MERCK MANUAL
Veterinary Manual

Lead Poisoning in Animals

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Lead poisoning in mammalian and avian species is characterized by neurologic disturbances, gastrointestinal upset, hematologic abnormalities, immunosuppression, infertility, and renal disease. The nature of the clinical manifestations is influenced by the dose and duration of lead exposure. Chelation therapy, cathartics, thiamine administration, and sedation are common treatment methods. In food-producing species, treatment is discouraged because of food safety concerns, the prolonged treatment period, permanent degenerative damage, and a poor prognosis. Tissue analysis for lead supported by pathologic abnormalities is essential to confirm the diagnosis.

Lead poisoning in animals and humans is a major concern worldwide. Poisoning in animal populations may serve as a sentinel to assess the extent of environmental contamination and human health problems related to lead. In veterinary medicine, lead poisoning is most common in dogs and cattle. Young animals, pica, and greater accessibility to lead are key risk factors associated with the toxicosis. Lead poisoning in other species is limited by reduced accessibility, more selective eating habits, or lower susceptibility. In cattle, many cases are associated with seeding and harvesting activities when used oil and battery disposal from machinery is handled improperly. With the elimination of tetraethyl lead from gasoline in many countries, the number of lead poisoning cases attributed to oil consumption has declined. Other sources of lead include paint, linoleum, grease, lead weights, lead shot, and contaminated foliage growing near smelters or along roadsides.

Lead poisoning is also encountered in urban environments and during the renovation of old houses that had been painted with lead-based paint, leading to exposure of small animals and children. Recent evidence in backyard chicken flocks has documented elevated tissue concentrations of lead in eggs associated with the ingestion of paint flecks. Because poultry are much more resistant to clinical lead poisoning compared with most mammalian species, the deposition of lead in eggs with no apparent clinical disease in the bird may be substantial and may pose a potential public health risk in these unregulated flocks.

The consumption, through grooming, of dust containing lead has been reported in cats. Improper disposal of lead-poisoned animal carcasses may result in toxicoses in nontarget scavenger animals. Scavenging by endangered species such as the condor is a concern. To prevent future occurrences of lead poisoning, it is crucial to identify the source.

Pathogenesis of Lead Poisoning in Animals

Absorbed lead enters the blood and soft tissues and eventually redistributes to the bone. The degree of absorption and retention is influenced by dietary factors such as calcium or iron levels. In ruminants, particulate lead lodged in the reticulum slowly dissolves and releases significant quantities of lead. Lead has a profound effect on sulfhydryl-containing enzymes, the thiol content of erythrocytes, antioxidant defenses, and tissues rich in mitochondria, which is reflected in the clinical syndrome. In addition to the cerebellar hemorrhage and edema associated with capillary damage, lead is also irritating, immunosuppressive, gametotoxic, teratogenic, nephrotoxic, and toxic to the hematopoietic system.

Clinical Findings for Lead Poisoning in Animals

Acute lead poisoning is more common in young animals. The prominent clinical signs are associated with the GI and nervous systems. In cattle, clinical signs that appear within 24–48 hours of exposure include ataxia, blindness, salivation, spastic twitching of eyelids, jaw champing, bruxism, muscle tremors, and convulsions.

Subacute lead poisoning, usually seen in sheep or older cattle, is characterized by anorexia, rumen stasis, colic, dullness, and transient constipation, frequently followed by diarrhea, blindness, head pressing, bruxism, hyperesthesia, and incoordination.

Chronic lead poisoning, occasionally seen in cattle, may produce a syndrome that has many features in common with acute or subacute lead poisoning. Impairment of the swallowing reflexes frequently contributes to development of

aspiration pneumonia. Embryotoxicity and poor semen quality may contribute to infertility.

GI abnormalities, including anorexia, colic, emesis, and diarrhea or constipation, are predominant manifestations in dogs. Anxiety, hysterical barking, jaw champing, salivation, blindness, ataxia, muscle spasms, opisthotonos, and convulsions may develop. CNS depression rather than CNS excitation may be evident in some dogs. In horses, lead poisoning usually produces a chronic syndrome characterized by weight loss, depression, weakness, colic, diarrhea, laryngeal or pharyngeal paralysis (roaring), and dysphagia that frequently results in aspiration pneumonia.

In birds, anorexia, ataxia, loss of condition, wing and leg weakness, and anemia are the most notable signs.

Lesions

Animals that die from acute lead poisoning may have few observable gross lesions. Oil or flakes of paint or battery may be evident in the GI tract. The caustic action of lead salts causes gastroenteritis. In the nervous system, edema, congestion of the cerebral cortex, and flattening of the cortical gyri are present. With histologic analysis, endothelial swelling, laminar cortical necrosis, and edema of the white matter may be evident. Tubular necrosis and degeneration and intranuclear acid-fast inclusion bodies may be seen in the kidneys. Osteoporosis has been reported in lambs. Placentitis and accumulation of lead in the fetus may result in abortion.

Diagnosis of Lead Poisoning in Animals

- Tentative diagnosis based on neurologic and gastrointestinal manifestations

- Confirmed by whole-blood analysis antemortem or by analysis of liver and kidney tissues postmortem

Lead concentrations in various tissues may be useful to evaluate excessive accumulation and to reflect the level or duration of exposure, severity, and prognosis and the success of treatment. Concentrations of lead in the blood at 0.35 ppm, liver at 10 ppm, or kidney cortex at 10 ppm are consistent with a diagnosis of lead poisoning in most species. Many countries have deemed blood lead concentrations >0.05–0.10 ppm to be a notifiable disease in food-producing animals. Inspection or clearance by a regulatory veterinary officer or biosecurity inspector is mandatory before shipment for food consumption is permitted.

