Cerebral Disorders of Calves

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KEYWORDS

• Hypernatremia • Sodium toxicity • Polioencephalomalacia • Meningitis • Neurology

KEY POINTS

- The most common causes of cerebral disorders in calves are polioencephalomalacia (PEM), bacterial meningitis (usually in calves less than 2–3 weeks of age), and hypernatremia (salt poisoning).
- PEM can be caused by several things, including thiamine deficiency and sulfur toxicity, as well as other dietary factors. Regardless of cause, thiamine supplementation is the treatment of choice in cattle suspected of having this disease.
- Bacterial meningitis generally occurs in calves less than 2 weeks of age as a sequelae of septicemia and failure of passive transfer. Treatment of meningitis is extremely difficult and, therefore, effort should be placed on prevention.
- Hypernatremia (salt poisoning) is becoming recognized more frequently in calves as related to ingestion of high sodium levels during treatment of diarrhea or possibly from ingestion of water with high sodium. Treatment requires careful attention to reduce sodium concentrations slowly over time to prevent the formation of cerebral edema.

Video content accompanies this article at http://www.vetfood.theclinics.com.

Diseases affecting the forebrain or cerebrum are probably the most common neurologic conditions encountered in cattle. In general, clinical signs attributable to cerebral disease are characterized by abnormal mentation with a normal gait and posture. Clinical signs suggestive of abnormal mentation can include circling, blindness, mania, aggressive behavior, and seizure activity, along with what is often described as head pressing. The list of diseases that could cause cerebral disease in cattle is long and includes lead toxicity, polioencephalomalacia (PEM), bacterial meningitis, thromboembolic meningoencephalitis due to *Histophilus somni*, Bovine herpesvirus type 1, rabies, nervous coccidiosis, brain abscess, pseudorabies, urea toxicity, and malignant catarrhal fever. Also included are metabolic disturbances such as hypernatremia,

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hypocalcemia, hypomagnesemia, hypokalemia, hepatic encephalopathy, Vitamin A deficiency, and severe acid-base derangements.[1] Consideration of the signalment and history of the animal is critical when investigating neurologic disease. Several of the previously listed diseases could affect dairy or beef calves under 6 to 9 months of age. A brain abscess is always possible in young calves, although they are relatively uncommon. Typically, these would show a slow but progressive onset of clinical signs resulting from the space-occupying lesion in the cerebrum. Thromboembolic meningoencephalitis and nervous coccidiosis can also be seen in feedlot calves on occasion and warrant mention. However, the most common causes of cerebral disorders in calves are PEM, bacterial meningitis (usually in calves less than 2–3 weeks of age), and hypernatremia (salt poisoning). These are the primary focus of this article.

POLIOENCEPHALOMALACIA

PEM or cerebrocortical necrosis is a common problem encountered in calves, especially those in feedlots. The disease has been associated with thiamine deficiency, high-sulfur diet, low-roughage diet, high doses of amprolium, cobalt-deficient diet, and molasses-urea diet, as well as ingestion of various toxic plants.[2–4] Although there may not be an exact cause, certainly nutrition plays an important role in the development of the disease. Histologic lesions are primarily necrosis of the gray matter in the brain, similar to lead and/or salt toxicity. In calves, the greatest risk period is between 6 to 18 months of age, with higher susceptibility in younger animals due to lower blood thiamine concentration and intensive feeding systems.[5]

Pathophysiology

Thiamine deficiency has long been recognized for its role in the pathophysiology of PEM in calves. Preruminants depend on ingestion of dietary thiamine, whereas older ruminants rely on rumen bacterial production of thiamine to meet their daily requirement. Thus, when normal rumen production of thiamine is altered or when ingestion is reduced, animals become at risk of deficiency.[3] Brain lesions are thought to be due to impairment of glucose metabolism. Thiamine is known to be necessary in glucose metabolism, playing a role in the Krebs cycle. Insufficient brain glucose will decrease the energy supply to the brain, reducing lipid synthesis, acetylcholine, and other neurotransmitters.[6] Neuronal function will be altered and will eventually lead to clinical signs of PEM.

Bacterial thiaminase has been considered the main factor leading to thiamine deficiency in ruminants with PEM. Thiaminase type I acts by catalyzing the cleavage of thiamine at the methylene bridge between the pyrimidinyl and thiazole ring. Cosubstrate (benzimidazoles, levamisole, or promazine) is required to combine with the pyrimidinyl to form a new compound. Thiaminase type II catalyzes the hydrolysis process between the 2 ring structures of the thiamine molecule. Toxic plants, such as bracken fern (*Pteridium aquilinum*), horsetail (*Equisetum arvense*), and Nardoo fern (*Marsilea drummondii*), contain thiaminase similar to thiaminase type I and have been incriminated in outbreaks of PEM.[4]

Several cases of PEM have been associated with either normal levels of thiamine or treatment failure after thiamine supplementation, therefore other causes were thought to exist than just simple thiamine deficiency. More recently it was discovered that PEM could also be caused by sulfur toxicity. Since the first report of this form of PEM, many cases have been associated with high sulfur in the feed or water.[7–11] The complete pathophysiology has not yet been fully elucidated but was initially thought to be due to inhalation of high quantities of hydrogen sulfide (H_2S) gas eructated by the