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Lecture title: Lead Toxicosis (Plumbism)

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Summary:

Lead poisoning (Both organic and inorganic lead) is associated with the accidental ingestion of lead metal or lead containing compounds.

Epidemiology

*Where groups of animals have access to the same source of lead, outbreaks occur and the morbidity rate ranges from 10% to 30%.

*The case–fatality rate may reach 100%.

*Risk Factors:

•Most cases of poisoning occur during the summer months from May to August.

•Incidence of the disease is highest in cattle in the spring after the animals have been turned out onto pasture.

•Poisoning is most common in younger cattle 6 months of age or less.

•The natural curiosity, licking habits, and lack of oral discrimination of cattle makes any available lead-containing material a potential source of poisoning.

• Cattle will readily drink motor oil; lick older machinery grease, paint, and paint ashes.

•Sheep and Horses are usually affected by eating soil or forage contaminated by environmental sources of lead.

•Environmental pollution: more likely to occur near smelters or other industrial enterprises or near major highways where pasture is contaminated by exhaust fumes of automobiles using leaded gasoline.

Sources of Lead:

•Phosphorus deficiency may also be a predisposing factor, because affected animals will chew solid objects as a manifestation of osteophagia.

•Discarded lead batteries and crankcase oil

•Batteries placed in garbage dumps on the farm and it is freezed during the winter months and break open, exposing the plates, which are attractive and palatable for cattle to lick and chew.

•Grease and lead-contaminated engine oil.

•linoleum, roofing felt, putty, automobile oil filters.

•Lead parasiticide sprays, particularly those containing lead arsenate.

Pathogenesis

*The toxic effect of lead varies depending on the chemical form of lead, amount ingested, age and species of animal.

*Once lead absorbed, 60% to 90% of lead is found in erythrocytes and the rest bound to albumin and other proteins.

*Lead disrupts the blood-brain barrier allowing albumin, water, and electrolytes to enter, resulting in edema.

*Lead mimics or inhibits the action of calcium altering the release of neurotransmitters and activating protein kinases.

*It also binds to a sulfhydryl group on proteins resulting in inhibition of enzymes, decreasing heme synthesis and hemoglobin production results in decreased oxygen carrying capacity and tissue ischemia.

Clinical findings

*The major effects of lead toxicity are often manifested in three main ways:

• Lead encephalopathy: following the ingestion of large doses in susceptible animals such as calves,

• Gastroenteritis: following moderate doses

• Degeneration of peripheral nerves: following long-term ingestion of small amounts of lead.

*The signs of acute lead poisoning, sudden onset and short duration, usually lasting only 12 to 24 hours.

•Many animals found dead without any observable signs.

•Staggering and muscle tremors of the head and neck, with champing of the jaws and frothing at the mouth are obvious.

•Rolling of the eyes, and bellowing are common.

•Blindness and auricular twitching.

•Eventually intermittent tonic-clonic convulsions occur and may continue until death.

•The animal maniacal, charges into fences,

*Sheep

•usually manifested by a subacute syndrome

Clinical pathology

-Poisoned animals, including horses, usually have levels above 0.35 ppm and deaths begin at 1.0 ppm.

Necropsy findings

•In most acute cases there are no gross lesions at necropsy.

•In cases of longer standing there may be some degree of abomasitis and enteritis, diffuse congestion of the lungs.

Differential diagnosis

cattle

•Arsenic poisoning • hypovitaminosis A •Hypomagnesemic tetany

Sheep

•Enzootic ataxia (copper deficiency) •Enzootic muscular dystrophy

Horse: •Botulism and Equine degenerative myeloencephalopathy

Treatment

1- Calcium versenate (calcium disodium EDTA [CaEDTA] Cattle may be treated with 73.3 mg/kg/day slow intravenously divided two to three times a day for 3 to 5 days.

2- Horses CaEDTA at 75 mg/kg BW divided two to three times a day by slow intravenous infusion for 4 to 5 days.

Note: side effects: Renal and gastrointestinal toxicity may occur.

3- Dimercaptosuccinic acid has been used for many years in human medicine as a specific chelator for arsenic, lead, and mercury.

4- Thiamine hydrochloride reduced the deposition of lead in most tissues. 2 mg/kg BW intramuscularly, given at the same time as CaEDTA,.

5- Oral dosing with small amounts of magnesium sulfate has been used

6- Rumenotomy to remove the ingested lead has been used but may be unsatisfactory.

Control

• Limit grazing on pastures near lead mines, smelters, or battery recycling depots.

• Keep trash out of pastures.