DOUBLE TROUBLE: TRAUMA AND A TOXIN IN A VIRGINIA OPOSSUM

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CLINICAL BRIEF

A young adult female Virginia opossum (*Didelphis virginiana*) presented after being found on the side of the road. Significant head trauma was present characterized by depressed mentation, facial swelling, and bilateral epistaxis. Radiographs revealed multiple skull fractures involving the sagittal crest, both frontal bones, both zygomatic arches, and the caudal right mandible. Also noted radiographically were two 3mm diameter round metal objects present within dilated, gas-filled intestinal loops. The opossum was treated with supportive care including fluids, antibiotics, and analgesia.

To facilitate healing of the skull fractures and allow for nutritional support, an esophagostomy tube was placed. Feeding occurred solely through the esophagostomy tube for two weeks, and then the opossum was allowed to eat small amounts of soft food on its own. The tube was removed 16 days after placement with no complications. Serial radiographs of feces and fecal dissection were conducted to monitor for passage of the metallic objects. The two lead pieces were passed at days 24 and 38 after hospitalization.

Blood lead was 240 µg/dl. Treatment was initiated with dimercaptosuccinic acid (DMSA) at 6.7mg/kg PO q12h for 5 days. After the course was completed. The blood lead level had risen to 246 µg/dl. Due to the failure of response to the DMSA, parenteral therapy with calcium ethylene-diamine tetraacetate (CaEDTA) (20mg/kg subcutaneously q12h) was initiated for the next 6 weeks with an alternating schedule of 5 consecutive days of chelation followed by 5 days of no chelation. A recheck lead level was 53.1 µg/dl. The opossum was discharged to a licensed wildlife rehabilitator with a prescription for oral DMSA due to ease of administration. After six weeks of DMSA treatment, the opossum was re-hospitalized due to increasing lethargy, and the lead level was determined to be 49.7 µg/dl. Chelation therapy with CaEDTA was repeated as before for three weeks, after which the blood lead level was 18.1 µg/dl. The opossum was returned to the rehabilitator, remained clinically normal, and was released the following spring when weather conditions were appropriate.

DISCUSSION

The Virginia opossum is a nocturnal solitary marsupial ranging throughout most of the United States. It is an opportunistic generalist omnivore and may readily consume carrion. Metal accumulation has been reported in the livers of free ranging opossums at levels higher than other mammals in the same area. It can be assumed that this species would be prone to accidental ingestion of lead if feeding on a carcass that had been hunted using lead ammunition. It is presumed that this is the origin of the lead toxicosis in the opossum in this case. The exposure of avian scavenger species to lead by this manner has been well-reported, and is a known cause of decline of reintroduced populations of California condors. Lead present in the environment in the form of fishing tackle or ammunition poses a health risk to numerous wildlife species, and in some cases, animals may be sentinels for human lead exposure.

The most salient clinical features of this case included significant central nervous system (CNS) depression and severe gastrointestinal ileus. The neurologic signs may have been related to the acute trauma that resulted in the multiple skull fractures and epistaxis. Lead can also have numerous neurotoxic effects as well as alterations in behavior and coordination that may have predisposed this opossum to trauma. Changes in gastrointestinal motility during lead toxicity has been reported in other species, and is thought to occur due to alterations at the neuromuscular junction of visceral smooth muscle and altered intestinal motility and tone. The opossum in this case had severely dilated intestinal loops visible on radiographs at the time of admission which persisting until 11 days after intake. Thirty-eight days were required to pass both of the suspected lead foreign objects from the intestinal tract. Based on a reported transit time of 24 hours for the Virginia opossum, this confirms a prolonged rate of digesta passage. The persistence of the suspected lead objects in the gastrointestinal tract likely contributed to the slow response to chelation therapy due to continued lead absorption. The uncertainty of the exact location of the small metal fragments, combined with the risks associated with an exploratory surgery and enterotomy in an unstable patient, strongly influenced the decision to initiate chelation therapy and allow the objects to pass on their own.

The placement of an esophagostomy tube in this patient was key to successful management in this case. By bypassing the mouth and precluding the need for the opossum to chew food, nutritional support and oral medications were able to be administered while allowing the multiple skull fractures to heal. The procedure to place the tube was similar to that reported in small domestic animals, and was without complication. Both the food and the medications provided to this opossum were reduced in dosage by approximately 30% based on the low metabolic rate reported in marsupials compared to similarly sized placental mammals.

Chelation therapy was initiated in this case with DMSA, due to the ease of oral administration and reported efficacy in humans and other species. This five-day course failed to lower the blood lead level while the metallic objects were still in the gastrointestinal tract. DMSA was repeated after

passage of the metal fragments, and again, there was no effect on the blood lead level. Therefore, the DMSA failed to have any impact on the blood lead level in this opossum. It is possible that the metabolically scaled dose used in this opossum was insufficient, but the possibility that DMSA is ineffective as a lead chelator in the Virginia opossum must also be considered.

CaEDTA was successful in lowering the blood lead level in this opossum, however it required 7 rounds of chelation. The repeated rounds of chelation therapy may have been necessary due to the extremely high initial blood lead level, the length of time (~6 weeks) necessary for the suspected lead fragments to pass, and the cautious dosing used based on the patient's suspected low metabolic rate and poor clinical condition. CaEDTA can have adverse effects, including nephrotoxicity and binding of minerals such as zinc, iron, calcium, and copper. No adverse effects secondary to chelation therapy were seen in this opossum.

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