AT RISK
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MANAGEMENT STRATEGIES

Lead and Zinc Toxicity in Birds

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Birds are extremely sensitive to changes in the environment, so much so that they have been used as sentinels for poisonous gas detection in coal mines and heavy metal, toxin, and/or chemical detection in bodies of water.[1,2] They are sensitive to heavy metals (e.g., lead, zinc, cadmium, copper, mercury, iron) in their environment. Toxicity occurs when ingested metal causes harm to the animal. The heavy metals that most commonly cause toxicity are lead and zinc; less common causes are mercury and copper.[1,3] Lead is a mineral not normally present in the body, but zinc is present in small quantities.[4] Thus, zinc toxicity results from chronic and/or repeated exposure.

Sources of metal include weights (including fishing weights), batteries, pellets, paints, galvanized wire, costume jewelry, ceramics, contaminated material, hardware, post-1982 pennies, stained glass, food/water bowls, galvanized wire cages, and toys.[1,3,5] In particular, lead can be found in house paints manufactured before 1978,[1] and zinc toxicity most commonly comes from post-1982 pennies, zinc-coated wire, galvanized cages, and/or other metallic foreign bodies.[4-6]

This article focuses on lead and zinc toxicity in birds and describes case management.

SIGNALMENT

Heavy metal toxicosis is typically more severe in birds that are very young, very old, and/or compromised in some way. The most commonly affected species are psittacines, waterfowl, and raptors that have eaten prey containing lead shot.[1]

Lead is considered to be the most commonly reported of avian toxicosis in both captive and free-ranging birds.[4] Waterfowl and raptors more commonly ingest lead shot (in prey or fishing gear) which leads to acute poisoning.

FIGURE 1. Polyuria from a bird with heavy metal toxicity.

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PRESENTATION

Clinical signs of heavy metal toxicity in birds are nonspecific and often difficult to differentiate from other causes. However, heavy metal toxicity should be a top differential for birds showing neurologic signs. Heavy metal toxicity also becomes a more likely differential if the history reveals that the bird has chewed on walls or baseboards, new toys, or enclosures.

Lead toxicity: Lead is relatively insoluble, and the acidity of the proventriculus along with the mechanical action of the ventriculus can increase absorption into soft tissue and bone.[4,6] Clinical signs of lead toxicosis can be multisystemic and affect the gastrointestinal, renal, urinary, and/or nervous systems and can cause gradual weight loss, polyuria/polydipsia, depression, weakness, anorexia, and/or rapid anemia[1,4,6] (**FIGURE 1**). Birds can exhibit seizures or other central nervous system clinical signs of toxicity. Hemoglobinuria has been reported for affected Amazon parrots.[1,4] Lead toxicosis can also cause a die-off of populations. Waterfowl, which may ingest fishing gear (e.g., lead weights, baits), can exhibit weight loss, neck and limb weakness, and bright green feces[4] (**FIGURE 2**). Because lead toxicity occurs when lead is ingested, toxicity typically does not develop in wild birds shot with lead bullets.

Zinc toxicity: Clinical signs for zinc toxicosis include passive regurgitation, lethargy, weakness, weight loss, anemia, cyanosis, polyuria/polydipsia, diarrhea, regurgitation, neurologic signs, feather picking, anorexia, and/or hemoglobinuria.[1,4,5] Cases of Amazon parrots with "chocolate milk" diarrhea and frank blood (hematuria) have been reported[6] (**FIGURE 3**). Gastrointestinal ulceration can occur secondary to zinc toxicosis, possibly resulting from irritation and damage as the foreign material is ingested.[4] If the gastrointestinal tract is perforated, the bird usually dies of sepsis and shock within 6 to 12 hours. In cockatiels, exposure to 2 mg zinc per week can be fatal.[5]

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DIAGNOSIS

Because the routes of heavy metal exposure are typically external (physical contact) and/or consumption, tissues that can be used to assess concentrations of metals include feathers, egg, liver, kidney, and others. The metal content of feathers can be used to monitor environmental contamination.[2] A tentative diagnosis can be based on history of exposure, clinical signs, laboratory results (mild anemia), and/or radiographs.[4,6] Lead is best measured in liver and bone.[1]

Baseline complete blood count of a bird with heavy metal toxicosis would show leukocytosis and hypochromic regenerative anemia.[1,3,4,6] Blood chemistry might show elevated levels of lactate dehydrogenase, aspartate aminotransferase, bile acids, and creatine phosphokinase. Birds with zinc toxicosis may also have elevated lipase, amylase, and uric acid levels.

