Lead poisoning/Toxicity

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Sources:

1. lead poisoning is most common in dogs and cattle.
2. Lead poisoning in other species is limited by reduced availability, more selective eating habits, or lower susceptibility.
3. In cattle, associated with seeding and harvesting activities through used oil and battery disposal from machinery left openly.
4. Paint, linoleum, grease, lead weights, lead shot, and contaminated foliage growing near highways/ roadsides .
5. In cities accidental licking of pets with lead-based paint.
6. Improper disposal of lead poisoned animal carcasses may result in toxicoses in scavenger animals.

Toxicokinetics:

1. Absorbed lead enters the blood and soft tissues
2. Redistributed to the bone.
3. In ruminants, particulate lead lodged in the reticulum slowly dissolves and releases significant quantities of lead.

Mechanism of action:

1. Lead interfere activity of sulfhydryl containing enzymes.
2. Lead can replace zinc as an enzyme cofactor in some metabolic pathways.
3. In bone marrow, lead can inhibit several steps related to hemoglobin and erythrocyte synthesis. the thiol content of erythrocytes
4. antioxidant defenses, and tissues rich in mitochondria is also affected by lead toxicity.
5. 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:

1. Acute lead poisoning –
   a. Common in young animals.
   b. The clinical signs are associated with the GI and nervous systems.
   c. In cattle, signs that appear within 24–48 hr of exposure include ataxia, blindness, salivation, spastic twitching of eyelids, jaw champing, bruxism, muscle tremors, and convulsions.
2. Subacute lead poisoning-
   a. 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.
3. Chronic lead poisoning –

a. occasionally seen in cattle, may produce a syndrome
b. Aspiration pneumonia.
c. Embryotoxicity and infertility.
d. GI abnormalities, including anorexia, colic, emesis, and diarrhea or constipation CNS depression in dogs.
e. Anxiety, hysterical barking, jaw champing, salivation, blindness, ataxia, muscle spasms, opisthotonos, and convulsions may develop.
f. In horses- weight loss, depression, weakness, colic, diarrhea, roaring, and dysphagia results in aspiration pneumonia.
g. In birds, anorexia, ataxia, loss of condition, wing and leg weakness, and anemia

PM Lesions:

1. Oil or flakes of paint or battery may be evident in the GI tract.
2. Gastroenteritis.
3. Edema, congestion of the cerebral cortex.
4. Osteoporosis in lambs.
5. Placentitis and accumulation of lead in the fetus may result in abortion.

Diagnosis:

1. History, clinical signs and PM lesions.
2. Lead concentrations in various tissues concentrations >0.6 ppm or >0.35 ppm with appropriate clinical signs.
3. 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
4. 90% of lead in whole blood is bound to erythrocytes.
5. Radiologic examination may be useful to determine the magnitude of lead exposure

Differential diagnosis:

1. Diseases that cause nervous or GI abnormalities.
2. In cattle, such diseases- polioencephalomalacia,, tetanus, hypovitaminosis A, hypomagnesemic tetany, organochlorine insecticide poisoning, arsenic or mercury poisoning, brain abscess, rabies, listeriosis
3. In dogs, rabies, distemper, and hepatitis

Treatment:

1. In livestock, calcium disodium edetate (Ca-EDTA) is given IV or SC (110 mg/kg/day) divided bid for 3 days should be repeated 2 days later.
2. In dogs, a similar dose divided qid is administered SC in 5% dextrose for 2–5 days.
3. Thiamine (2–4 mg/kg/day, SC
4. Combined CaEDTA and thiamine treatment
5. D-Penicillamine can be administered PO to dogs (110 mg/kg/day) for 2 wk. emesis and anorexia have been associated with this treatment.
6. Cathartics such as magnesium sulfate (400 mg/kg, PO)
7. Rumenotomy may be useful to remove lead from the GI tract.
8. Barbiturates or tranquilizers may be indicated to control convulsions.
9. Chelation therapy
10. Antioxidants such as Nacetylcysteine (50 mg/kg/day, PO) have been used in combination with DMSA.

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