingested per body weight, clinical effects reported, and the potential impact of therapies provided. Cases involving co-ingestants or chronic exposures were excluded.
Results: Of the 418 cases identified, 283 cases met all inclusion criteria. Of these cases, 269 (95%) were unintentional ingestions; 14 were therapeutic errors. Males accounted for 55%. Children <2 years comprised 70%. A majority of the cases (52%) were either referred into a HCF or already being treated in a HCF at the time of the call to the GPC; the remaining were managed at home. Of those managed in HCF, 86 were treated and released; 12 admitted to the ICU; 15 admitted to a non-ICU; 35 either refused the referral, were AMA or lost to follow-up. Out of the 112 cases followed to a known outcome, 2 cases had moderate effects, 34 with minor, and the remaining reported no effects. Of the 15 cases with exact amounts and known outcomes (range =25 – 150 mg; 2.2 – 11 mg/kg), drowsiness was reported in 11 cases (3.1 – 11 mg/kg) and vomiting in 2 (3.3 – 5.8 mg/kg). One 2 year old male who ingested 6.9 mg/kg experienced priapism while admitted to the hospital. Of the 27 cases with estimated amounts and known outcomes (range =25 – 200 MG; 1.1 – 12.5 mg/kg), 9 reported drowsiness, 2 ataxia, and 1 vomiting.
Conclusions: Based on the results, pediatric ingestions of <12 mg/kg trazodone are not likely to cause serious toxicity and can be safely managed outside of a HCF. Utilizing this triage guideline, 53 of the 148 cases treated in a HCF could have been managed at home. However, any case where CNS symptoms beyond drowsiness, or other concerning systemic symptoms, are noted in the home setting should continue to warrant HCF referral and evaluation. Continued monitoring of trazodone ingestions in children <6 years of age will be needed to reconfirm this triage guideline as safe and reliable.

KEYWORDS Trazodone; Toxicity; Pediatrics

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261. Let Them Sleep Or Send Them In? Accidental Exposures to Alprazolam in the Pediatric Population

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Background: Alprazolam (Xanax®) is a benzodiazepine prescribed for anxiety disorders, panic attacks, and seizures. Alprazolam's somewhat different chemical structure than other benzodiazepine drugs makes it uncertain whether a different triage level might be more appropriate than one extrapolated from other benzodiazepines. The purpose of this study is to determine the amount of alprazolam ingested by children that could be safely managed at home.
Methods: In this retrospective case study, electronic records over a 15 year period within a regional poison center (RPC) were queried for all cases involving children ages <6 years with a reported acute ingestion of alprazolam only. Other inclusion criteria required being followed to a known outcome and a specific ingested amount recorded. Cases with a documented negative UDS were excluded despite report of ingestion.
Results: Of an initial 941 cases, 200 cases met all the inclusion criteria. Males accounted for 51%. Ten patients (pts) were <12 months of age (5%), 58 pts 12 months to <2 years (29%), 79 pts 2 years (39.5%), and 53 pts >3 years (26.5%). Of the cases, 64% were either referred into a HCF or already being treated in a HCF at the time of the call to RPC; 36% were managed at home. Of the 45 cases reporting exact amounts (0.13-3.5mg, 0.011-0.31 mg/kg), 24 cases (53%) resulted in no effects; 18 pts had drowsiness (0.011-0.15 mg/kg), 7 pts had ataxia (0.031-0.14mg/kg), 2 pt were agitated (0.087, 0.2mg/kg), and 2 were confused (0.039, 0.087mg/kg). One pt with an exact amount developed hypotension (BP 84/47, 0.10 mg/kg) with no intervention necessary, and another pt developed respiratory depression (0.13 mg/kg) with O2 sats at 78% while sleeping, requiring oxygen supplementation, and a dose of flumazenil. Of the 42 cases reporting estimated amounts ingested (0.025-64mg, 0.003-5.33mg/kg), 12 pts had drowsiness (0.02-0.4 mg/kg), 4 pts were ataxic (0.012-0.12mg/kg), 1 vomited (0.21mg/kg), and 1 pt was tachycardic (HR 134, 0.4mg/kg). Only 1 case had report of respiratory arrest (0.37 mg/kg), requiring 4 doses of flumazenil before returning to baseline; however the amount was classified as "maximum possible". In all, there were 3 cases in which flumazenil was given, with 2 for respiratory related issues (0.13, 0.37mg/kg) and 1 for CNS depression (0.13 mg/kg). No deaths were reported in any cases.
Conclusions: Data from our RPC suggests that pediatric ingestions of <0.13 mg/kg of alprazolam (based on cases with firm dose history) are not likely to cause serious toxicity and could be safely managed at home with sufficient follow-up. However, any case where CNS symptoms beyond drowsiness occurs should warrant HCF referral. Continued monitoring of alprazolam ingestions in children will be needed to reconfirm this triage guideline as safe and reliable.

KEYWORDS Alprazolam; Pediatric; Toxicity

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262. The Biological Agent Ricin: No Need to Panic