FIGURE 2. Green feces from a bird with liver damage.

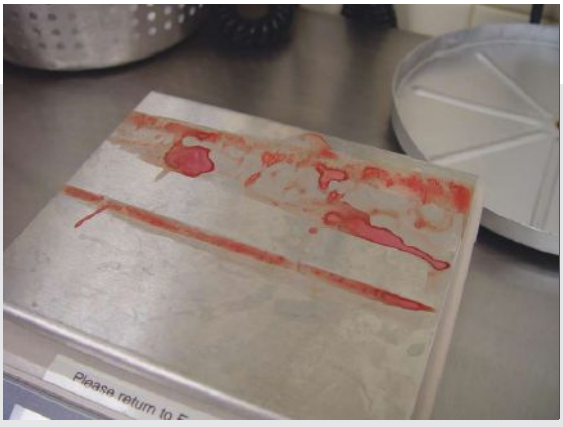

FIGURE 3. Hematuria from a bird with heavy metal toxicity.

Radiographs can reveal metal opacity in the gastrointestinal system, especially the ventriculus, but radiographic findings are not definitive[1,3,4] (**FIGURE 4**). Definitive diagnosis is based on postmortem examination of the pancreas, liver, and kidneys. The organ of choice to test for zinc is the pancreas[1,5]; pancreatic zinc levels greater than 1000 mcg/g are suggestive of toxicosis.[4]

More specific testing can also be performed. In heparinized whole blood, 20 mcg/dL (0.2 ppm) lead is suggestive of lead toxicosis, and a concentration of 50 mcg/dL (0.5 ppm), paired with clinical signs, is diagnostic.[1,3,4] Point-of-care lead testing machines are available for around $3300. Zinc serum/plasma levels can range from 2.5 to 29 ppm. Zinc serum/plasma levels as low as 2.5 ppm can cause clinical signs; 200 mcg/dL (2.0 ppm) is suggestive of toxicosis, and levels of 4.0 ppm are even more suggestive.[1]

TREATMENT

Treatment will vary according to whether the toxicity is acute or chronic. Treatment typically starts with stabilization/supportive care, prevention of further exposure, and delay of further absorption, all of which should be tailored to the patient's clinical signs and may include gastric lavage, antagonist therapy, and toxicant removal.[4,5]

Supportive care: Supportive care includes controlling seizures and correcting anemia, immunosuppression, and anorexia. Supportive care includes administration of fluids (50 mL/kg SC or 10 to 25 mL/kg over 5 minutes as a bolus IV or intraosseously (IO) or at a continuous rate (100 mL/hr/day IV or IO), a heated environment (85°F to 95°F), and nutritional support.[1,3] If the patient is severely anemic, a blood transfusion may be warranted. If the patient is stable, removal of the toxicant, if present, may not be necessary.

Medical treatment: Medical treatment includes anticonvulsants, fluid therapy, chelation, nutritional support, and antibiotics/antifungals. Antiemetic and/or prokinetics such as metoclopramide may also be indicated.[3,4] Laboratory testing should be monitored during treatment and checked a few days after completion of treatment. Anticonvulsants include midazolam or diazepam given at 0.5 mg/kg IV, IM, or IN; midazolam is the preferred drug for IM administration.

