COPPER TOXICITY/POISONING

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Source:

1. Copper is a metal considered an essential nutrient to be incorporated in the diet of all the animals.
2. If taken in large amount it may prove harmful. Sheep are by far the most susceptible farm animal species to copper poisoning, with goats being less susceptible than sheep and cattle being less susceptible than either sheep or goats.
3. Pigs are the least susceptible to copper poisoning, their rations often contain added copper (125-250ppm) in amounts that, if consumed by sheep, can cause acute copper poisoning.
4. Sources of copper that can cause copper poisoning in sheep and goats •
   a) Trace mineral-supplemented salt used for cattle or horses
   b) Vitamin and mineral supplements intended for horses, cattle, swine, or poultry
   c) Complete feeds for swine, horses, poultry, or cattle
   d) Pasture that has been fertilized with swine manure and poultry litter.
   e) Copper-containing disinfectant foot baths for cattle.
   f) When liver disease is present chances of toxicity increases.
   g) Certain plants of *Senecio* species that contain toxic alkaloids when eaten by sheep over a period of weeks can cause sufficient liver damage to precipitate a sudden release of stored copper from the liver to cause copper poisoning.

Toxicokinetics

1. Acute poisoning may follow intakes of 20–100 mg of copper/kg in sheep and young calves and of 200–800 mg/kg in mature cattle. Chronic poisoning of sheep may occur with daily intakes of 3.5 mg of copper/kg when grazing pastures that contain 15–20 ppm (dry matter) of copper and low levels of molybdenum.
2. Poisoning occur when sheep or goats ingest small amounts of copper over a prolonged time period.
3. Oral intake of copper lead to necrosis of GIT mucosa (acute toxicity), then it is absorbed and comes to systemic circulation and enters the RBCs which become more fragile leading to haemolysis
4. Copper that is ingested is stored in the animal's liver, and repeated ingestion of small amounts of copper above the animal's requirement becomes a toxic amount of copper for the animal.
5. Referred to as secondary copper poisoning, liver disease causes normally stored copper in the liver to be released into the blood stream where it causes hemolysis.
6. Once copper has accumulated to a toxic amount in the liver, the sheep or goat can develop sudden and severe signs of disease.

Mechanism of action

1. Cupric ions interact with sulphur group of glutathione in the RBC resulting in decreased glutathione concentration in RBC.
2. In RBC ferrous ions is replaced by cupric ions and formation of methaemoglobin take place which ultimately leads to haemolytic crisis.
3. High concentration of copper inhibit various metabolic function and causes liver necrosis.
4. Blood copper concentrations increase suddenly, causing lipid peroxidation and intravascular haemolysis

Clinical findings:

1. Acute copper poisoning causes severe gastroenteritis characterized by abdominal pain, diarrhea, anorexia, dehydration, and shock.
2. Hemolysis and hemoglobinuria may develop after 3 days if the animal survives the GI disturbances.
3. Weakness, panting, and dull attitude
4. Recumbency, rumen stasis, anorexia, thirst, dyspnoea
5. Pale mucous membranes Yellow discoloration (jaundice) of the mucous membranes of the eyes, gums and genitalia
6. Dark brown or red colored urine hemoglobinuria
7. Abortion in pregnant ewes and does
8. The sudden onset of clinical signs in chronic copper poisoning is associated with the hemolytic crisis.

P M Lesions:

1. Acute copper poisoning- gastroenteritis with erosions and ulcerations in the abomasum of ruminants.
2. Icterus develops in animals that survive >24 hr.
3. Tissues discoloured by icterus and methaemoglobin are characteristic of chronic poisoning.
4. Swollen, **gunmetal-coloured kidneys**, **port-wine-colored urine**.
5. Enlarged spleen with dark brown-black parenchyma with hemolytic crisis.
6. The liver is enlarged and friable centrilobular hepatic and renal tubular necrosis.

Diagnosis:

1. History, clinical signs, PM lesions
2. Blood copper concentrations above 2 micrograms/ml and liver copper conc. above 150ppm (wet weight) are highly suggestive of copper toxicity.

Treatment:

1. Medications are administered to affected animals to increase the rate of excretion of copper from the liver.
2. Oxygen administration and other supportive care may be required.
3. GI sedatives and symptomatic treatment for shock may be useful in acute toxicity.

4. Penicillamine (50 mglkg, PO, sid, for 6 days) or calcium versenate may be useful if administered in the early stages of disease.
5. Daily administration of ammonium molybdate (100 mg) and sodium sulfate (1 g) reduces losses in affected lambs.
6. Dietary supplementation with zinc acetate (250 ppm) may be useful to reduce the absorption of copper.
7. Plant eradication or reducing access to plants that cause phytogenous or hepatogenous copper poisoning should be done.
8. Primary chronic or phytogenous poisoning may be prevented by top-dressing pastures with molybdenum per acre (70 g/ hectare) in the form of molybdenized superphosphate or by molybdenum supplementation or restriction 'of copper intake.

Prevention:

1. Owners of sheep and goats must remain aware that premixed complete feeds, salts, and nutritional supplements designed for other species may contain concentrations of copper that are dangerous to sheep and goats. Check before adding any supplemental mixtures of vitamins or minerals to the diet of sheep or goat
2. Poultry manure (litter) and swine manure contain potentially dangerous concentrations of copper, sheep and goats should not be allowed to graze pastures where these have been applied as fertilizer.

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