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CASE REPORT

Zinc Toxicosis in a Boxer Dog Secondary to Ingestion of Holiday Garland

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Abstract
Introduction Increased admissions occur in small animal veterinary emergency clinics during some holidays, and some of the increased caseload is due to ingestion of toxic substances. This report documents zinc toxicosis contributing to the death of a dog after ingestion of holiday tinsel garland.
Case study A mature boxer dog presented with a 4-day history of vomiting and diarrhea. Radiodense foreign material was detected in the stomach and removed via gastrotomy. The patient clinically worsened over the next several days with evidence of hemolytic anemia, severe hypernatremia, and an elevated WBC count with a suspected dehiscence of the surgical site and acute renal failure. The serum zinc concentration was moderately elevated. Postmortem findings included surgical dehiscence from the gastrotomy and enterotomy sites, hepatic extramedullary hematopoiesis, hemoglobinuric nephrosis, and pancreatic fibrosis. The foreign material removed from the stomach also contained zinc.
Discussion Ingestion of holiday tinsel garland made from metal-coated plastic film has not previously been implicated in zinc toxicosis. Zinc toxicosis has a good prognosis in veterinary medicine when diagnosed and treated promptly, but the unique source of zinc in this dog contributed to the delay in diagnosis and grave outcome in this case.

Keywords Dog · Zinc · Zinc toxicosis · Holiday · Emergency

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Introduction

There is evidence of increased caseload in small animal veterinary emergency clinics on or around certain holidays [1]. Companion animal poisonings, particularly methylxanthine toxicosis from chocolate ingestion, occur most frequently around Christmas, Valentine's Day, Easter, and Halloween [2]. Lily ingestion in cats is associated with the Easter holiday [3]. Holiday ornament-related injuries have been reported in the pediatric literature and are often secondary to ingestion of glass, though toxin exposure has also been noted [4]. This report documents zinc toxicosis resulting from ingestion of holiday garland by a dog.

Zinc toxicosis has been reported in humans and a variety of nonhuman animals and became more common in the USA after 1982, when the US Treasury ceased minting one-cent coins from copper alone and began using a zinc core [5]. Other reported sources of zinc include metal from galvanized hardware, toys, buttons, jewelry, fixtures, game pieces, and animal identification tags [5–10]. Sources of zinc salts can include zinc supplements, liquid zinc chloride, calamine lotion, and zinc oxide ointment [5–9]. The garland ingested by

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this dog was made from a plastic film coated by a thin layer of metal, in this case, zinc.

Case Study

A 4-year-old spayed female boxer dog weighing 22 kg (49 lbs) presented to the primary care veterinarian on December 16, 2015 with a 4 day history of vomiting and diarrhea. The dog and her owner had been staying with friends since December 12. The patient had a PCV of 54% (reference values (ref) 29 to 54%) and a distended stomach in which radiopaque material was observed on radiographs. The dog underwent gastrotomy and enterotomy. Abundant material resembling metallic tinsel garland was removed from the stomach and small intestines. Material was identified as a holiday tree decoration from the home in which the dog was staying. The dog was given antibiotics (Clavamox 13 mg/kg per os (PO) every 12 h and metronidazole 17 mg/kg PO every 12 h) and analgesics (tramadol 1.7 mg/kg PO, every 12 h).

The next day, the dog's urine was red and her PCV was 22%. She was transfused with 600 mL of whole blood and given dexamethasone SP intravenously at an immunosuppressive dose of 0.82 mg/kg, based on the tentative diagnosis of immune-mediated hemolytic anemia. The dog was put on a new antibiotic regimen of metronidazole 15 mg/kg intravenously (IV) every 12 h, ampicillin 5 mg/kg IV every 8 h, and cefazolin 28 mg/kg PO every 12 h to treat for potential urinary tract infection. Gastroprotectants were also given (maropitant 1 mg/kg subcutaneously every 8 h, famotidine 1 mg/kg subcutaneously once per day).