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Background: Ricin is classified as a Category B biothreat by the Centers for Disease Control and Prevention (CDC); its very name can evoke fear. Considered fairly easy to disseminate, it can cause moderate morbidity and requires enhanced vigilance by law enforcement and public health officials. Ricin is rarely successful as an agent for suicide and/or homicide. The most hazardous routes of exposure are inhalation and parenteral. Airborne ricin is unlikely to persist for more than several hours and symptoms from an inhalation exposure would manifest within 12 hours. Parenteral exposure is rare; the most cited case is the assassination of Bulgarian Markov, via injection of a ricin-containing pellet. Ingestion, the route most often reported to poison centers (PCs), requires mastication or maceration of many seeds to cause toxicity. Gastroenteritis, electrolyte abnormalities and shock are possible. We report on the public safety issues associated with an intentional ingestion of castor bean seeds and their remediation through collaboration and educational efforts spearheaded by a PC.
Case Report:A 40-year-old suicidal male ingested 50 ground castor bean seeds obtained on the internet. He developed severe hemorrhagic gastroenteritis but survived with aggressive intravenous fluid replacement and transfusions. A self-purported "botany student", he stated he took precautions to avoid aerosolization and poisoning others. Toxic ingestion was evident by patient history, clinical presentation and confirmation of the presence of urine ricinine.
Case Discussion: There was no evidence that the patient was motivated to produce a weapons-grade form of ricin, nor evidence of powder residues suggesting inhalation or dermal exposure. The castor bean slurry was contained in the blender. The patient received decontamination. Throughout his transport and hospital stay, concerns were expressed among those involved regarding risk of exposure and cross-contamination. First-line responders contacted the PC about patient handling and transportation. Law enforcement inquired about potential risk incurred when bagging evidence. A nursing supervisor questioned hospital staff exposure to bodily fluids, as well as potential for inhalation and cross-contamination. An ICU nurse had been discarding bedpans after each stool. To help alleviate anxiety and halt unnecessary practices, the PC entered into a partnership with the local health department, law enforcement and the CDC. Each set of healthcare professionals were provided with up-to-date concise information on toxicity and safety issues. Written information was quickly provided to the ICU staff to help alleviate anxiety and promote good practices. Teaching points were reinforced daily. Reassurance was given that person-person transmission was very unlikely. Testing of environmental samples was undertaken and ricin was present in the slurry residue contained in the blender.
Conclusions: A homicidal or suicidal individual can research and acquire highly toxic compounds via the internet. Potentially deadly biological toxins, such as ricin, are not routinely encountered by healthcare providers and law enforcement and may lead to aberrant practices as well as undue anxiety. In addition to patient management, a PC can guide collaboration among various responders and agencies to disseminate necessary information and manage public health aspects. PCs can indeed play a vital role in the public health arena.

KEYWORDS Hazard preparedness; education; public health

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263. Planting the Seeds, Watching Them Grow: Evaluation of a Poison Prevention Outreach Training Program

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Background: The objectives of this project were to evaluate an ongoing "train-the-trainer" program to see whether program participants were 1) gaining factual knowledge about poison center services and poisoning prevention methods, and 2) subsequently sharing that information with others. The ongoing creation of multiple, well-educated poison prevention advocates throughout the poison center's service area greatly increases the reach of the poison center's education program.
Methods: This project involved delivery of one 90 minute training (in-person or video streamed) to individuals who communicate safety messages to the public as part of their paid employment or training. Participants included parent educators, community safety educators, pre-service and hospital-based healthcare professionals, and youth counsellors. Evaluation of participants involved pre-tests of 10 specific facts, a corresponding post-test and satisfaction survey, and a follow-up survey of behavioral objectives 30 days post training. The 10 questions include basic information about the services of the poison control center, who gets poisoned most frequently and by what substances, and what poison prevention techniques are most effective.
Results: In 2015, 29 training sessions were conducted to a total of 751 participants working across 4 counties. Sixty-two percent of sessions (18/29) involving a total of 234 individuals were formally evaluated. Participants' mean pre-test score was 54.32 out of 100, with scores ranging from 0 to 100. The mean post-test score was 91.05, with a reduced range of 50 to 100. On average, participants' scores improved by 37 points. For the 213 participants who completed both pre- and post-tests, results of a paired t-test of scores were statistically significant at the <0.0001 level, meaning these results were extremely unlikely to have occurred by chance if there was no actual change in participant knowledge. Score improvements differed by healthcare provider status, with higher mean gains (43.8) achieved by non-health care providers versus health care providers (32.5). Knowledge gains were also slightly lower for students rotating at the poison center than for others, although every session showed gains above the a priori standard set for success of at least a 20% score improvement. Of the 55 participants responding to the 30 day follow-up survey:

- 92.5% (50/54) stated they found the information useful and relevant for their work
- 74.6% (41/55) reported feeling "very comfortable" or "comfortable" answering questions from the public about poison control centers
- 64.8% (35/54) promoted the poison center informally to friends and family
- 51.9% (28/54) received bulk printed materials from the poison center educators
- 29.6% (16/54) held an event which featured poison control materials.

Conclusions: This evaluation indicates that a well-planned "train-the-trainer" program can markedly increase the knowledge of individuals sharing safety information with the public in a single session.

KEYWORDS evaluation; prevention; train-the-trainer

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264. Test Your Medicine IQ—A Comparison of Educational Outreach Methods in Older Adults

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Background: Older adults taking multiple medicines are at risk for improper use of these medicines, accidental overdose and adverse events. Educating older adults in safe use of medicines can be done using educational programs and/or medicine safety literature. There are challenges with both approaches. Educational programs are resource intensive and medicine safety literature may present health literacy issues.
Methods: Older adults were recruited from senior living facilities. Subjects either participated in an interactive medicine safety education session ("Game Group") or read medicine safety literature ("Brochure Group"). Both groups were given pre and post surveys to assess baseline and change in knowledge about medicine safety and poison prevention before and after each intervention.
Results: A total of 101 older adults participated in the study. All survey questions pre- and post-intervention were answered by 27 participants in the game group and 26 participants in the brochure group. In the game group, the median improvement in the index score between baseline and post-intervention was 3, moving from a median score of 9 (IQR 6,9) to 11 (IQR 9, 12). In the brochure group, the median improvement was 1, moving from a median score of 7.5 (IQR 6,8) to 8 (IQR 5, 10). Among those with the incorrect response at baseline, participants in the game group were significantly more likely to report the correct response post-intervention than participants in the brochure group for questions about child-resistant caps, including herbals and vitamins on a medicine list, and whom to call in the middle of the night for advice about medications. Knowledge of poison center phone number and keeping a dietary supplement list improved in both groups but there was no significant difference between groups. Pre-survey results indicated that both groups knew to read the label before using a medicine, not to share medicines and to keep prescription, OTC and vitamin lists.
Conclusions: Older adults demonstrated prior knowledge of some medicine safety information. While improving older adults' knowledge about medicine safety can be achieved using an interactive game and medicine safety literature, the interactive game was more effective. Educational efforts should emphasize the importance of child-resistant caps, including herbals and vitamins on medicine lists and using the poison center as a resource for medicine information. Additionally, complex health tasks such as reading and interpreting medicine labels may require more active learning techniques to be sure that older adults are able to perform the task independently.