The indication for chelation is based on severity, and supportive care is indicated. Calcium disodiumversentate ($CaNa_2EDTA$) can be given at various dosages: 20 to 70 mg/kg IV diluted in sterile saline to a 4 mg/mL solution given q12h or q8h or 30 mg/kg SC given q12h until diagnosis is confirmed.[7] Chelation is the preferred treatment and can also be given at 30 to 50 mg/kg IM or IV for 3 to 5 days, stopped for 5 to 7 days, and repeated as needed.[1,3-5,7] Dimercaptosuccinic acid (DMSA/succimer) is a chelator that can decrease lead plasma levels and is given at 25 to 35 mg/kg PO q12h for 7 to 10 days or at 25 to 35 mg/kg PO q12h for 5 days a week for 3 to 5 weeks. Doses 80 mg/kg or higher are lethal.[1,3,4,6,7] $CaNa_2EDTA$ (the preferred initial chelator) and DMSA are both effective, but the DMSA range of safety is smaller.[1,2] D-penicillamine is another oral chelator that is given at 50 to 55 mg/kg PO q12h for 1 week, stopped for 1 week, and repeated

FIGURE 4. Radiograph of bird with metal in its gastrointestinal tract **(A)** compared to a radiograph of a bird with normal gastrointestinal content **(B)**.

if needed along with $CaNa_2EDTA$.[4,7] D-penicillamine can be given at 30 to 55 mg/kg PO q12h for 7 to 14 days. The main adverse effect is gastrointestinal upset.[1,3,7] Long-term chelation can deplete zinc, iron, and/or manganese, so these values should be monitored during and after chelation.

For other toxicoses, hypercalcemia induced by lead toxicosis can be treated with disodium EDTA (Na_2EDTA).[1] Cadmium toxicosis can be treated with $CaNa_2EDTA$. And mercury toxicosis can be treated with DMSA, vitamin E, and/or selenium.[1,4]

Source removal: To prevent further toxicosis, the source of the heavy metal needs to be removed and/or the patient needs to be prevented from chewing the source.[6] If metal objects need to be removed, surgical procedures are indicated, but typically the need is not urgent if the bird improves after chelation therapy.[1,3,4] Objects can be removed endoscopically (**FIGURE 6**), surgically, or with gastric lavage.

For gastrointestinal evacuation you can use bulk diets, lubricants, and/or cathartics (e.g., lactulose, mineral oil, barium sulfate, magnesium sulfate, or psyllium). Psyllium or other oral cellulose products (e.g., sodium sulfate) can be used at 125 to 250 mg/kg.[4] Lubricants do not work well in waterfowl. For zinc-coated objects, a neodymium-ferro-boron alloy magnet attached to enteral tube can be used for removal.[4]

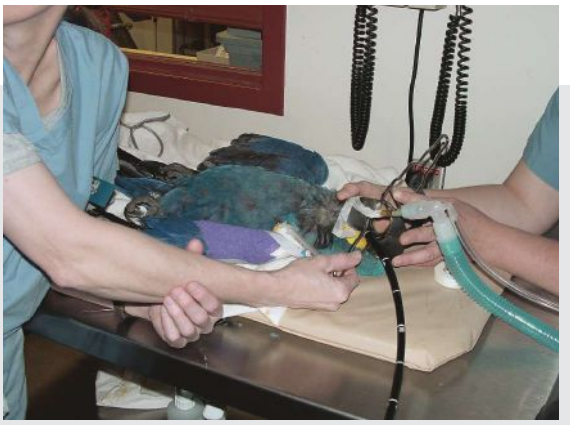

FIGURE 6. The bird is prepared to have heavy metal removed from the upper gastrointestinal tract via endoscopy.

PROGNOSIS

Prognosis depends on the patient's response to treatment. Severely affected birds may be dehydrated and in hypovolemic shock, thereby contraindicating initial chelation therapy.[3] If the patient responds well to initial treatment and the source of toxicity is removed, prognosis is generally good. However, if the patient does not respond to treatment or its neurologic status is poor, the prognosis is guarded to poor. Chronic exposure can lead to permanent neurologic, and sometimes gastrointestinal, damage. TVP

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