The dog became more obtunded over the next few days and, on December 19, her PCV was 12%; the plasma was dark red, and her urine was port wine in color, presumably secondary to hemolysis though urinalysis was not pursued. The prothrombin time of 15.6 s (reference values 13–19 s) and a prolonged partial thromboplastin time of 192.9 s (reference values 75 to 105 s). Transfusion was repeated, with the addition of vitamin K_1 (phytonadione, dose not known) and antibiotics. She was transferred to an internal medicine practice on December 21. She presented with an arched gait, cachexia, tachycardia, hyperkinetic pulses, pale, dry mucous membranes, and a low-grade systolic murmur consistent with anemia. The dog had clear urine, a PCV of 19%, total serum protein of 6.2 g/dL (ref 5.0 to 7.4 g/dL), albumin of 2.2 g/dL (ref 2.3 to 4.0), and mild azotemia, with a serum urea nitrogen (SUN) of 45 mg/dL (ref 7–25 mg/dL) and a creatinine value of 2.4 mg/dL (ref 0.1 to 1.5 mg/dL). Additionally, her alkaline phosphatase (ALP) was 688 u/dL (ref 23–212 U/L) and her alanine transferase (ALT) was 134 U/L (ref 10–25). The serum sodium concentration was elevated at 160 mmol/L and increased over 1 day to 172 mmol/L (ref 144–160 mmol/L). Based on an automated complete blood cell count, she had a leukocytosis of 46.13 K/μL (reference values 5.05 to 16.76 K/μL), with 77% neutrophils and 10.9% lymphocytes. Urinalysis revealed a pH of 5.5, 2+ protein; the presence of erythrocytes and a specific gravity of 1.024, which, in the presences of azotemia, hypernatremia, and dry mucous membranes is consistent with renal failure and secondary dehydration. A serum sample was taken at this time for zinc analysis. The patient became more obtunded and ataxic the next day and was euthanized due to suspected dehiscence of the gastrotomy or enterotomy site, and a hypoechoic pancreas on ultrasound.

Post mortem examination took place on December 22. There were two spaces, 2 mm in diameter, along the sutured gastric incision with leakage of gastric contents, consistent with perimortem surgical dehiscence, and mild fibrinous peritonitis. Histologic examination of the gastric wall revealed necrotizing and fibrinous transmural gastritis with intralesional foreign material consistent with suture. Microscopic examination of the kidneys revealed red to orange intratubular casts consistent with hemoglobinuric or myoglobinuric nephrosis. Chronic fibrosing pancreatitis and transmural gastritis were also noted. The serum zinc concentration from the sample taken on December 21 was reported as 7.03 ppm (ref 0.70 to 2.00 ppm) via inductively coupled plasma atomic emissions spectroscopy (ICP-AES). Samples of garland from the home were collected and analyzed using X-ray fluorescence spectroscopy (XRF) and it was determined that the metallic garland contained zinc, which was quantitated at 9% based on ICP-AES analysis.

Discussion

Zinc toxicosis occurs occasionally in humans, dogs, and various other species associated with ingestion of foreign material containing zinc. The tinsel garland in this case was made of material resembling polyvinyl chloride or polyethylene terephthalate film with a metallic finish, similar to Mylar®. Zinc was most likely used in this product because it has an attractive metallic luster and is relatively inexpensive.

Metallic zinc reacts with gastric hydrochloric acid to form soluble zinc chloride salt [10]. Zinc salts are absorbed within the duodenum [11]. Circulating zinc is bound to serum proteins, including β2-macroglobin and albumin [12]. Zinc distributes to the pancreas, liver, and kidney [5, 11]. Fecal excretion predominates, with about 25% excretion through pancreatic juice and approximately 50% through bile [8, 9]. Some zinc is also excreted through urine and saliva [12]. The reported median lethal oral dose for zinc in dogs is 100 mg/kg "zinc salt" [12]. The toxicity of zinc is influenced by numerous factors, including the chemical form, nutritional status of the patient, and other patient factors [13]. Assuming that the toxicity of metallic zinc is similar, since the dog was 22 kg and the garland contained 9% zinc, the dog would need to ingest approximately 24 g of the garland to reach a median lethal dose.

The mechanism of zinc toxicosis is incompletely understood. Zinc salts cause direct irritation of the gastrointestinal mucosa [11]. Intravascular hemolysis is most likely due to damage to the erythrocyte membrane rather than hemoglobin, since Heinz body formation is only noted in approximately one third of cases [6, 7]. There have been several proposed mechanisms for the oxidative damage to erythrocytes: glutathione depletion, effects on the hexose-monophosphate pathway, or hapten formation [6, 7, 11]. Impairment of copper absorption has been implicated as a cause of anemia in chronic cases of zinc toxicosis [6]. Common clinical signs in acute cases, both in humans and dogs, include hemolytic anemia, fever, anorexia, salivation, vomiting, and diarrhea [6, 7, 10, 11]. Additionally, severely affected individuals can have icterus, pigmenturia, renal failure, and pancreatitis [5, 6].

