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Suspected Canine Zinc Toxicosis Caused by Ingestion of Pennies

Thomas C. Carlson
Iowa State University

John G. Devries
Oradell Animal Hospital

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A 21 pound, nine year old spayed female Shih Tzu was presented to the Oradell Animal Hospital at 3 a.m. on Sunday morning with a complaint of anorexia, lethargy and vomiting for the past two days. The dog's previous medical history was unremarkable, except for being hit by a car in November 1983. A right ilial fracture sustained at that time had been plated. The animal was current on all routine vaccinations.

Physical examination revealed a weak, but alert, patient with normal temperature, pulse and respiratory parameters (T=102, P=120 bpm, R=20 rpm). The animal's mucous membranes were, however, markedly icteric and capillary refill time was greater than two seconds. The patient was admitted and samples were obtained for complete blood count, biochemical profiles, packed cell volume (PCV) quantification and total protein analysis. Attempts to obtain a urine sample via cystocentesis were unsuccessful. A whole blood smear showed spherocytosis with an adequate platelet count. Initial hemotological abnormalities indicated a marked anemia (PCV=15.0%) and elevated total plasma proteins (TPP=8.0g/dl). The serum was markedly hemolyzed.

Abdominal radiographs obtained at admission showed a foreign body of metallic density in the area of the stomach (Figure 1). Upon questioning, the owner indicated that some loose change was missing from the nightstand in an area the dog frequented. A presumptive diagnosis of a zinc-induced hemolytic crisis was made and a gastrotomy planned.

Presurgically, a transfusion of 200 mls of fresh, unmatched, whole blood was initiated and Keflin, (Eli Lilly and Co., Indianapolis, IN) 200 mg intravenously, was administered. Once the patient appeared clinically stronger, surgery was performed and four pennies of post 1982 mintage were removed via gastrotomy. The remaining contents of the abdominal cavity appeared grossly normal. Immediately following surgery the patient's PCV and TPP were 21.0% and 8.0 g/dl, respectively. Following completion of the blood transfusion several hours later, the same parameters were 32.0% and 8.4 g/dl. A lactated ringer's solution supplemented with B vitamins was then initiated at a maintenance rate.

Intravenous antibiotics identical in nature and dose to the presurgical administration were given TID until the fluid therapy was discontinued.

The patient eagerly ate and kept down soft baby food on day three, and the intravenous fluids were discontinued at that time. The dog was then placed on oral Keflex (Eli Lilly and Co., Indianapolis, IN) 250 mg TID. Results of the CBC and biochemical profile were also obtained on day three and indicated a strongly regenerative anemia, PCV - 15.0% with a reticulocyte count of 20.6%. Hypochromasia, microcytosis, anisocytosis and poikilocytosis were also noted. The patient had leukocytosis, (WBC - 38,300/ul), with a hyper proteinemia, (TPP - 7.5 g/dl), azotemia, (BUN - 106 mg/dl), and elevations in serum alkaline phosphatase, (332 IU/l), amylase,( 5390 IU/l), and lipase, (9350 IU/l). At the owner's request, followup hematological screens were not performed.

Packed cell volume values were obtained twice daily until day four and never fell below 30.0%. The dog's mucous membranes also became progressively less icteric until a normal pink color had been established. The patient was discharged on day four with a seven day course of the oral antibiotic mentioned above. The patient's subsequent medical history has been unremarkable.
Discussion

This patient’s most striking physical examination finding was marked icterus. With this fact established, one must then begin to search for a source of this symptom: i.e. is the icterus of prehepatic, hepatic or posthepatic origin? The low PCV of this patient in combination with the hemolyzed serum sample gave strong support to the diagnosis of a hemolytic crisis, i.e. a prehepatic jaundice was surmised. Hemolysis-induced jaundice is produced when the metabolism of destroyed red blood cell contents exceeds the liver’s processing capabilities. Thus levels of unconjugated bilirubin, a major by-product of heme metabolism, increase. This situation manifests itself via the classic yellow hue of this material being visible in body tissues, most evident in the mucous membranes.

The next diagnostic step dealt with the identification of the etiology behind the hemolysis. Zinc-induced hemolysis caused by the ingestion of pennies was the presumptive diagnosis in this case. Support came from findings of the physical examination, hematological abnormalities, results of abdominal radiographs, and testimony from the owner that some change was indeed missing from an area to which the dog had access. The diagnosis was supported by the findings at surgery, as well as the patient’s clinical recovery. Serum and tissue zinc levels would have been further supportive of this diagnosis but, unfortunately, were not obtained.

Zinc-induced hemolysis in the dog is an incompletely understood syndrome which has not been commonly reported. Perhaps this is due to the fact that this condition previously went unrecognized or because potential sources of exposure to zinc have increased in recent years. Both of these explanations may, in fact, be valid. In the case of this patient, the source of the zinc was pennies which had been ingested and thus exposed to the acidic digestive environment of the stomach. Zinc-induced hemolysis from such a source poses a much greater threat from such coins minted after 1982. At that time the composition of the Lincoln penny was dramatically altered to reflect the rising price of copper. It is not unreasonable to equate the modern penny as essentially a copper-coated zinc disc.

The weight of pennies removed from the gastrointestinal tract may not accurately reflect the amount of zinc released, since zinc may be replaced by other elemental salts in the presence of an acidic environment. Also, the condition of the coin’s jacket may be misleading as to the amount of this metal released, as zinc has been reported to leech from the interior of pennies even when the coin’s surface is unabraded.

Sources of zinc other than pennies have been reported to have caused hemolytic episodes in dogs. Ingestion of topical zinc-oxide dermatological ointments as well as zinc nuts used to secure pet transport cages have recently been implicated in this syndrome.

While the exact mechanism of zinc-induced hemolysis has not been determined, it is known that erythrocyte fragility is affected by elevated zinc levels via inhibition of a number of enzymatic pathways. Besides hemolysis, zinc has been known to cause lesions in other body systems. Hepatic renal changes reported in cases of zinc toxicosis were consistent with centrilobular cellular vacuolization with hepatocytic necrosis, and protein deposition in glomerular capillaries with associated renal tubular casts. Hematological parameters dealing with the liver and kidneys are thus often elevated, as are serum lipase and amylase which are themselves cleared through the kidney. Such values were evidenced in this case.

Conclusion

The definitive diagnosis of zinc toxicosis requires documentation of serum and tissue zinc levels significantly elevated above established normals. Treatment of this condition involves removal of the inciting agent and, when indicated, further supportive measures such as whole blood transfusions and fluid therapy. The hemodynamic changes seen in zinc toxicosis may be remarkably similar to those seen in immune-mediated hemolytic anemias. One source reported initial misdiagnosis of autoimmune hemolytic anemia in three dogs actually suffering from zinc toxicosis. As zinc foreign bodies may be easily identified radiographically, and because of the potentially fatal nature of this syndrome, abdominal radiographs should be considered prudent in any case where the patient presents with signs consistent with hemolytic anemia.

Iowa State University Veterinarian
References


Figure 1