Polioencephalomalacia

A PowerPage Presented By

Polioencephalomalacia is a neurologic disorder seen in ruminants. A deficiency or disorder of thiamine is believed to be the main cause of the disease although others have been suggested as well, including excess intake of sulfur, lead, or sodium. Early recognition of the signs and treatment with thiamine is required to have success in treating the condition. This PowerPage reviews the pathophysiology, clinical signs, and treatment considerations for polioencephalomalacia.

Predisposing Factors

- **High concentrate diets** (pastured animals can also develop disease)
- Feed with corn or sugar cane byproducts
- **High grain intake** may promote proliferation of thiaminase producing bacteria
- Rations with added sulfate to limit intake
- **Ingestion of plant thiaminases** or **thiamine analogs**

Clinical Signs

- Depending on the nature of the underlying cause, this can appear as an isolated disease or a herd problem

Acute Form

- Blindness
- Seizures
- Recumbency

Subacute Form

- Initial signs
  - Decreased appetite
  - Twitching (ears and face)
  - Separation from the herd
  - Hold head up in an elevated position
  - Staggering, hypermetric gait
- Later signs
  - **Cortical blindness**
    - Absent menace, intact palpebral reflex and pupillary light response
  - Dorsomedial strabismus
  - **Head pressing**
  - **Teeth grinding**
  - Opisthotonus

Diagnosis

- Diagnosis can be challenging and one should consider differentials such as listeriosis, nervous coccidiosis, salt toxicity, meningoencephalitis, rabies, vitamin A deficiency, pregnancy toxemia (sheep)
    - Clinical presentation is useful to help differentiate polioencephalomalaica from many of these conditions
- Pre-mortem diagnostic testing can be unreliable and not available in many laboratories
    - ie. Blood thiamine levels, transketolase activity to assess thiamine
- Post-mortem findings
    - Gyral flattening, brain swelling
    - **Brain tissue may fluoresce under UV light**
    - Cerebrocortical neuronal necrosis
    - Cortical spongiosis
    - Cavitation of cortical tissue
    - Multifocal vascular necrosis, hemorrhage, and necrosis in deep gray matter

Treatment

- Early treatment is critical
- **Treat with thiamine, regardless of cause**
    - 10-20mg/kg IM or SC TID
    - Expect to see improvement within 1-3 days
- If cerebral edema is suspected, can administer 1-2 mg/kg of dexamethasone
- Supportive care
- Consider thiamine supplementation in feed for prevention of disease in other animals, supply roughage, and investigate environment for sources of thiaminases or excessive sulfur levels
- Animals with advanced disease may continue to show significant neurologic impairment after treatment