KEYWORDS Medication Safety; Older Adults; Education

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265. 911 Dispatcher Curriculum & Outcomes: "We Can Call Poison Control for That?!"

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Background: A hyper-focused intervention was designed, implemented and evaluated, after conducting surveys, key informant interviews, and a focus group with 911 personnel. "Interfacing with the PC" was designed with participant input to address previous findings and close learning gaps. The intervention and outcomes follow.
Methods: The 911 dispatcher curriculum (911DC) delves into the roles of poison specialists vs. dispatchers, effective communication, liability, and extensive case-based learning. Dispatchers read & discuss redacted PC cases to determine if callers need the PC or 911 or both. The 911DC also includes PC basics, poisoning across the lifespan, and hot poisoning trends. Pre and post-tests evaluate 911 dispatchers' knowledge, skills and attitudes towards a PC.
Results: The 911DC training had 81 dispatchers in 7 sessions. 52 participants in 5 sessions completed written evaluations. Pre and post-tests shared six core questions, demographic queries, and program satisfaction questions. Dispatchers had experience of 0-2 years (12), 3-5 (6), 5+(10), 10+(14), and 15+ years (10). Less experience correlated with greater increase in correct answers post intervention. Most had at least one type of dispatcher training and a few had other emergency services training. Average dispatcher scores reflected a 19% increase in correct answers between the pre-test score (74) and the post-test (92). Notably, dispatchers increasingly chose hazmat, carbon monoxide, and bio terrorism calls as appropriate to refer to a PC. Top items learned include: when to call PC, PC resources/uses, triaging calls, current poison trends, PC staffing, and communication tips.
Conclusions: A hyper-segmented target audience shows significant increase of knowledge, skills, and attitudes related to calling the PC. Evaluations place high value on and deep satisfaction with the tailored 911DC. Other PCs may benefit from teaching a similar 911DC highlighting roles & responsibilities, liability, and intensive case-based learning.

KEYWORDS 911 dispatcher; outcome evaluation; education

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266. Pediatric death after exposure to cannabis

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Background: Since marijuana legalization, pediatric exposures to cannabis have increased, resulting in increased emergency department (ED) visits.1 Neurologic toxicity is most common after pediatric exposure; however, gastrointestinal and cardiopulmonary toxicity are reported.1 Prognosis is often reassuring.1 Specifically, myocardial complications related to cannabis include acute coronary syndrome, cardiomyopathy, myocarditis, pericarditis, and dysrhythmias.2 To date, pediatric deaths from cannabis exposure have not been reported. The authors report an 11-month-old male who, following cannabis exposure, presented in cardiac arrest after seizure and died. Myocarditis was diagnosed post mortem.

Case Report: An 11-month-old male presented to the ED in cardiac arrest. The patient was lethargic for two hours after awakening and then seized. During the prior 24-48 hours, he was irritable with decreased activity and later was retching. The cause of illness and death were unknown. Positive findings in the ED included metabolic acidosis and urine ELISA positive for THC-COOH. Route of exposure was unknown. Autopsy revealed myocarditis without evidence of concomitant bacterial or viral infection. Post mortem cardiac blood analysis confirmed the presence of Δ-9 Carboxy-THC at 7.8 ng/mL. Additional history disclosed parental admission of drug possession.
Case Discussion: Given the existing relationship between cannabis and cardiovascular (CV) toxicity, the authors propose a relationship between cannabis exposure in this patient and myocarditis. CV effects of cannabis include tachycardia and decreased vascular resistance with acute use and bradycardia in repeat exposures.2-5 In addition, several case reports link cannabis use to myocarditis. In 2008, Leontiadis reported a 16-year-old with severe heart failure requiring a left ventricular assist device, secondary to biopsy-diagnosed myocarditis ultimately attributed to cannabis use of unknown chronicity.6 In 2014, Rodríguez-Castro reported a 29-year-old male who had two episodes of myopericarditis within two days of smoking cannabis believed to be adulterated, but unconfirmed.7 In 2016, Tournebize reported a 15-year-old male diagnosed with MRI evidence of myocarditis after initiating cannabis use eight months earlier.8 There were no adulterants identified in this patient's consumed marijuana.
Conclusions: Of the reported cases of cannabis-induced myocarditis, patients were previously healthy and no evidence was found for other etiologies – including active infection. All of the prior reported cases were associated with full recovery. In the reported case, however, the patient died after myocarditis-associated cardiac arrest. The patient was exposed to cannabis, which we believe played a significant role in his death.

