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Popular Article

Hypomagnesemia in Sheep

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Introduction

Hypomagnesemia is an acute metabolic disease of sheep, characterized by a variety of clinical signs like excitability, grinding of the teeth, salivation, ataxia, recumbency, and tetanic muscle spasms and is caused by a depressed supply of metabolizable magnesium from prolonged consumption of lush immature grasses. It is also known as grass tetany or grass staggers or wheat pasture poisoning. Adult lactating animals are most susceptible because of the loss of Mg in milk. Due to its wide geographic distribution and as a causative of extensive economic losses, it has major importance and high concern for the entire sheep industry. Economic losses result from deaths, determent of full utilization of pasture feed and the cost of preventive programs.

Occurrence

Hypomagnesemia occurs in all breeds of lactating sheep grazing early spring pastures. Although most cases develop in ewes during the first 4 wks of lactation, other classes of sheep occasionally develop the disease. Most cases occur during spring and autumn following rains and rapid growth of grasses.

Etiology and pathogenesis

- Continuous and exclusive grazing on lush grasses causes Hypomagnesemia.
- In normal sheep, the plasma levels of magnesium and calcium are 2-3 mg/dl and 8-12 mg/dl, respectively. Grazing lush grasses may depress the plasma levels of Mg and Ca to pathogenic levels and result in the disease.

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- Lush green plants growing on ammonium fertilized soil, contain high concentrations of protein, potassium and aconitate.
- Absorption of ammonia by plants results in reduced uptake of Magnesium and Calcium with little effect on potassium and produces high amide concentrations in plants with carbohydrate depletion. These factors combine in the animal to create high conc. of free ammonia in the rumen, an increase in rumen pH and depletion of carbohydrate and further reduces the availability of Mg and Ca.
- Mobilization of Mg from its reserve in bone to extracellular fluid commences when plasma Mg level falls below1.8 mg/dl. When this reservoir is exhausted the plasma level of Mg falls; when the conc. reaches about 1.0 mg/dl, the plasma Ca levels also declines. When it reaches 0.7 mg/dl, irritability develops, and when it reaches 0.5 mg/dl, fatal tetany and convulsions ensue. Death probably results from respiratory failure. Recovered animals are susceptible to recurrence of the disease.

Clinical signs and Postmortem lesions

In acute cases:

- Affected sheep which may appear to be grazing normally, suddenly throw up their heads, gallop in a blind frenzy, fall, and exhibit severe paddling seizures
- chomping of the jaws
- frothy salivation
- fluttering of the eyelids and nystagmus
- Seizures may recur at short intervals, and death usually occurs within a few hours
- In many instances, animals at pasture are found dead without observed illness.

In less severe cases:

- Hyperesthesia
- Twitching of ears
- Grinding of teeth
- Small muscle fasciculations
- Breathing acceleration
- Tremors, walking and running movements
- Muscular incoordination

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- Mild cases may recover spontaneously and episodes may recur.
- Some fall to a recumbent position, convulse, pass into coma and finally die

Post mortem lesions: At necropsy, petechial and echymotic hemorrhages over serosal surfaces of heart and intestines

Diagnosis

- Based on clinical signs and laboratory findings
- History of excitement and convulsions among lactating ewes grazing lush grasses strongly suggest the disease.
- Plasma Mg levels of 0.50 to 0.25 mg/dl in blood

Differential diagnosis

should be differentiated from hypocalcemia and enterotoxaemia.

- Hypocalcaemic animals respond to intravenously administered Calcium boro gluconate and their blood analysis shows plasma Ca levels as low as 3 mg/dl and Mg levels near normal values.
- Enterotoxaemia causes sudden deaths and affected animals contain identifiable toxin in the small intestine.

Prevention and treatment

- It can be Prevented by oral feeding of crude magnesium oxide either by individual dosing with 15 g dissolved in water and given at 2day intervals or by feeding in a mineral mix containing 20% of the compound.
- Fertilizers rich in potassium and nitrogen reduce the availability of magnesium from the pasture, and increase the risk of grass tetany. Hence, animals should not be allowed for grazing these pastures soon after fertilizer application.
- Treatment includes parenteral administration of 50 ml of a 20% solution of calcium boro gluconate and 25 ml of 50% solution of magnesium sulfate.
- Daily oral supplements of Mg oxide (10 g) should be given in the danger period

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