

Zinc Toxicosis from penny ingestion in dogs

by

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It is common for pets, especially dogs, to ingest coins. Among the circulating U.S. coins, only pennies pose a toxicity hazard. Pennies minted after 1982 contain a zinc core surrounded by copper plating. Each penny contains about 2,440 mg of elemental zinc¹. Subacute or chronic zinc toxicosis can affect the renal, hepatic, gastrointestinal, and hematopoietic tissues.¹⁻³ Zinc toxicosis can cause hemolytic anemia, which can lead to hemoglobinemia and hemoglobinuria.¹⁻³ Because of these severe effects, consider all penny ingestions potentially dangerous, and treat each case aggressively.

Mechanism of Action

The stomach's acidic environment results in rapid release of zinc from ingested pennies.² The release rate depends on the stomach's pH, the presence or absence of food, and the length of time the pennies are in the stomach.² Once absorbed, zinc is transported to the liver largely bound to plasma proteins. Zinc is primarily excreted through bile, pancreatic secretions, and gastrointestinal mucosal cells, with less than 25% excreted in the urine.^{2,3} The toxic dose of zinc in dogs is unknown.

The exact mechanism of zinc-mediated hemolysis is also unknown.³ Possibilities include direct damage to red blood cell membranes, damage to red blood cell organelles, immune-mediated destruction from hapten formation, or inhibition of specific red blood cell biochemical mechanisms.^{2,4} Similarly, the mode of zinc-mediated renal injury is not fully understood. Renal damage may occur secondary to anemia, hypoxia, or hemoglobinemia. Or zinc may directly injure renal tubular epithelium, similar to renal injuries seen in other heavy metal toxicoses.⁴

Clinical Signs

Since most penny ingestions are not witnessed, the time between ingestion and the onset of clinical signs is not well-documented. Signs of zinc toxicosis in dogs include icterus, anorexia, vomiting, hemolytic anemia, hemoglobinuria, and renal failure.⁵ Clinical laboratory abnormalities usually suggest hemolysis (*e.g.* bilirubinemia, bilirubinuria, hemoglobinemia, hemoglobinuria) and may indicate kidney failure (*e.g.* azotemia, hyposthenuria). Nucleated red blood cells, basophilic stippling, target cells, polychromasia, or evidence of disseminated intravascular coagulation (*e.g.* anemia, thrombocytopenia, increased fibrin degradation products, increased clotting times) may be seen.² Increased pancreatic and liver enzyme activities could occur but are not commonly reported with penny ingestion. Death may result from severe anemia or multiorgan failure.² Differential diagnoses for hemolytic anemia other than zinc toxicosis include autoimmune hemolytic anemia, onion poisoning, and other causes of oxidative injury to the red blood cells.

In a three-year period (January 1998 to December 2000), 18 cases of presumptive zinc toxicoses in dogs ingesting pennies were reported to the ASPCA Animal Poison Control Center (APCC). Reported clinical signs included anemia (72%), depression (66%), vomiting (61%), hemolysis (33%), and hemoglobinuria (22%). Renal effects, such as azotemia, polyuria, and polydipsia, were reported in 22% of the cases. Other signs include anorexia, diarrhea, pale mucous membranes, icterus, and abdominal pain.⁶

Diagnosis

The diagnosis of zinc toxicosis by penny ingestion is based on clinical signs and radiographic identification of coins. Pennies may lodge in the gastric lining but may also be found throughout the intestinal tract, including the distal colon.

Measuring plasma or urine zinc concentrations can help diagnose zinc toxicosis and is particularly useful in monitoring treatment if chelation therapy is used. Ideally, collect samples in royal-blue-top tubes to avoid

zinc contamination, since rubber stoppers and syringes may contribute up to 4 ppm of zinc.⁴ If these are not available, use heparinized glass blood collection tubes, or plastic serum tubes, since they contribute less extraneous zinc than EDTA blood tubes.⁴ Normal serum and urine zinc concentrations in dogs are 0.7 to 2 ppm, and zinc concentrations in cases of toxicosis are usually above 10 ppm.^{2,4}

Histopathologic changes seen postmortem include tubular nephrosis, centrilobular to midzonal hepatic necrosis, and fibrosis.³ Zinc concentrations can be determined from kidney, liver, and pancreatic tissue in postmortem examinations.

Treatment

Patient stabilization is the first priority in cases of zinc toxicosis. Administer oxygen in dyspneic patients and lactated Ringer's solution intravenously at 90 ml/kg to restore vascular volume if a patient is in shock.⁷ Blood transfusions or bovine hemoglobin glutamer (Oxyglobin-Biopure) supplementation is needed to increase the oxygen-carrying capacity if a patient's packed cell volume is less than 10% or if clinical signs of severe anemia are present (*e.g.* severe weakness, heart murmur, tachycardia, orthopnea).^{8,9} Discoloration of mucous membranes can occur after bovine hemoglobin glutamer administration and may hinder visual evaluation of hemolysis.¹⁰ Other treatment goals include decreasing zinc absorption, correcting anemia, minimizing renal damage, and removing the penny.

Removing the penny from the gastrointestinal tract is critical, though dogs may die as a result of complications from anemia or kidney failure even after a penny is removed. Penny removal may require surgical or endoscopic retrieval.^{2,9} But nonsurgical removal by inducing emesis may be successful in asymptomatic patients soon after penny ingestion. Administer an H₂-receptor blocker, such as famotidine (0.5 mg/kg orally, subcutaneously, intramuscularly, or intravenously once or twice a day) to reduce zinc absorption by decreasing gastric acidity.^{2,11} Activated charcoal is not recommended because it is ineffective in adsorbing elemental zinc.¹

Chelation therapy for zinc toxicosis is controversial. In general, zinc concentrations are expected to decline without chelation after the zinc source is removed from the gastrointestinal tract. Failure of the zinc concentration to decline after penny removal suggests that there is still zinc in the gastrointestinal tract or that renal insufficiency is preventing zinc excretion. In either of these situations, chelation is contraindicated, since chelating agents may increase gastrointestinal absorption of zinc, and the chelated metal is potentially nephrotoxic.^{2,11}

After penny removal, obtain radiographs to make sure all the pennies have been removed, and continue intravenous fluids to maintain hydration, correct electrolyte imbalances, and preserve renal function.⁹ The fluid choice depends on the patient's electrolyte status. Fluid therapy should be temporarily discontinued during bovine hemoglobin glutamer infusion, since its volume-expanding properties may lead to circulatory overload, especially when you use colloidal fluids.¹⁰ Monitor blood urea nitrogen, creatinine, and phosphorus concentrations; packed cell volume; and urine output and specific gravity throughout treatment. Clinical signs of renal failure include lethargy, anorexia, vomiting, polyuria, polydipsia, oliguria, and anuria.¹² Peritoneal dialysis is recommended for anuric patients. Administering dopamine (2 to 5 µg/kg/min) may increase renal perfusion.¹¹

If the tubular basement membrane remains intact and sufficient viable epithelial cells are present, tubular lesions can recover.¹² Consider administering sucralfate (0.5 to 1 g orally b.i.d. to t.i.d.) as a gastrointestinal tract protectant and metoclopramide hydrochloride (0.2 to 0.4 mg/kg orally or subcutaneously t.i.d. to q.i.d.) to control vomiting.¹¹ Supportive care may be needed for several days to weeks until the patient fully recovers. The prognosis depends on the severity of clinical signs and the patient's response to treatment.

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