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Nervous Coccidiosis in Calves

Larry D. Hauptmeier, DVM
Extension Veterinarian
Iowa State University

A variety of clinical neurological syndromes exists in stocker and feeder cattle. Causes may be infectious, nutritional/metabolic, or toxins. A less common syndrome is referred to as "nervous" coccidiosis, named so because of the observation that many of the calves that experience this neurological syndrome concurrently exhibit clinical enteric coccidiosis.⁽¹⁾ This entity was first reported in 1921.⁽²⁾

Nervous coccidiosis can occur at any time of the year but appears most often in the fall and early winter, coinciding with the time of the year when many calves move to feedlots. This syndrome reportedly can occur in up to 30% of the calves affected with enteric coccidiosis. As many as 10,000 cattle die annually in the U.S. from this problem. The pathogenic mechanisms for nervous coccidiosis, however, are not clearly resolved.⁽¹⁾ Coccidial organisms or microscopic lesions consistent with coccidial infection cannot always be found in affected calves.⁽³⁾ Researchers have not been able to experimentally reproduce this syndrome. The absence of significant brain lesions eliminates infectious and some nutritional factors and toxic agents as primary causes.⁽¹⁾

Clinical signs of nervous coccidiosis may vary in severity and frequency and may range from minor muscular incoordination, twitching, and loss of balance to intermittent or continuous seizures. During seizures, affected calves collapse into lateral recumbency and exhibit a variety of signs, including opisthotonos, tetany, medio-ventral strabismus, nystagmus, paddling movements, exaggerated snapping of the eyelids, salivation, star gazing, nervousness, occasional bellowing, and rapid and irregular respiration. Affected calves may get up and experience periods of apparent normalcy between seizures. Seizures often recur when the calves are stressed or handled.⁽¹⁾

Conflicting theories have been proposed to explain this syndrome:

1) One school of thought proposes that this malady in calves and yearling cattle is not due to the coccidia *per se* but to a combination of stressors, including the damage by the coccidia to the intestinal tract, which results in tissue magnesium and serum calcium depletion and the production of the neurological syndrome observed. To support this theory, proponents point out that treatment of affected animals with intravenous and subcutaneous calcium, magnesium, and dextrose solution, balanced electrolyte solutions, oral magnesium oxide and sulfamethazine has been relatively successful when given early, while the animal is still able to stand and appears normal until disturbed. In the later stages, however, whether the animal is standing or recumbent, treatment has been disappointing.⁽²⁾

2) Recently, researchers demonstrated that calves with nervous coccidiosis had a lower liver copper concentration than calves experiencing coccidiosis without neurological signs. The results of this same study suggest that the following entities are not involved in the pathogenesis of nervous coccidiosis: disturbance of serum Na, K, Ca, P, and/or Mg concentration, vitamin A deficiency, thiamine deficiency, anemia, lead intoxication, uremia, *Hemophilus somnus* meningoencephalitis, severity of coccidial infection, gross alterations in intestinal bacterial flora, and hepatopathy.⁽¹⁾

3) In a subsequent study by the same researchers, a neurotoxin reported to be present in the serum of calves with nervous coccidiosis was not found in the serum of control calves or calves with only enteric coccidiosis. These authors state that the significance of this labile neurotoxin with respect to

the pathogenesis of the neurological signs associated with bovine enteric coccidiosis is unknown. This neurotoxin causes muscle tremors, tachypnea or dyspnea, star gazing, loss of righting reflex, seizures and death in mice when serum from neurologically affected calves is inoculated intravenously. Serum from control calves and from calves with enteric coccidiosis, but not exhibiting neurological signs, does not cause these alterations in behavior when inoculated into mice at the same dosage.⁽⁴⁾

Other proposed hypotheses for causes of nervous coccidiosis include: uremia, anemia, lead toxicity, thiamine deficiency, vitamin A deficiency, hypoglycemia, electrolyte imbalance (Mg, Na, K, P, and/or Ca), absorption of a toxic material through the damaged gut wall, toxin production by coccidia, host immune response to coccidia, gross alterations in intestinal bacterial flora, hepatopathy, and toxemia.⁽¹⁾

The clinical signs of nervous coccidiosis are similar to those of other neurological diseases that affect the function of the cerebral cortex.⁽⁵⁾ Differential diagnoses include lead poisoning, polioencephalomalacia, TEME, listeriosis, rabies, sporadic bovine encephalomyelitis, and salt poisoning. Consideration of history, clinical examination, necropsy, laboratory tests, histopathology, and response to treatment should differentiate these diseases.⁽²⁾

Multiple etiologies appear to be involved and must be present at the same time or in proper sequence to see clinical "nervous" coccidiosis. Some form of large bowel disease/inflammation, usually associated with enteric coccidiosis, and the absence of significant brain lesions are the only constant features of the disease. Placing the affected animals in a warm, quiet environment reportedly helps to alleviate clinical signs and may facilitate recovery. The recovery rate is quite low, especially in severely affected cases. In spite of therapy and supportive management, the mortality rate has been reported as 72% in one part of the U.S. and 90% in another area.⁽¹⁾

The author's experience with this problem in the field closely parallels that cited in the literature except that I would emphasize the virtues of early treatment and the pessimism associated with late treatment. Treatment of choice consisted of parenteral sulfonamides the first day followed by oral administration of sustained release boluses; antibiotics to treat and prevent

secondary infection; plus 250-500 ml calcium solution with magnesium and glucose, often repeated on the second day. A non-steroidal anti-inflammatory product was given to control toxin absorption from the gut. This is extra-label use and must be administered by or under the supervision of the attending veterinarian and requires a veterinarian-client-patient relationship.

The most logical approach to the problem of nervous coccidiosis is the prevention of all coccidiosis through good herd management, sanitation, reduction of stress, and the use of coccidiostats.⁽²⁾ My personal preference is to start all incoming feeder cattle on a coccidiostat in the diet for 28 days.

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