Canine blood smear, basophilic stippling, lead toxicity
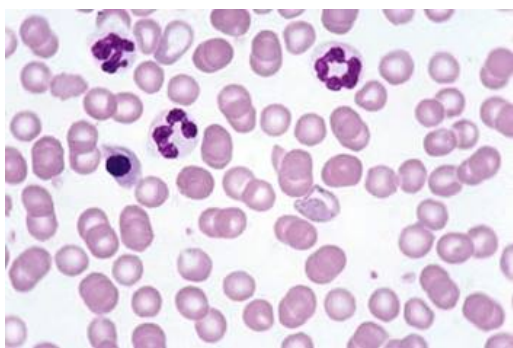
COURTESY OF DR. JOHN W. HARVEY.

Hematologic abnormalities, which may be indicative but not confirmatory of lead poisoning, include anemia, anisocytosis, poikilocytosis, polychromasia, basophilic stippling, metarubricytosis, and hypochromia. Blood or urinary delta-aminolevulinic acid and free erythrocyte protoporphyrin levels are sensitive indicators of lead exposure but may not be reliable indicators of clinical disease. Radiologic examination may be useful to determine the extent of lead exposure. Lead poisoning may be confused with other diseases that cause nervous system or GI abnormalities. In cattle, such diseases may include polioencephalomalacia, nervous coccidiosis, tetanus, hypovitaminosis A, hypomagnesemic tetany, nervous acetonemia, organochlorine insecticide poisoning, arsenic or mercury poisoning, brain abscess or neoplasia, rabies, listeriosis, and *Haemophilus* infections.

In dogs, rabies, distemper, and hepatitis may appear similar to lead poisoning.

Treatment of Lead Poisoning in Animals

- Chelation therapy for companion animals

- Treatment not recommended for food-producing animals

In food-producing animals, treatment is not recommended. A poor prognosis related to a poor response to treatment, extensive supportive care associated with anorexia, permanent tissue damage, the lack of approved chelation products, and significant tissue residues create significant economic and public health concerns. Prompt chelation therapy in companion animal species is often successful.

If tissue damage is extensive, particularly to the nervous system, treatment may not be successful. In food-producing animals, calcium disodium edetate (Ca-EDTA) is administered IV or SC (110 mg/kg per day) divided twice a day for 3 days; this treatment should be repeated 2 days later. In dogs, a similar dose divided 4 times a day is administered SC in 5% dextrose for 2–5 days. After a 1-week rest period, an additional 5-day treatment may be required if clinical signs persist. No approved veterinary product containing Ca-EDTA is currently commercially available.

Thiamine (2–4 mg/kg per day, SC) alleviates clinical manifestations and reduces tissue deposition of lead. Combined Ca-EDTA and thiamine treatment appears to produce the most beneficial response.

D-Penicillamine can be administered PO to dogs (110 mg/kg per day) for 2 weeks. However, undesirable adverse effects such as emesis and anorexia have been associated with this treatment. D-Penicillamine is not recommended for food-producing animals. Succimer (meso 2,3-dimercaptosuccinic acid [DMSA]) is a chelating agent that has been effective in dogs (10 mg/kg, PO, 3 times a day for 10 days) and is also useful in birds. Fewer adverse effects have been associated with DMSA than with Ca-EDTA.

Cathartics such as magnesium sulfate (400 mg/kg, PO) or a rumenotomy may be useful to remove lead from the GI tract. In cattle, surgery to remove particulate lead material from the reticulum after the ingestion of batteries is rarely successful. Barbiturates or tranquilizers may be indicated to control convulsions. Chelation therapy, in combination with antioxidant treatment, may limit oxidative damage associated with acute lead poisoning. Antioxidants such as *N*-acetylcysteine (50 mg/kg per day, PO) have been administered in combination with DMSA.

Mobilization of lead at parturition, excretion of lead into milk, and lengthy withdrawal times in food-producing animals raise considerable controversy regarding the rationale for treatment from both public health and animal management perspectives. The half-life of lead in the blood of cattle ingesting particulate lead is usually >9 weeks. Withdrawal times, which may be >1 year, should be estimated by periodic monitoring of blood lead concentrations. In a herd of cattle with confirmed cases of lead poisoning, all potentially exposed cattle should be evaluated. A small but significant portion of the asymptomatic cattle may have concentrations of lead in tissues that exceed recognized food safety standards. In dairy animals, lead is secreted into the milk. Children, who often consume large quantities of milk, are at much greater risk. Milk standards vary from 0.02 to 0.04 ppm in many countries. Many suggest this standard should be reduced to 0.005–0.01 ppm, which is comparable to human drinking water standards. The food-producing animal water standard in many countries is 0.1 ppm.

Key Points

- Lead poisoning is a common toxicosis worldwide. Sources of exposure vary from region to region.

- The public health risks, particularly in children, and the permanent impairment associated with lead exposure, in part related to the consumption of contaminated animal products, continue to be a concern for humans and animals.

For More Information

- Cowan V, Blakley B. Acute lead poisoning in Western Canada cattle. A 16 year retrospective study of diagnostic case records. Can Vet J. 2016;57(4):421-426.

- WHO: Lead poisoning fact sheet

- California Department of Food and Agriculture: Lead toxicity