Popular Article

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Source, clinical signs, post-mortem findings, diagnosis, treatment and preventive measures of selenium toxicity in animals

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Sources of Poisoning: The primary cause of acute selenium poisoning in cattle, sheep, and horses is high selenium-containing plants. Swine and poultry that consume grains grown on seleniferous soils or as a result of an error in the feed formulation have been documented to become ill. Products containing parenteral selenium are quite hazardous, especially to young animals. Selenium toxicosis in dogs and cats has been linked to medicated shampoos (containing selenium disulfide) used to treat certain dermatitis and combat dandruff. Seleniumis also engaged in toxicity as an antioxidant in lubricating oils, fungicides, and insect repellents.

Clinical signs:

Acute selenium poisoning: After consuming extremely seleniferous feed or forage, acute poisoning typically results in death a few hours later. Atypical posture, an unstable walk, and adistinctive "rooted-to-one-spot" attitude with the head and ears depressed are all signs of poisoning in ruminants. Blood-tinged, foamy nasal discharge, prostration, fever, mydriasis, abdominal pain, diarrhoea, polyuria, and elevated heart and breathing rates. Instead, sheep mayexperience depression and pass away quickly without exhibiting many toxin-related symptoms.

Chronic selenium poisoning: It is 2 types.

Blind staggers: This condition in cattle is manifested in 3 stages:

1. Animals with the condition may roam intentionally and run into items. The body temperature is typically normal. The animal's appetite declines and its vision becomesblurry.
2. The wandering increases, the front legs become weak and the vision becomes further impaired.
3. The animal's tongue and mouth become paralysed, its body temperature drops, and it eventually dies of respiratory failure.

In sheep, these stages are less clearly differentiated.

Alkali disease: Alkali sickness is characterised by cracked hooves, lameness, stiff joints, dullness and lack of vigour, emaciation, and hair loss. The earliest clinical symptom of long hair loss in the mane and tail of horses typically comes before the foot begins to fracture at thecoronary band. Dead tissue is forced downward by the hoof's new growth, which results in sloughing. Deformed cattle hooves that are 15–18 cm long and pointed upward are common. Hoof breaks in pigs are comparable to those in cattle. Piglets die more often at birth and sowshave a lower pregnancy rate. Selenium levels greater than 2.5 ppm in eggs result in reduced hatchability and typically deformed embryos with ropy feathers and no beaks. Reducedfibrinogen levels and prothrombin activity, as well as an increase in serum alkaline phosphatase, ALT, AST, and a11d succinic dehydrogenase activity, are among the blood's biochemical abnormalities in selenium toxicosis.

Post-mortem lesions: Acute toxicosis in animals results in pulmonary oedema, congestion, and degeneration of the liver and kidneys. Necropsy revealed blind staggers necrosis and cirrhosis of the liver, spleen enlargement with isolated hemorrhagic regions, renal medulla congestion, epicardial petechiae, hyperaemia and ulceration of the abdomen and small intestine, and erosion of the articular, surface (particularly of the tibia). Ascites is virtually always discovered. The most noticeable lesions in alkali disease are liver cirrhosis and cardiacshrinkage, which are essentially identical to those seen in blind staggers.

Diagnosis: The diagnosis is made based on the patient's medical history, clinical symptoms, necropsy results, and laboratory confirmation of selenium levels in the blood or tissue (liver, kidney) and diet

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(feed, forage, grains) of the animal. Selenium poisoning is indicated by dietaryselenium levels above 5 ppm. Selenosis is confirmed by severe toxicity signs at 10–25 ppm ofselenium. Blood selenium levels in acute toxicosis can reach up to 25 ppm, whereas those in chronic toxicity can range from 1-4 ppm. Both acute and chronic poisoning may contain 4–25ppm in the kidney or liver. The presence of selenium in the urine or hair could also be a sign of toxicosis.

Differential diagnosis: Pneumonia, anthrax, infectious hepatitis, enterotoxaemia, and pasteurellosis are among the illnesses/conditions that can be misdiagnosed with acute toxicosisor blind staggers. Ergotism, molybdenosis, fluorosis, and laminitis are diseases that resemble certain symptoms of chronic selenosis (alkali illness). Acute toxicosis is indicated by the smellof rotten horseradish or garlic in a fresh corpse, but the lack of such a smell may not entirely rule out this illness because the volatile selenides may escape quickly. A tissue selenium test, however, could support the diagnosis.

Treatment and preventive measures. Other than removing the source of exposure, there is no specific therapy or cure for selenium toxicosis. Animals in need of symptomatic and supportive care should begin treatment as soon as feasible. It has been claimed that a high- protein diet, linseed meal, sulphur, arsenic, copper, and cadmium minimise selenium toxicity in laboratory species, but usage of all these in the field needs to be confirmed. To lessen the likelihood of selenium toxicity, arsenic has been recommended in drinking water (5 ppm of arsenic as sodium arsenite), arsenic salt (containing 25 ppm of arsenic), or arsinilic acid at 0.02%.

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