KEYWORDS Cannabis; pediatric; death

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267. Don't Drink NOS: Unintentional Ingestion of Methylcyclopentadienyl Manganese

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Background: NOS Energy Drink is produced by Monster Beverage and labeled with an identical logo and similar packaging to NOS (nitrous oxide systems) octane booster, a fuel additive sold by Holley Performance Products. NOS octane booster contains methylcyclopentadienyl manganese tricarbonyl (MMT) and kerosene. MMT is used to boost the octane rating of gasoline; it is also a known neurotoxin associated with seizures. Documented human poisoning cases are scarce. We report a case of MMT poisoning associated with status epilepticus secondary to mistaking NOS octane booster for NOS Energy Drink.
Case Report: A 54-year-old man with history of methamphetamine and marijuana use presented to an emergency department (ED) following 3 witnessed tonic clonic seizures. He had consumed a 12 oz bottle of NOS brand octane booster, mistaking it for the energy drink, after acquiring the bottle at a food bank. He felt sick immediately after ingestion and shortly thereafter had a generalized tonic clonic seizure. EMS was contacted. He had two additional seizures, which were followed by a postictal period. Upon ED arrival his vital signs were: temperature 36.1 °C, heart rate 123/minute, respiratory rate 16/min, blood pressure 129/74 mmHg, and oxygen saturation 94%. He was severely agitated and combative, requiring sedation with 4 mg lorazepam and 4 mg midazolam followed by intubation. Lab studies showed a mixed respiratory and metabolic acidosis with a pH of 6.95. Initial lactate was 19.4 mmol/L. Urine toxicology screen was positive for benzodiazepines and cannabinoids. He remained on mechanical ventilation for four days and received levetiracetam and continuous EEG monitoring which showed no ongoing epileptiform abnormalities. He was also treated for aspiration pneumonia. Upon extubation, he was alert and oriented but with significant ataxia that improved with physical therapy. He was discharged with instructions to continue levetiracetam and follow up with neurology as an outpatient.
Case Discussion: MMT became a popular fuel additive for boosting fuel octane rating following the banning of tetraethyl lead in the US in gasoline in 1995. Seizures have been reported in humans and experimental animals following MMT exposure, though human poisoning is rarely documented. In this case, ingestion led to status epilepticus and marked ataxia, which have not been previously reported. The remarkably similar packaging to NOS Energy Drink represents a significant risk to those who might mistake one product for the other.
Conclusions: Ingestion of methylcyclopentadienyl manganese tricarbonyl, an ingredient in NOS octane booster, was associated with status epilepticus. The identical logo and similar packaging of this fuel additive to NOS Energy Drink represents a significant hazard to consumers.

KEYWORDS Energy drink; Methylcyclopentadienyl manganese tricarbonyl; NOS octane booster

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268. Emergency Medicine Residency toxicology education: a survey study

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Background: There is currently limited published information describing US emergency medicine (EM) resident toxicology (TOX) education. We sought to determine the nature of this education through survey of EM residency program directors.
Methods: A computerized survey was sent to program and assistant program directors of the 164 ACGME approved US EM residency programs. A follow-up email was sent 1 and 2 weeks following initial request. Respondents were asked whether a rotation in TOX (mandatory or elective) was part of the residency curriculum, the duration and nature of rotation, whether the rotation was available locally (<1hr drive from home institution), the number of full-time board-certified/eligible TOX faculty, and the number of TOX lectures that were given to residents each calendar year outside of a rotation.
Results: 107 programs responded (65%). Seventy-one programs reported a mandatory rotation (66%) and 22 reported an available elective (21%). Rotation durations were 4 weeks (n = 67, 72%), 3 weeks (n = 7, 4%), 2 weeks (n = 18, 18%) and 1 week (n = 1, 1%). Lecture-based didactics (n = 87, 94%); 'on-call' to respond to toxicology consults (n = 59, 63%); rounds on emergency department or inpatient toxicology patients (n = 65, 70%); call-backs to healthcare providers from a poison control center (n = 60, 65%); answering poison control center calls (n = 32, 34%); self-directed learning (n = 70, 75%); accessing online lectures or other resources (n = 27,

29%); and case-based presentation or discussion (n = 22, 24%) were reported components of rotation. Nine programs with a mandatory rotation were more than 1hr drive from home institution (mean: 6hrs; range: 2-12hrs). Seven programs with available elective were more than 1hr drive from the home institution (mean: 4hrs; range: 2-5hrs). Twenty-one programs reported no full-time TOX faculty (23%), 22 reported 1 full-time faculty (24%), 41 reported 2-5 full-time faculty (44%,), 7 reported 6-10 full-time faculty (8%), and 2 programs reported more than 10 full-time faculty (2%). Of the 14 responding programs with no available rotation, 11 reported zero full-time TOX faculty (79%), 1 reported one full-time faculty (7%), and 2 reported 2 full-time faculty (14%). The mean number of TOX lectures in programs with mandatory or elective rotations was 11 (range: 0-35) and 11.6 (range 0-20) amongst programs without an available rotation.
Conclusion: The majority of responding EM training programs have mandatory or elective medical toxicology rotations of 2-4 weeks. However, almost half of the responding programs report 1 or less full time medical toxicology faculty. Further investigation is warranted to determine the impact this discrepancy may have on medical toxicology education to EM residents.

KEYWORDS toxicology education; emergency medicine residency; toxicology rotations

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269. Bringing the National Library of Medicine Toxicology Tutorials into the 21st Century

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Background: The (U.S.) National Library of Medicine (NLM) is the world's largest biomedical library, providing free resources to both professionals and consumers. NLM's Specialized Information Services Division (SIS) is responsible for information resources and services in toxicology, including clinical toxicology, and in environmental health, chemistry, and other topics. NLM's Toxicology and Environmental Health Information Programs (TEHIP) include the TOXNET (TOXicology Data NETwork) suite of databases. Examples of TOXNET's databases include the Hazardous Substance Data Bank (HSDB), Household Products Database (HPD), and the Comparative Toxicogenomics Database (CTD). People using TOXNET may have limited knowledge of the science behind some of the resources. The Toxicology Tutorials were created in 1998 to help TOXNET users comprehend what they are reading. The tutorials cover the basic toxic mechanisms in terms and concepts that are understandable to beginning college students.
Methods: SIS worked with subject matter experts and others to review the contents of the 1998 Toxicology Tutorials for areas to enhance and new topics to add.
Results: The updated and enhanced tutorials include a large amount of new information on topics like clinical toxicology, routes of exposure, risk assessment, and alternatives to the use of animals in research and testing. In addition, the new design for the tutorials lets users access them via smartphone, tablet, or desktop.
Conclusions: The new (2016) version of the Toxicology Tutorials is the first update in 18 years, and includes up-to-date information on clinical toxicology and other types of toxicology, routes of exposure, risk assessment, and alternatives to the use of animals in research and testing, etc. In addition, the new design lets users access the tutorials where, when, and how they want to have access, e.g., via smartphones and tablets.

