



Polioencephalomalacia (PEM, Cerebrocortical necrosis)

Extended Version

Presentation:

Classic case: Fast-growing calf or lamb, NEUROLOGIC, blind, staggering, down

Poorly understood nutritional disease of ruminants,
associated w/ **altered thiamine** status and/or **high sulfur** intake

Bilaterally symmetric clinical signs of cerebral dysfunction: especially in calves, lambs, kids

Acute:

Blindness
Recumbency
Rigidity, seizures
Coma
Vocalization (goats)

Subacute:

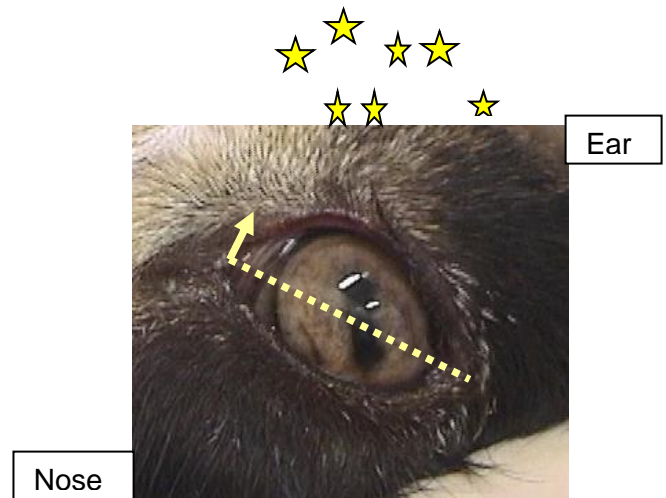
Depression/anorexia
Withdrawal from group
Facial, ear twitches
Head held elevated
Staggering, +/- hypermetria

With progression:

Cortical blindness = ↓ menace + N pupil reflexes
Dorsomedial strabismus
Head pressing
Opisthotonus



Opisthotonos, goat



*Dorsomedial strabismus, goat
"Star gazing"*

Differential Dx: (Diseases of ruminant cerebral dysfunction)

Cattle

Lead poisoning
Vitamin A deficiency
Hypomagnesemia
H₂O deprivation/ SaltTox
Nervous ketosis
Brain abscess
Coccidiosis w/CNS involvement
TEME: *H. somnus* meningoencephalitis
Rabies/other meningitis, encephalitis
Hepatoencephalopathy

Sheep

Pregnancy toxemia
Type D enterotoxemia
Listeriosis
Lead poisoning
Salt Tox/H₂O deprivation
Nervous ketosis



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Test(s) of choice:

Clinical signs; History - diet change, poor pasture, other dz causing anorexia

Dorsomedial strabismus pathognomonic (trochlear nerve damage, cause unknown)

Rotten egg smell in rumen gases w/ high sulfur

Response to Rx w/ vitamin B₁ is suggestive of PEM (but you still may not ID cause)

Laboratory tests:

- RBC transketolase - inconsistent, not always available
- Pyruvate, lactate, pyruvate kinase in blood
- Thiaminase in feces, rumen
- CSF - r/o encephalitis, ketosis - ketones-urine/milk/blood
- Lead (Pb) poisoning – Pb levels in blood/tissues

Necropsy lesions inconsistent, often subtle

Late pathology: Cavitation visible in cortex

Early cases: +/- Autofluorescent bands of necrotic cerebral cortex w/ UV light

Rx of Choice:

EARLY TREATMENT IS VITAL PEM Sx are seen before CNS damage occurs

1.) **Thiamine (vitamin B₁):** 10-20 mg/kg TID dosing

- **INITIAL dose should be IV** then IM, SC after
- **Continue vitamin B₁ for days past improvement**

2.) +/- Rx to decrease cerebral edema

- Mannitol
- Dexamethasone - 1-2 mg/kg IM or SC

3.) Rx primary disease(s)

4.) +/- Rx for seizures: Diazepam, Phenobarbital

5.) Support/prevent- Correct diet, transfaunate, force feed



Opisthotonos, calf

Prevention:

Institute dietary changes slowly, ensure appropriate roughage/concentrate ratios, avoid bad pasture

Prognosis:

PEM is reversible with early treatment - **Blindness is last sign to disappear;**

Grave Px for advanced, prolonged cases, permanent brain damage possible



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Pearls/Pathogenesis:

Thiamine deficiency, sulfur toxicosis, sodium toxicity, water deprivation, and lead toxicosis can ALL produce the clinical signs and lesions of PEM.

Aerobic energy metabolism disrupted in the brain → changes in membrane function, water and sodium accumulate inside the cell – cerebral edema, swelling, pressure necrosis.

Two causes of PEM:

1.) Thiamine deficiency - Vitamin B₁ is required for production of several enzymes (transketolase, etc.) needed for glucose metabolism and as a cofactor for ATP production in the brain

Normal thiamine production by rumen microbes barely meets dietary requirements

Anything that disrupts microflora thiamine production can lead to PEM:

- Animal off feed for any reason, adjusting to new diet, inappropriate diet
- High grain/low fiber diet favors growth of thiaminase producing bacteria
- Ingestion of thiaminase-containing plants - horsetail, pigweed, bracken fern, nardoo fern (Australia)
- Thiamine analogs given – amprolium, pyrimethamine, levamisole, thiabendazole

2.) Increased sulfur intake - H₂S gas in rumen (rotten egg smell!); **Sulfur toxic to cellular metabolism**

- Water with high sulfur content High molasses in diet (urea)
- Additives – gypsum (CaSO₄), Urinary acidifier AmmSO₄
- Alfalfa (Hi protein w/ S amino acids)
- Synthesis of S rich compounds - Cruciferous plants - Turnips, rape, mustard
- Acidifying agents w/ added S corn, sugar beet, cane sugar by-products
- Sulfate accumulators - kochia, lambsquarter, Canada Thistle

References: Pasquini's Guide to Bovine Clinics 4th ed., p. 140, Divers and Peek, Rebhun's Diseases of Dairy Cattle, 2nd ed., pp. 521-3, Pugh and Baird's Sheep and Goat Medicine 2nd ed., pp. 378-80, and the Merck Veterinary Manual, Polioencephalomalacia

My Notes: