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Polioencephalomalacia in Ruminants

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Polioencephalomalacia (PEM) is a condition in which brain cells become damaged due to a lack of oxygen. The brain requires oxygen to meet its high energy demands. Energy, in the form of ATP (adenosine triphosphate), allows cells to maintain the correct balance of ions inside and outside the cell. When these ion balances are disrupted, the cells become swollen. When these cells swell, they become compressed against the animal's hard skull and are damaged further, causing neuron cell death.

Causes: PEM can occur in ruminants for several reasons. A deficiency in thiamine, an essential micronutrient also known as vitamin B1, or a disruption in its absorption and use, can cause PEM. Other causes include sulfur poisoning, lead poisoning, salt poisoning, not having enough water and some parasite treatments such as amprolium.

Epidemiology: Animals at higher risk of developing PEM include those transitioning to new diets near the time of weaning or shifting to high-concentrate diets for finishing, animals that consume feed or water with high sulfur content or are exposed to amprolium or plants that produce thiaminase, an enzyme that degrades thiamine.

Clinical Signs: PEM can be difficult to distinguish from other neurological diseases, such as trauma and rabies, as the signs are similar. Animals affected with PEM might hold their necks in an extended position, lift their legs too high when walking (called a hypermetric gait), stargaze, appear blind, pace along the fence line, grind their teeth, excessively drool, move their eyes abnormally or show other signs of distress, such as head pressing or recumbency.

Diagnosis: Diagnosing PEM usually relies on an animal's symptoms and its history related to the risk factors for PEM. Thiamine-responsive PEM is a type of PEM that can be treated with thiamine. The response to thiamine treatment is often rapid, which means it can be used to confirm the diagnosis of thiamine-responsive PEM. Additional tests may be done to check for levels of thiamine or sulfur in the animal's system, which can help determine the underlying cause of the disease. Excessive hydrogen sulfide in the rumen will cause an odor like rotten eggs. Postmortem examinations can confirm PEM.

Treatment: Treatment for PEM may include providing thiamine supplements, supportive care (like fluids and anti-inflammatory medications) and addressing the underlying issues that caused the condition. Early intervention will yield the best outcomes, and many animals can recover fully if treated in time. In cases of lead poisoning, chelation may be necessary to lower lead levels in the body. Chelation is a medical treatment that uses a chemical substance to bind with heavy metals in the body, allowing them to be passed through urine or feces.

Prevention and Management Considerations: To prevent PEM, it is important that animals get enough thiamine. In healthy ruminants, the rumen microbes produce the thiamine that the animal needs. However, this thiamine is not stored in the body, so if the rumen environment is changed to alter the production or absorption of thiamine, the animal can become thiamine-deficient within a short period of time. Thiamine can be degraded by thiaminase, an enzyme produced by some rumen microbes and found in certain plants, such as horsetail and bracken fern.

Sudden transitions to high-concentrate diets and certain medications used to treat parasitic worm infections (called anthelmintics), such as levamisole and fenbendazole, may contribute to thiaminase production in the rumen.

Amprolium, a medication used to treat coccidiosis, a parasitic infection, may interfere with the body's ability to use thiamine.

Excess sulfur in the diet can complicate matters. Ruminants require sulfur in their diets, but too

much sulfur can block the body from using copper and thiamine effectively. Supplementing diets with thiamine has been shown to protect against PEM in the face of excessive sulfur.

Some rumen microbes produce hydrogen sulfide, which is detoxified by the liver. Sulfide can cause PEM when the amount of sulfur dioxide produced is more than the liver can detoxify.

Sulfur sources may include water, feeds, calcium sulfate (gypsum) or ammonium sulfate — a nonprotein nitrogen source that can be added to feed to supply nitrogen and sulfur. Gypsum may be used as bedding or as a supplement to provide added calcium to diets. Feed ingredients that contain high levels of sulfur are molasses, distillers' spent grain, brewer's spent grain, and cruciferous vegetables such as cabbage and broccoli. Sulfur levels of water may vary by region and should be evaluated and considered when designing a balanced ration.

BASIC MANAGEMENT STRATEGIES TO DECREASE THE RISK OF POLIOENCEPHALOMALACIA

- Avoid thiaminase-producing plants in the forage or feed.
- Provide adequate fiber to maintain normal rumen pH.
- Gradually transition into higher-concentration diets.
- Evaluate sulfur content of water, feeds, forage and additives.
- Be aware of feed material that contains high levels of sulfur (i.e., molasses, distillers' spent grain, brewer's spent grain, cruciferous vegetables).
- Be aware of other sources of sulfur, such as ammonium sulfate or calcium sulfate (gypsum).
- Careful use of antiparasitic medications (amprolium and fenbendazole) along with adequate monitoring and timely treatment for PEM if needed.
- Supplement thiamine, if necessary.

REFERENCES

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