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Popular Article

Pathogenesis, diagnosis and treatment of copper toxicity in animals

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Occurrence

Accidental administration of a copper sulphate solution that is overly powerful or in excess for treating parasite disorders. consuming food that contains copper salts. The consumption of copper-rich foods and salt licks can both lead to chronic poisoning. grazing onpasture that had been treated with a copper salts-containing fungicide. Copper salts used in snail eradication campaigns have contaminated drinking water. In regions with copper-rich soil, chronic poisoning is possible. Consuming some plants, such as *Heliotropum europaeum* and *Echium plantagineum*, causes aberrant copper metabolism. Acute poisoning may developfrom copper intakes of 20–100 mg/kg in sheep and young calves, and 200–800 mg/kg in mature cattle. Sheep grazing on pastures containing 15-20 ppm (dry matter) of copper and low levels of molybdenum may experience chronic poisoning with daily copper intakes of 3.5 mg/kg.

Pathogenesis

Protein coagulants are copper salts that are soluble in water. As a result, they severely irritate the alimentary mucosa when consumed. If the initial intake is too high, gastrointestinal distress could result in shock and death. In other situations, where the irritability is not extreme enough to result in shock, copper enters the bloodstream and causes severe intravascular hemolysis. Hepatic insufficiency and hemoglobinuric nephrosis are present together. Hemolytic

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crisis, which includes methemoglobinemia and deteriorating lesions in the brain'swhite matter, is the name given to the entire occurrence. It takes a certain amount of copper totrigger a hemolytic crisis, and if less than that, as in chronic copper poisoning in sheep, it tendsto be stored in the liver. After six months, for example, when the maximum liver levels are reached, copper is rapidly released into the bloodstream, causing an acute, deadly hemolytic crisis. Thus, these sheep seem to be in good health before the hemolysis starts. Because the release of hepatic copper is influenced by stress, situations like fasting, travel, and lactation frequently trigger theillness. The liver and kidneys' copper levels rise during the prehemolytic and hemolytic stages of the illness. Additionally, during hemolysis, iron levels in the kidneys significantly rose, notably in the apical lysosomes of the proximal tubule cells. Digestive system absorption of copper results in a cumulative effect. Copper absorption is inhibited by high molybdenumlevels. Copper is eliminated through the gastrointestinal tract, urine, and bile.

Symptoms

There will be severe thirst and vomiting in the acute stage. vomiting that is green and has mucous. Abdominal discomfort that is severe will be felt. Diarrhea of a vivid green color.Death usually occurs within 24 hours of convulsions and coma. Loss of appetite and severe depression in chronic form. sluggishness and emaciation. Diarrhea followed by constipation.Jaundice with hemoglobinuria. loss of hair and thickening of the skin.

Post-mortem appearance

In its acute form, gastro-enteritis is clearly present, and the mucous membranes thicken. Abdominal abrasion. spleen, liver, and renal enlargement. heart with hemorrhagic spots. Gastroenteritis in its chronic phase. hepatic fatty degeneration and widespread icterus. The liverand spleen accumulate hemosiderin. Friable and enlarged spleen. myocarditis, cystitis, and nephritis. The tubules in the kidneys may be clogged with bloody casts and enlarged. Greenish-brown bile that is viscous and bloated fills the gall bladder.

Diagnosis

On the basis of a sudden death preceded by a hemolytic crisis and severe liver damage, a presumptive diagnosis is made. Measure copper concentrations in samples of the liver and kidney at the time of death if there is an elevated serum copper concentration, albeit the increase may be

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temporary. Acute copper poisoning is characterized by blue-green ingesta, elevated faecal (8,000–10,000 ppm), and renal (>15 ppm, wet wt.) copper levels. Copper concentrations in the blood and liver rise during the hemolytic phase in chronic poisoning.

Treatment

Symptomatic treatment for shock. Calcium versenate and penicillamine may be worth trying. Intervention should emphasize prevention because once liver damage and the hemolytic crisis have occurred, treatment is rarely effective. All animals have dismal prognoses. In animals with acute poisoning, supportive care for shock and gastrointestinal symptoms may be beneficial. If given in the early stages of the condition, penicillamine (50 mg/kg per day, PO, for 6 days) or calcium versenate may also be helpful to increase copper excretion. During a hemolytic crisis, vitamin C (500 mg/day per sheep, SC) may be able to lessen oxidative damage to RBCs. Both the treatment and prevention of copper poisoning work well with ammonium tetra thiomolybdate (1.7 mg/kg, IV, every other day for 6 days). Use this medication with caution as it improves copper excretion while decreasing copper absorption. This drug requires 10 days detox period. For three weeks, daily oral dosing of sodium thiosulfate (1 g) and ammonium molybdate (100 mg) may minimize the number of deaths in affected lambs. To lessen the absorption of copper, dietary supplementation with zinc acetate (250 ppm) may be helpful. Eliminate or restrict access to plants that cause phytogenous or hepatogenous copper toxicity.

In sheep flocks at high risk, dietary sodium thiosulfate supplementation may be used to prevent or treat chronic copper toxicosis. Additionally, when molybdenum or thiosulfate is supplied, periodic assessment of the copper and molybdenum concentrations in feed as well as refraining from feeding cattle feeds to sensitive species like sheep may help prevent chronic copper poisoning. Additionally, it is preferable to refrain from overdosing animals with chelated minerals.

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