Massive parenteral manganese overdose: minimal role for hemodialysis

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Objective
Manganese-associated parkinsonism with brain MRI findings is well described in occupational exposures, among chronic methamphetamine users, and in patients receiving chronic total parenteral nutrition (TPN).1-3

We present the first reported case of acute poisoning by parenteral manganese administration with a systematic evaluation of hemodialysis efficacy.

Case Report
A 52-year-old woman underwent elective outpatient parenteral chelation with adjunctive vitamin and mineral therapy for perceived lead toxicity.

Through errors in prescribing, compounding, dispensing and administration, she was inadvertently administered 800 mg of compounded manganese chloride (4 mL of 200 mg/mL) instead of magnesium chloride.

The error was rapidly discovered, and the patient was sent to the hospital.

Upon arrival to the ED, the patient was asymptomatic, with normal vital signs and physical examination.

Her initial blood manganese concentration obtained six hours after exposure was 120 μg/L (2.19 μmol/L); normal 5 - 18 μg/L (0.09 μmol/L to 0.33 μmol/L). (Figure 1).

Blood manganese concentrations were measured by NMS Labs using inductively coupled plasma/mass spectrometry.

An attempt to minimize CNS manganese deposition and in the face of limited human pharmacokinetic data following acute parenteral manganese exposure,4 the patient underwent two hemodialysis sessions.

Hemodialysis
The patient underwent two (4-hour) sessions of intermittent HD at 7 and 21 hours after exposure.

Blood flow rate of 360 mL/min.
Dialysate flow rate of 700 mL/min.

The extraction ratio at hemodialysis onset was 0.28 and decreased to 0.10 at hemodialysis conclusion (Figure 2).

Following the first hemodialysis session, blood manganese concentration had decreased to 20 μg/L (0.36 μmol/L).

Analysis of dialysate from the first hemodialysis session revealed a total elimination of 604 mg (11 μmol) manganese, only 1.4% of total manganese burden.

An MRI obtained on hospital day two revealed T1 hyperintensities within the bilateral globi pallidi, consistent with manganese poisoning (Figure 3).

The patient also received five days of parenteral chelation with CaNa₂EDTA (1g/m2 over eight hours).

On day five, patient was discharged with a repeat blood manganese concentration of 2.2 μg/L (0.04 μmol/L).

At one-month follow up, the patient’s MRI was unchanged.

At eight-month follow up, patient remains asymptomatic with a normal neurologic examination.

Conclusion
Manganese poisoning is known to be associated with irreversible neurologic toxicity.

Hemodialysis did not appear to offer significant elimination benefit in the treatment of this case of acute parenteral manganese toxicity, beyond supportive care and chelation with CaNa₂EDTA.

References