KEYWORDS Education; Online; Toxicology

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270. Demographics of Twitter-Based Toxicology Learners

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Background: Access to experts and pertinent medical literature through social media platforms like Twitter guides learners in multiple fields of study. Toxicologists have leveraged Twitter to disseminate literature, share conference information, and address controversies in various domains. Twitter-based learners utilize hashtags (#) to aggregate topical tweets for downstream use. The demographic of users who utilize Twitter to learn toxicology is unknown. The purpose of this study is to improve toxicologists' ability to communicate through Twitter by characterizing the demographic and tweeting preferences of users who leverage Twitter to learn toxicology.
Methods: We developed a 15 question, multiple-choice survey to assess the demographics of Twitter-based toxicology learners and the preference of information contained in tweets. Users were asked basic twitter-graphics (age, gender, location, number of followers, year they joined Twitter, and number of times they accessed and posted on Twitter), and asked to rank on a 5-point Likert scale their willingness to engage with a variety of tweet categories. The survey was reviewed and piloted by members of the study team to ensure readability. Study investigators tweeted a link to the survey labeled with the hashtags #FOAMtox, #NACCT15, and #FOAMed. Anonymous survey responses were collected through a secure Surveymonkey database.
Results: Over a 2-month period, 91 participants responded to the survey. The average age of respondents was 38, and the majority of users were located in the United States and Canada. Fifty seven percent of respondents were male; 43% were female. Only 20% (n = 16) respondents identified as toxicologists or fellows in training; the majority of participants identified as emergency medicine physicians while 48% identified as pharmacists or poison specialists. The majority (64%; n = 46) of users joined Twitter between 2010-2014, reported accessing Twitter daily and generated unique posts 1-3 times a week. Users felt that they were most likely to be engaged by tweets containing opinions of toxicologists, links to literature on toxicology, or a link to information on a medical education blog. Users ranked opinions from major news outlets and posts from governmental regulatory agencies as least useful. Seventy percent of users are not active users of the #FOAMtox hashtag.
Conclusions: Twitter users who interact with toxicologists tend to be emergency medicine physicians, pharmacists, and poison specialists. These users are willing to interact with tweets containing clinical toxicology information or links to pertinent literature. This study suggests that toxicologists who wish to interact with learners on Twitter should tailor their tweets to maximize engagement.

KEYWORDS Twitter; Social Media; Education

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271. American Indian Communities: Who Are You And Do You Know Who We Are?

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Background: There are nine federally recognized American Indian tribes in our service area; we sought to identify which tribes predominated in the most populated urban area, where they lived and if they were familiar with or had ever called a poison center. This information was sought to better design effective outreach programs for this population.
Methods: We administered a needs assessment in the form of a survey at a PowWow held at a downtown urban site, promoted as a regional event. Survey participants were randomly selected males and females over the age of 12.
Results: The event sponsors estimated approximately 750 people attended. 34 self identified American Indians were queried, and 29 of those completed the survey; 5 declined to complete the survey (all males). Results showed 26 different tribal affiliations with 38% claiming more than one tribal affiliation and 3% did not know their tribal affiliation. When asked if they had ever heard of a poison control center (PCC), 79% said they had. 28% had called a PCC. Survey participants lived in 23 different zip codes. 82% lived in our service area and 4% lived in a border state serviced by another PCC. The majority of those surveyed lived within a 25 mile radius of our service area's urban center but only 17% were affiliated state tribes.
Conclusions: We were surprised by the number of different tribes represented, and will attempt to use this information to develop public education programs to appeal to a variety of different tribal cultures.

KEYWORDS American Indian; tribes; needs assessment

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272. Patterns in NRMP Match Data 2013-2015: Medical Toxicology and Beyond

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Background: Medical toxicology is a growing field but continues to face challenges in recruiting enough qualified applicants to fill available fellowship positions. Previous work has described similar concerns in other subspecialties, but little is known about contributing factors in medical toxicology training. This study reviewed recent match data to assess for patterns to further identify contributing factors in fellowship recruitment.
Methods: National Resident Matching Program (NRMP) Fellowship Match data for matriculation years 2013-2015 were reviewed for all participating specialties, including those which only participated during a portion of the study period. Data collected for each specialty included number of positions available, percent of positions filled in the match, number of applicants, and percent of applicants who went unmatched. For comparison, single year data were collected on percent of positions filled for the residency matches in emergency medicine and pediatrics, the two largest contributors to medical toxicology fellowships.
Results: For the two years participating in the NRMP match, medical toxicology averaged 38.5 positions available per year and filled 62.0% of these, with as many as four applicants per year not matching into any position. Other specialties with similar position fill rates during the same period include adult infectious disease (76%), adolescent medicine (62.6%), pediatric infectious disease (60.7%), pediatric rheumatology (57.8%), pediatric pulmonology (55.2%), medical genetics (49.1%), pediatric nephrology (46.4%), and geriatric medicine (43%). Higher performing specialties included pain medicine (99%), pulmonary/critical care (99%), pediatric emergency medicine (96.9%), and sports medicine (95%). Emergency medicine and pediatric residencies each exceeded 99% of available positions filled.
Conclusions: With the exception of pediatric emergency medicine, most pediatric subspecialties had position fill rates similar to or lower than that of medical toxicology. This may indicate a greater supply of subspecialty positions than demand from pediatric residents. In contrast, the subspecialties fed by emergency medicine programs had relatively high match rates. This may indicate a relatively greater supply of fellowship interested emergency medicine residents and an opportunity for improved targeting of recruitment efforts. Further study into motivating factors for residents pursuing fellowships is needed. This study is limited by its retrospective nature and the lack of availability of most recent (2016 matriculation year) data, as well as by confounders such as positions filling outside the NRMP match.

