Polioencephalomalacia: Case Report & Review

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Case Report

A Holstein-cross heifer approximately six months of age was presented to Boren Veterinary Medical Teaching Hospital on September 21, 1987. The owner reported that the animal had been missing for two days and that the calf had been found recumbent that morning.

Upon physical examination, the heifer had a rectal temperature of 99.2° F, pulse rate of 112, and respiratory rate of 20. The heifer was found to be approximately 5% dehydrated. The heifer was recumbent and was showing signs of severe opisthotonos. Upon examination of the animal's eyes, pupillary light reflexes were found to be normal, but the heifer had no menace reflex. The calf did not have diarrhea.

Differential diagnosis included lead poisoning, nitrofuran toxicity, hypomagnesemia, vitamin A deficiency, chlorinated hydrocarbon toxicity, infectious thromboembolic meningoencephalitis, brain abscesses, and polioencephalomalacia.

Based upon the clinical findings and the history of two previous cases of polioencephalomalacia in the herd several months earlier, the diagnosis of polioencephalomalacia was made.

The heifer was initially treated with 4 g thiamin IV and 4 g thiamin IM. The heifer was also treated with 3 million IU of procaine Pen G IM on the first day. By that evening, the calf was sternal.

On day two, the calf was standing but was still blind. She was also showing signs of torticollis. Elevation of feed and water enabled the heifer to eat and drink. On that day, the heifer was treated with 4 g thiamin IM in the morning and then 2 g thiamin IM that afternoon. She was given 3 million IU of Procaine Pen G IM BID.

The heifer was still blind on day three, and was treated with 2 g thiamin IM in the morning, and that afternoon she received 1.5 g thiamin IM. Three million IU of Procaine Pen G were administered IM BID. Feed and water continued to be offered to the calf periodically.

Because of the animal's hydration status, she was given one gallon rumen supernatum, 6 ounces of propylene glycol, and electrolytes* via stomach tube on day three. Again that evening, she received one gallon rumen supernatum and one-half package of electrolytes* through a gastric tube.

On day four, the heifer was still showing signs of blindness and was treated with 1.5 g thiamin IM SID. She was also given 3 million IU of Procaine Pen G IM.

The heifer remained in the clinic for nine days, at which time she was discharged. At the time of discharge, the heifer was still blind but seemed to be responsive to noises and movement and was able to follow shadows.

Follow-up revealed that after four months this heifer has regained her body condition and appears normal. The actual extent of her visual capabilities has not been determined.

Discussion

Polioencephalomalacia is a noninfectious neurologic disease which affects cattle, sheep, goats, antelope, and white-tail deer. It is characterized pathologically by cerebral edema, laminar necrosis of the cerebral cortex, and foci of necrosis throughout the brain.

It is thought that an inadequacy of thiamin is associated with polioencephalomalacia. Evidence to substantiate this includes the fact that affected animals respond to the parenteral administration of thiamin if given within a few hours after the onset of clinical signs. Also, affected animals have biochemical findings consistent with thiamin diphosphate inadequacy. Oral or intraperitoneal administration of large daily doses of pyrimidine containing structural analogs of thiamin, such as amprolium, has reproduced the clinical signs and pathological lesions of polioencephalomalacia in cattle and sheep.[1]

Thiamin in adequate amounts is usually derived in ruminants from symbiotic ruminal activity. Inadequacy could be a result of intraruminal thiamin destruction by either the enzymes of microbes or other dietary sources. These intrarumninal changes are seen in animals on rations rich in readily fermentable carbohydrates.[2] Thiamin inadequacy can also occur in concentrate-fed animals receiving insufficent roughage resulting in inadequate net microbial synthesis of thiamin in the rumin. Other causes for thiamin inadequacy in ruminants would include impaired absorption and/or phosphorylation of thiamin, lack of sufficient or appropriate apoenzyme binding for thiamin dependent systems, the presence of a thiamin inhibitor in the tissues of the host, increased metabolic

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demands for thiamin in the absence of increased supply, and increased rate of excretion of thiamin resulting in its net loss from the body.[1]

Naturally occurring thiaminases can also destroy thiamin. Significant amounts of these thiaminases have been found in the rumen contents of sheep and cattle affected with naturally occurring polioencephalomalacia.[1]

Further evidence of thiamin's role in polioencephalomalacia comes from the experimental induction of the disease by feeding diets containing 15% to 25% dried bracken fern rhizome, which is a natural source of type 1 thiaminase, a thiamin-free diet, or the thiamin antagonist amprolium. Lesions identical to polioencephalomalacia have been induced with a molasses-urea diet or a cobalt-deficient diet, which suggest that intraruminal thiamin degradation is but one route to a common metabolic end point.[3]

There is evidence that there may be a relationship between polioencephalomalacia in cattle and high-sulfate rations which contain at least 2% inorganic sulfate, usually gypsum, which is used as an intake-limiting additive. Three hypotheses which might explain a causal relationship between polioencephalomalacia and inorganic sulfate are ruminal degradation of thiamin by SO_4 or a ruminal product of SO_4, SO_4 inhibition of thiamin synthesis by alteration of the rumen flora or its nutrient balance, or a direct toxic action by a contaminant of the gypsum or other SO_4 salt used.[3]

