Chlorfenapyr Intoxication: a Fatal Case with Acute Pancreatitis

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Objective: Chlorfenapyr is a new agricultural insecticide and has been widely used since 1995. It is a pro-pesticide that means it changes to an active compound by the metabolism of liver. Chlorfenapyr intoxication has rarely been reported in human. We present herein a case with profound sweating, acute pancreatitis and eventually fatality after ingesting Chlorfenapyr.

Case report: An 80-year-old male presented to our emergency department because of abdominal pain and profound diaphoresis lasted for five days. Eight days earlier, he had attempted to commit suicide by ingesting about 200 mL of 10% Chlorfenapyr. He denied ingestion of other toxic substance or medicine. He didn’t go to hospital until this visit to our ED. Tracing back his history, he initially denied fever, diarrhea or vomiting, but had poor appetite and no stool passage for several days after ingesting Chlorfenapyr. His vital signs on arrival were: blood pressure 177/83 mmHg, heart rate 102 beats/minute, respiratory rate 26 breaths/minute and body temperature 36.1 °C. The physical examination revealed only mild epigastria tenderness and wet body. The laboratory results showed elevated lipase (2163 IU/L), aspartate transaminase (AST: 232 IU/L), alanine transaminase (ALT: 132 IU/L) and serum creatinine (Cr: 1.96 mg/dL). Other laboratory data including bilirubin were within normal limits. Acute pancreatitis was impressed and admission was suggested. However, the patient requested “Discharge Against Medical Advice”. Eventually, he died after eleven days of ingesting Chlorfenapyr.

Conclusion: Chlorfenapyr intoxication is uncommonly reported in human. It causes fever, diaphoresis, tachycardia, vomiting, altered metal status, rhabdomyolysis and abnormal liver function tests in one case series that composed of 26 Chlorfenapyr intoxication patients. Chlorfenapyr is a pro-insecticide that causes the interference of mitochondrial oxidative phosphorylation by disrupting the production of adenosine triphosphate. Body temperature regulation in warm-blooded animals is largely governed through hypothalamic regulation of sympathetic nervous system and mitochondrial oxidative phosphorylation. Chlorfenapyr intoxication causes hyperthermia and energy consumption due to breaking the production of adenosine triphosphate, that finally inducing cellular death, and organism mortality. One journal reported a death after the vapor exposure to Chlorgenapyr in 2007 and another reported central nervous system involvement with neuroimaging of Chlorfenapyr intoxication. Chlorfenapyr intoxication may cause any organ required high-energy
damage. To the best our knowledge, our case is the first report of Chlorfenapyr related acute pancreatitis and expired after 11 days of intoxication. This is not commonly seen in the patients with Chlorfenapyr intoxication in literature. As a result, physicians maybe consider acute pancreatitis if the patient has abdominal pain after ingesting Chlorfenapyr and alert “a latent period” which gives a false safe condition while initial examination was within normal finding.

References: