

Polioencephalomalacia (Goat Polio)

Introduction

Polioencephalomalacia (PEM) is a common metabolic disorder characterized by neuro-muscular alterations of goats that are thiamine deficient. This disorder may be acute or sub-acute in nature. Adults and young animals are equally at high risk for developing the disorder. Goat polio is usually seen in animals that are under a high nutritional management condition such as feedlots, or animals on lush pasturage fed with highly concentrated rations.

Thiamine deficiency usually implies the depletion of carbohydrates in brain cells that manifest as a neurological disorder. Thiamine (Vitamin B1) is produced by the bacteria and protozoa of the rumen under normal environmental conditions. Any change in the ruminal environment will affect the natural microorganismal production of thiamine, increase the degradation of thiamine, or prevent thiamine from functioning properly in sheep and goats.

In highly grain-fed animals, thiamin molecules produced in the rumen can be inactivated or degraded by the thiaminases, enzymatic proteins that catabolize thiamine. In the presence of excessive concentrated rations, thiaminase I is excessively produced by *Bacillus sp.* and *Clostridium sporogenes*. Thiaminase II is produced by *B. aneurinolyticus*. Both enzymes are responsible for degrading thiamine. Lower levels of thiamine will promote a lower supply of carbohydrates to the nerve cells, causing central nervous system disorders, PEM, and death. Thiamine is a cofactor in the metabolism of carbohydrates (sugars). The lack of thiamine will induce a lower supply of carbohydrates to the neurons in the brain. The neurons require carbohydrates as an energy source necessary for nerve function. The depletion of carbohydrates causes alterations of the mechanism of action of the nervous system and

neuronal death. In order for PEM to occur, thiaminases I and II must be produced to inhibit thiamin-dependent biochemical reactions.

A higher production of thiaminase in the rumen is often due to sudden dietary changes, whereby diets are rich in concentrated ration and low in roughage. Increased thiaminase production can also result from prolonged treatment with antiprotozoa substances such as CORID® (amprolium), the administration of dewormers, and animals grazing in recently fertilized pasture, and in animals exposed to high sulfur intake.

Clinical Signs

Clinical signs of goat polio are associated with cerebral edema, cerebellar, and herniation of the brain, and the death of brain cells controlling motor and visual functions. Convulsions occur in 2 to 5-minute intervals.

Goats may be standing or lying down when having convulsions. Goats appear dull and depressed and unable to coordinate muscular movements. They may also show signs of increased aggression, muscle tremors, and temporary blindness that can last 2 to 3 weeks. Body temperature, pulse, and respiration rates can be increased. Rumen motility is maintained normally. Other signs of PEM include opisthotonos, a condition of abnormal posturing where the head is thrown backward accompanied by rigidity, severe arching of the back, muscular contractions, and teeth grinding. As the condition progresses, the animal becomes recumbent with frequent convulsions, nystagmus (rapid involuntary movement of the eyeballs), blindness, and unaltered palpebral and pupillary responses.

Diagnosis

Diagnosis is based on clinical signs and history of the herd management and laboratory analysis. Laboratory analysis shows that the levels of thiaminase, pyruvate in the urine, blood pyruvate, lactate levels, and pyruvate kinase levels are all increased; transketolase activity values are lower in affected animals.

Differential diagnosis consists of the exclusion of other diseases and disorders of the neuro-muscular systems such as caprine arthritis encephalitis, listeriosis, enterotoxemia, pregnancy toxemia, grain poisoning, plant poisoning, rabies, and tetanus.

Treatment, Prevention, and Control

The response to treatment depends on the condition and extent of brain lesions. In the early stages of thiamine deficiency, animals will respond promptly to treatment. In delayed diagnosis and treatment, full clinical recovery may not be possible. Administer thiamine HCl in the dosage of 4.5 to 10 mg/lb intravenously. Animals will show improvement within minutes or a few hours. In delayed diagnosis and treatment, full clinical recovery may not be possible. For animals that show slower signs of improvement, administer the dosage of thiamine HCl intramuscularly in 3- to 6-hour intervals.

In more severe cases where animals present blindness, thiamine HCl should be given intravenously in 4.5 mg/lb BW dosages at 4-6-hour intervals until animals show improvement or every 3 hours for a total of five doses. The administration of Dexamethasone 0.5 to 1/0 mg/lb IM or SC is recommended to decrease edema and inflammation of the brain. Fluid therapy with dextrose solution intravenously or subcutaneously, is also recommended.

Prevention

- Observe sulfur intake: source of water and consumed dry matter.
- Provide feed with thiamine levels of 1.5 – 4.5 mg/kg of feed.
- Provide sufficient levels of roughage; provide good quality pasture or hay as part of the diet.
- Monitor animals after you have administered antihelminths dewormers and/or amprolium (Corid®).
- Check sulfur content on water source and forages.

Consult your local veterinarian for disease treatment and prevention.

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