Polioencephalomalacia is most common in cattle and sheep being fed concentrate rations under intensified conditions such as in feedlots. It usually occurs in well-nourished thrifty cattle 6-18 months of age which have been in the feedlot for several weeks. The disease can be reproduced in cattle fed a molasses-urea and roughage diet by gradual removal of the roughage without the use of thiamin analogs. It rarely occurs in adult cattle, possibly due to the greater quantities of roughage they usually consume.[1]

Outbreaks of polioencephalomalacia can occur suddenly with up to 25% of groups of feeder cattle being affected. Case mortality rates can range from 25-50%. Mortality rates are higher in young cattle (6-9 months) than in older age groups (12-18 months). If treatment with thiamin is delayed for more than a few hours after the onset of signs, mortality increases.[1]

Thiamin is a coenzyme in carbohydrate metabolism. A deficiency of thiamin would be expected to cause increases in the blood concentrations of pyruvic, lactic, and α-ketoglutariac acids and a decrease in the activity of the tissue enzyme transketolase, α-ketoglutarate dehydrogenase, and pyruvate dehydrogenase. The dependence of neurons and glial cells of the brain on carbohydrate catabolism using these enzymatic pathways accounts for the prominent neurologic signs seen with the disease.[2]

Polioencephalomalacia is characterized by three forms which comprise a continuum. The first is a convulsive form in which animals have convulsions, especially when excited. Impairment of sight is inconsistent. Animals tend to be spastic and incoordinated when they walk. These animals may die in a convulsion, progress to the third form and die in several days, or recover from any intervening stage.[4]

In the second form, there is bilateral blindness and fourth nerve paralysis. The animal can walk, but holds its head aloft and steps high with its front feet. Circling and head-pressing may be present, also. The animal may progress to the third form and die in several day or begin to recover from any intervening stage.[4]

With the third form, affected animals are blind with fourth nerve paralysis and are in lateral recumbency. Forelegs are usually extended and spastic. Paddling may occur with one or more legs. Animals can hear and are aware of manipulations, which they may resist. Opisthotonos is present when the animal is lifted to sternal recumbency. Spinal reflexes may be reversed in the spastic forelimbs. The animal resists manipulation of the feet by trying to shake free. The clinical manifestations are relative to the sites and extent of cerebral cortex involvement.[4]

The rumen is usually contracting, but the action is weak and infrequent. This is an important differential feature from lead poisoning, in which the rumen is invariably static.[1]

Menace reflex is always absent in the acute stage, and its slow return to normal following treatment is a valuable aid in prognosis.[1] Pupils are usually of normal size and respond to light. Dorsal strabismus is due to the stretching of the trochlear nerve. Nystagmus is common and may be vertical or horizontal.

Calves 6-9 months of age may die in 24-48 hours. Older cattle up to 18 months of age may survive for several days. Recovery is more common in the older age group.[1]

Cerebrospinal fluid usually contains normal (40mg/100ml) to mildly elevated protein levels and normal (5 WBC/mm^3) numbers of cells in acute cases. In chronic cases, mild to moderate elevations in protein concentration and white blood cell count may be due to cerebrocortical necrosis. Thiamine diphosphate is necessary for the formation of the enzyme transketolase. Blood transketolase activity declines before the onset of clinical signs and remains low until after treatment. Fecal thiaminase levels are frequently elevated in affected ruminants.[5]

Gross postmortem changes seen with polio-encephalomalacia include swelling and edema of the cerebrum with thickened and occasionally flattened gyri. Cerebral meningeal vessels may be congested. The gray matter of the cerebral cortex, especially the occipital lobes, may have focal or laminar areas of necrosis. Microscopic changes are found primarily in the gray matter and consist of neuronal necrosis, gliosis, neurophagia, and pericellular edema.[5]

Differential diagnosis should include heavy metal toxicity such as lead poisoning, space occupying mass such as an abscess or neoplasm, nitrofuran toxicity, hypomagnesemia, vitamin A deficiency, chlorinated hydrocarbon toxicity, hepatic encephalopathy, nervous ketosis, Type-D clostridial entero-toxemia, and infectious thromboembolic meningoencephalitis.

Treatment with thiamin hydrochloride at a dose of 10 mg/kg initially should be administered intravenously as early in the course of the disease as possible. Treatment should be repeated intramuscularly twice daily for 2 to 3 days. Clinical improvement should be observed in 1 to 3 days. Complete resolution of signs may be observed in one to several days, or some cerebral damage may be irreversible, resulting in some degree of blindness, depression, or ataxia. Blindness may be the last sign to resolve, and this may require 1 to 7 days.[5]

Anti-inflammatory and anti-edema drugs may also be helpful in the treatment of the disease. These would include mannitol, dexamethasone, prednisolone, furosemide, and dimethyl sulfoxide.[5]

Affected animals should be placed in a well-bedded stall. Fluids, electrolytes, and nutrients should be administered orally or intravenously if the animal is anorectic or dehydrated. Antibiotics should be given to prevent aspiration pneumonia in animals which are recumbent or dysphagic.[5]

If a specific cause can be found, it should be eliminated. Ruminants on high concentrate rations need to have the diet changed slowly to include more roughage and less carbohydrate. Dietary supplementation of thiamin at a rate of 3 to 5 mg/kg of feed should decrease the incidence of the disease if many animals are affected.[5]

Prognosis can be improved significantly if the signs of polioencephalomalacia are recognized early in the course of the disease and therapy is instituted promptly.

References

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