Anemia in dogs with zinc toxicosis is regenerative [6–8, 11]. Heinz bodies, as noted, are documented in about a third of the cases. Spherocytes are present in low numbers in about 20% of canine patients with zinc toxicosis, which can be helpful in distinguishing this diagnosis from immune-mediated hemolytic anemia [6]. As seen in this patient, left-shift leukocytosis is a common finding in dogs with zinc toxicosis but also could be due to early dehiscence at the surgical site [7, 8, 11]. Azotemia is commonly reported in dogs and people with zinc toxicosis as a consequence of hemoglobinuric nephrosis [5, 7, 8, 11]. Elevated bilirubin and liver enzymes are also frequently reported in both dogs and humans [5, 6, 11]. Elevated lipase and amylase, signifying pancreatic damage, are often reported in affected people and dogs, and the pancreas is most likely targeted because pancreatic juice is an excretory pathway for zinc [11]. Hemoglobinuria, proteinuria, hematuria, and, occasionally, granular casts in the urine are expected findings in dogs with zinc toxicosis [6, 7].

Reference values for serum zinc in dogs range from 0.7 to 2.0 ppm. Serum zinc concentrations above 5 ppm, as in this case, can support the diagnosis of zinc toxicosis [14]. The serum zinc concentration in this patient was 7 ppm; however, serum was not collected for analysis until five days after removal of the gastrointestinal foreign body. It was likely that the circulating zinc concentration was much higher in this patient at the time of surgery. Treatment involves removal of the source of zinc, usually a metallic foreign body, from the gastrointestinal tract [6–8, 10]. Serum zinc concentrations decline after gastrointestinal exposure is ended, thus chelation therapy is rarely warranted [7, 8]. Dramatic decreases in serum zinc concentration after foreign body removal have been documented in dogs in previous case reports, which found a decline from 28.8 to 16.8 ppm within 48 h [8] and from 28.6 to 6.78 ppm within 48 h [7].

Supportive care for hemolytic anemia associated with zinc toxicosis can include transfusion, as was performed in this case [6–8, 10]. Hydration and renal support to maintain electrolyte balance and protect against hemoglobinuric nephrosis are also frequently recommended [6, 8, 9]. Gastroprotectants are often used due to the gastrointestinal irritant properties of zinc salts but can also decrease gastric acid production and therefore the bioavailability of metallic zinc [10]. Calcium EDTA and penicillamine are the most commonly used when chelating agents are indicated for zinc toxicosis in domestic animals [7]. We do not know what the peak serum zinc concentration in this dog was, but earlier diagnosis and chelation therapy in this case may have prevented hemolysis and subsequent renal damage.

Postmortem changes associated with zinc toxicosis in dogs include icterus, centrilobular to diffuse hepatocellular degeneration and necrosis, hemoglobinuric nephrosis with tubular epithelial degeneration, and pancreatic necrosis and fibrosis [11]. Because the pancreas is a route of zinc excretion, pancreatic lesions can be prominent in cases of zinc toxicosis. Although hepatopathy was not described in this case, zinc may have contributed to the pigmenturia, renal disease, and pancreatitis that were noted.

The prognosis for zinc toxicosis is usually good with prompt diagnosis and treatment [6]. This case had a poor outcome: the patient was euthanized, a decision made in part due to surgical dehiscence at the gastrotomy and enterotomy sites, which was confirmed at necropsy. Zinc is known to cause gastrointestinal irritation, which, combined with hypoxia related to anemia and the early treatment with corticosteroids based on the presumptive diagnosis of immune-mediated hemolytic anemia could have contributed to the surgical dehiscence [11, 15]. Single-dose corticosteroid use is not known to impair healing, but corticosteroids have been associated with gastric mucosal damage in critically ill human patients and can impair immune system function, increasing the risk of infection [15, 16]. Diagnosis in this case was delayed because the foreign material ingested by the dog was not known to contain zinc. Zinc toxicosis resulted from ingestion of tinsel garland made from metal-coated plastic film. It is also possible that similar plastic film products with metallic surfaces also contain zinc. Based on the amount of garland ingested, dogs are probably more likely to be affected than other companion animals or small children.

Conclusion

Zinc toxicosis has a good prognosis in veterinary medicine when recognized early and treated promptly—the unique source of zinc in this dog contributed to the delay in diagnosis and poor outcome in this case.

Compliance with Ethical Standards

Conflict of Interest None

Sources of Funding None

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