KEYWORDS Graduate medical education; Fellowship recruitment; Subspecialty match

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273. Clinical Toxicology Learning Experiences in Post-Graduate Emergency Medicine Pharmacy Residency Programs

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Background: Specialized post-graduate residency training in emergency medicine pharmacy is currently in place across several health-systems in the United States, and most of these residencies are accredited or candidates for accreditation by the American Society of Health-System Pharmacists (ASHP). One area included within the educational outcomes and goals as well as instructional objectives put forth by ASHP for post-graduate residents engaged in emergency medicine pharmacy training includes competency in caring for patients experience toxicological emergencies. The objective of this study is to characterize the extent and level of involvement of clinical toxicology training within post-graduate emergency medicine pharmacy residencies across the United States.
Methods: An electronic cross-sectional survey was distributed to program directors of post-graduate emergency medicine pharmacy residencies in the United States. The survey tool consisted of questions aimed to assess the extent of education and training of post-graduate emergency medicine pharmacy residents in the management of patients who present to the emergency department with toxicological emergencies. Responses were analyzed using descriptive statistics.
Results: Of the 24 responses (response rate =58.5%), the majority of respondents (87.5%) indicated that a formal clinical toxicology learning experience was required for post-graduate emergency medicine pharmacy residents. Most of these learning experiences were characterized as short-term, generally consisting of four weeks in duration, but one-quarter of respondents indicated that learning experiences in clinical toxicology for post-graduate

emergency medicine pharmacy residents were longitudinal in nature. In further characterization of the learning experience, 18 respondents (75%) reported that the learning experience in clinical toxicology was offered in conjunction with the local poison control center, while 10 respondents (41.7%) reported that the learning experience was provided in conjunction with the emergency medicine pharmacy service and/or on-site clinical toxicology service available at the institution. Activities that post-graduate emergency medicine pharmacy residents engage in related to clinical toxicology included participation in learning experiences and activities offered by the local poison control center related to clinical toxicology (18, 75%), presentation of formal didactic lectures (17, 70.8%), formal written toxicology consultation (12, 50%), longitudinal research related to clinical toxicology (11, 45.8%), and on-call services for toxicological emergencies (9, 37.5%).

Conclusions: Most post-graduate emergency medicine residency programs offer rigorous formal training in clinical toxicology with varied activities for residents during their learning experience. Potential areas of growth for formal training of clinical toxicology within such programs that exist to maximize the role of the emergency medicine pharmacists in the management of patients with toxicological emergencies include formal documentation of consultation, engagement in longitudinal research in the discipline, and on-call services for response. As emergency medicine pharmacists often serve as one of the first points of contact for patients who present with toxicological emergencies, these areas of growth can enhance continuous professional development of emergency medicine pharmacists in the provision of clinical toxicology services in the emergency department.

KEYWORDS Emergency medicine pharmacy; Clinical toxicology; Residency training

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274. YouTube: Effective Communication of Health Information to Refugees

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Background: Communicating health information to those with limited English proficiency can be a challenge. Finding an appropriate venue is crucial for an effective prevention campaign. Bhutanese refugees, the largest newly resettled group in a targeted county, were the focus of a medication safety campaign that started in 2015. This project's goal was to see if social media could be a cost effective and accessible way to educate refugees and influence behavior.

Methods: After consulting Bhutanese community leaders to identify community characteristics, it was determined that most had smart phones or computers to access health information. Video software was used to create a three minute Nepali video on medication safety which incorporated prevention tips. The video was recorded by an interpreter from a local refugee agency and included Nepali script. It was uploaded to YouTube on February 26, 2015 and the link was sent to the Bhutanese community via their list serve, verbally and Facebook. A post test was conducted during a gathering to help evaluate the effectiveness of social media for education and to assess if a prevention step (programming phone with poison center number) was implemented. Case managers also had new arrivals view the video and asked for feedback. There was a minimal cost to purchase the video software. The video is being played in the lobby of the refugee agency.

Results: There have been 378 YouTube views as of March 10, 2016. 25% of the viewers watched the entire video. This is above average audience retention among videos of this length according to YouTube analytics. On average, users watched 1:19 of the 3 minute video (46%). Traffic source to the video reported that 36% had a direct link, 30% conducted a YouTube search and 26% got to the video via an external app or website. Feedback from the refugee agency along with 92% of refugees surveyed (n= 25) reported that the format and venue were favorable for learning about medication safety. 76% programmed their phones. 100% correctly responded to a question on over-the-counter medication safety. There was a 79% increase in views since posting on the state's Bhutanese Association's list serve and Facebook page. The English as a Second Language Program at the state's refugee agency incorporated the English version of the video into their health section.

Conclusions: YouTube is a cost effective venue for making health information accessible to those with limited English proficiency. Involving local partners increased visibility and credibility. The language barrier and literacy issues were addressed by having the video recorded in Nepali. The Nepali script along with images helped to reinforce the message. Incorporating a measurable action step in the video message was successful in influencing behavior. Additional topics and ethnic groups will be targeted in the future.

KEYWORDS Prevention; Education; Refugees

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275. Pharmacy-Led RSI Simulation Program Benefits Emergency Physicians

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Background: The benefits of having clinical pharmacists in the ED has been well recognized, but 24/7 bedside pharmacist coverage for safer medication administration is not feasible everywhere. Therefore we conducted an educational high fidelity simulation (Sim) program to enhance Rapid Sequence Intubation (RSI) pharmaceutical care in a busy emergency medicine training (EM) program.

Methods: This study was conducted in a large academic teaching hospital with a PGY1-4 EM residency program in response to an earlier needs assessment initiative. Two different Sim cases that required RSI were developed, piloted, and revised by content experts prior to the Sim lab sessions. The 2 cases covered all potential RSI drugs used at our institution. Residents were grouped by PGY level then randomly chosen to run these cases while other trainees watched and scored their performance using a standardized checklist. The ED pharmacist provided checklist feedback to each participant and then conducted an interactive lecture after cases. All participants completed an anonymous closed-format instrument to evaluate the program.

Results: A total of 35 residents participated: 11/35 (31%) were female. All 35 (100%) reported the Sim lab improved overall understanding of RSI pharmacotherapy. Five (15%) preferred pharmacist-led teaching before the Sim lab case, 30/35 (85%) preferred teaching after cases. Eleven (31%) "strongly agreed" and 24/35 (69%) "agreed" the Sim lab enhanced understanding of alternative agents used in RSI. All participants (100%) agreed the Sim lab improved their overall ability to formulate a pharmacotherapy plan for ED patients in need of RSI. There were no differences based on PGY level.

Conclusions: A pharmacist-led high fidelity educational simulation lab provided a positive experience to EM trainees and can be implemented in other programs. Future research will assess knowledge retention and translation to the bedside.

KEYWORDS Education; Medication Safety; Simulation

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276. Education Innovation: Toxicology Clue

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Background: Emergency Medicine residents are responsible for a basic knowledge of toxicology. This presents a challenge to educators, as the topic is extremely broad and infrequently encountered in practice. We sought an engaging and informative means to teach basic toxicology, including some specific toxins and envenomations that may appear on EM boards, without resorting to an exhaustive list of exposure-antidote pairings for memorization.
The purpose of the project is to introduce natural toxin exposures, including clinical presentation, pharmacology, mechanism of action, and antidotes. Additionally, we aimed to familiarize residents with the appearance and names of toxic plants and animals, while encouraging group discussions among residents in order to identify knowledge gaps and facilitate peer teaching.
Methods: We developed a Clue (Hasbro, Pawtucket, RI) -inspired game asking teams of Emergency Medicine residents to solve each of 6 toxicologic exposure cases. Each case was presented as a clinical scenario describing an affliction suffered by one of the Clue characters, including location of exposure, activities associated with exposure, clinical presentation, and course of illness. Residents were then asked to: a. Identify the toxic agent (from a collections of pictures), b. Name the toxic agent (from cards including both common and scientific names), c. Identify the toxin (from cards noting specific toxin names and descriptions of effects), and d. Propose a treatment/antidote (from cards naming antidotes or "symptomatic care, no antidote available"). Teams worked together to identify the causative agent by sight, name, and toxin, as well as propose a treatment, for each toxic exposure. Available answers included various plants, animals, insects, and fungi. Each team also was given one "toxicology consult," enabling a question to a toxicologist to assist them.
Results: Of 62 Emergency Medicine residents, 15 completed an optional post-activity survey. Of the respondents, 93% of residents described the activity as innovative, and 87% stated that it was an effective learning tool. One notable critique from a PGY-1 resident stated that the activity would have been more effective if s/he had been introduced to general toxicology prior to the exercise. 87% of residents polled stated that they would like to participate in the activity again, with one write-in comment specifically requesting to repeat the activity with new cases, if possible.
Conclusions: This activity met its objectives as an innovative and enjoyable teaching tool, especially for residents or students possessing novice level general toxicology knowledge.

KEYWORDS Toxicology; Clue; education

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277. Poison Center Awareness Project amongst the Homeless Population

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Background: Homeless populations face many health risks while living in environmentally unstable conditions. These conditions pose hazardous indoor/outdoor threats for all ages, including women and children. Data from successful poison center outreach to this population is limited. There is data demonstrating that many homeless have or have access to personal cell phones. This poison center will attempt to demonstrate increased poison prevention awareness amongst the homeless population by providing basic information about poison control center services and the Poison Helpline phone number.
Methods: This poison center will partner with agencies providing assistance to the homeless by hosting events for unsheltered (literally homeless) and sheltered (transitional/emergency housing homeless) populations. We will host two poison control/prevention resource table events at soup kitchens to reach unsheltered populations. At the first event we will pre-test participants' basic knowledge about the poison center, (phone number, hours of operation, experts who answer the calls, cost and privacy concerns about the service and inquire if the person has previously called the poison center). We will have a follow up event at the same location to capture returning participants and give a post-test using the same questions from the pre-test. In addition to the previous questions, we will ask if the person called the poison center since the last event. Poison Helpline incentive items will be distributed at each event once the participant has completed the survey. Transitional/emergency housing facilities will receive presentations to learn about indoor poison hazards, poison control services offered and why they should refer their clients to use our services regarding poisonous exposures. Pre- and post-presentation surveys will be use to evaluate staff knowledge about poison control centers, services offered and if the program is perceived as a beneficial resource for their scope of work. Each presentation will include distribution materials for facilities for distribution to their clients.
Results: To be determined by the number of contacts reached at soup kitchen events, presentations, number of presentations conducted and number of materials distributed by partnering facilities. Results will also be collected through pre- and post-presentation surveys.
Conclusions This project is ongoing and in an early stage with additional results to follow. To date, 300 materials were distributed to six housing facilities with scheduled staff presentations for seven facilities.

KEYWORDS Homeless; Education; Shelters

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278. Severe burns associated with E-Cigarette malfunction

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Background: The prevalence and use of Electronic-cigarette (E-cig) and vaporizing products are increasing. These were introduced to the public as products that would potentially protect smokers from the dangerous effects of cigarette smoking while still delivering nicotine. Preliminary research suggests that there

are potential negative health consequences from use of E-cigs including inflammation of lung tissue, exacerbation of asthma, and allergic reactions. This case describes a more immediate hazard in a patient who sustained severe burns while carrying an E-cig product in their pants pocket.

Case Report: A 35 year-old male was evaluated in the Emergency Department after the battery pack on an E-cig exploded in his pocket. He presented immediately after the incident. On examination, vitals signs were stable but he was in significant pain. Exam revealed anterior thigh burns, blistering and charring extending down to the anterior and lateral knee. There were also burned areas on the abdomen. Both full thickness and partial thickness burns were noted. Areas of white discoloration as well as areas with lack of sensation were present. Blisters were both intact and ruptured with the largest approximately 3-4 cm in size. There was no evidence of nicotine toxicity or inhalational injury. Total body surface area of the burn was estimated at 5%. His initial treatment involved debridement, pain control and wound care. Follow up care was necessary with continued debridement and wound care appointments, and he ultimately required surgical excision and intermediate closure of a 3X4 cm portion of the largest full thickness burn on his thigh.

Case Discussion: An E-cig device is composed of a battery, an atomizer, and a nicotine cartridge which creates a nicotine vapor for inhalation. These devices are unregulated and their relative safety remains unknown and unstudied. This case reveals a significant hazard, that of full-thickness thermal burns, from the explosion of the lithium battery that powers many brands of these devices. Lithium batteries are high density energy storage cells that are known to experience "thermal runway." Lithium is an inherently unstable metal which if heated to its melting point can cause a violent explosion. Thermal runaway can occur secondary to poor design, use of low-quality materials, manufacturing flaws and defects, or improper use and handling. Lithium batteries in cell phones and laptop computers have also been shown to experience this same hazard with similar resulting injuries.

Conclusions: Lithium batteries in E-cig devices may become unstable and explode causing severe burns. Because no government agency regulates E-cigs, there are no safety standards in place for their production and use. Standards need to be developed and regulated to ensure quality and safety in these devices.

	Year			
Substance	2012	2013	2014	Total
Codeine	121 (24.15%)	93 (18.6%)	78 (15.95%)	292 (19.60%)
Hydrocodone	221 (44.11%)	224 (44.8%)	224 (45.81%)	672 (45.1%)
Hydromorphone	3 (0.60%)	4 (0.80%)	1 (0.20%)	8 (0.05%)
Morphine	18 (3.59%)	30 (6.0%)	17 (3.47%)	65 (4.36%)
Oxycodone	45 (8.98%)	58 (11.6%)	48 (9.82%)	151 (8.12%)
Oxymorphone	1 (0.2%)	1 (0.2%)	0 (0%)	2 (0.01%)
Tramadol	92 (18.36%)	90 (18.00%)	121 (24.74%)	303 (20.75%)
Total	501	500	489	1490

KEYWORDS Electronic-Cigarette; Burn; Malfunction

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279. Establishing a New Poison Prevention Outreach Program for Older Adults in Washington State

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Background: As the population of the United States ages, healthcare providers and public health agencies across the country must adapt programs and services to better meet the needs of adults aged 65 and over. Older Adults are the fastest growing age group in the state of Washington, and the U.S. Census estimated that in 2014 over 14% of Washington's population of 7.06 million people were 65 years or older. Prior to 2014, Washington's poison center health education only targeted parents and caregivers of young children as they represented a majority of the call volume.

Methods: As part of its responsibility to serve the entire state, the Washington Poison Center initiated a Needs Assessment in the summer of 2015 to accomplish the following: determine the landscape of older adult services and resources in Washington, examine utilization trends of poison center services by older adults, and identify relevant materials and partnerships that could be developed to establish a successful outreach program geared towards older adults. After analyzing poison center call history and U.S. Census data, interviewing key partners, and garnering feedback directly from two small groups of older adults, the Washington Poison Center developed an educational program with a focus on medication management and household safety tips for inter-generational families with materials created specifically for older adult caregivers and older adults themselves.

Results: Review of Washington Poison Center data revealed that 50% of all calls made by or on behalf of an older adult were due to a therapeutic error, and one partner identified medication management as the most prominent factor influencing an older adult's ability to maintain his or her independence.

Conclusions: Thus, targeted education and outreach to address medication management became a cornerstone of the program supported through the sharing of case examples. This poster will highlight the major conclusions of the Needs Assessment, present the new materials created for older adults and caregivers, as well as discuss the successes, challenges, and future directions of the program 12-months after initiation with the hopes of helping other poison centers develop new ideas and strategies to target the aging population.

KEYWORDS Older Adults; Medication Management; Education

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Substances (case number)	Codeine (292)	Hydrocodone (672)	Hydromorphone (8)	Morphine (65)	Oxycodone (151)	Oxymorphone (2)	Tramadol (303)	Total (1490)
Altered mental status	20	34	3	19	24	2	19	121
Respiratory depression	4	2	3	2	9	1	3	24
Seizure	1	0	0	0	0	0	2	3
Naloxone given	4	3	3	5	11	2	1	29
Intubation	0	0	0	0	2	0	1	3
Referred or sent to hospital	72	273	7	50	113	2	121	639
Admitted to floor	2	1	0	9	4	0	3	19
Admitted to critical care unit	2	2	3	8	6	2	2	25
Moderate + major effect	5	3	3	7	10	2	5	35

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