"Nervous" Coccidiosis in Calves

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A variety of clinical neurological syndromes exist in stocker and feeder calves. These include infectious, nutritional/metabolic and toxic etiologies. One less common neurological entity in newly weaned calves is a condition referred to in the literature as "nervous" coccidiosis.[1,2,3,4] This nomenclature is predicated on the observation that many of these calves with this neurological syndrome concurrently have clinical enteric coccidiosis.

Even though not a routinely observed entity in all areas of the cattle industry, this syndrome reportedly can occur in up to 30% of the calves affected with enteric coccidiosis[4] and as many as 10,000 cattle die annually in the United States from this syndrome.[5] However, at this point and time, the pathogenic mechanisms for nervous coccidiosis are not clearly resolved. Because coccidial organisms or microscopic lesions consistent with coccidial organisms or microscopic lesions consistent with coccidiosis cannot always be found in some of these calves, I sometimes refer to this entity as nervous enteritis.

Cases of nervous coccidiosis occur year around but are most numerous during the fall and early winter months. In our area, this is the time of the year we get the largest influx of freshly weaned stocker and feeder calves.

Clinical signs of nervous coccidiosis may vary in severity and frequency and 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, medioventral strabismus, nystagmus, paddling movements, exaggerated snapping of the eyelids, frothing at the mouth, star gazing, rolling of eyes, nervousness, occasional bellowing, 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.

Placement in a warm, quiet environment reportedly helps somewhat to alleviate clinical signs and may facilitate recovery.[6] In spite of intense therapy and supportive management, the mortality rate in cases of nervous coccidiosis is quite high. Calves may die on the first day of illness or can survive as long as three to five days.[4] Recovery rate is especially poor in severely affected calves. In one survey of veterinarians treating this condition in the field, 72% died.[7] Unofficial reports from veterinarians in the Texas Panhandle suggest that mortality in this region exceeds 90%.

The pathogenesis of nervous coccidiosis is still unknown and therefore, researchers have not been able to experimentally induce the neurologic manifestation of coccidiosis. The absence of significant brain lesions eliminates infectious causes and some nutritional and toxic etiologies.

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 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, toxemia, and copper deficiency.[6]

One of the more recent papers was a prospective study of cases of bovine enteric coccidiosis with and without nervous signs.[6] The results of this study indicated that calves with neurological signs had a lower liver copper concentration ($p<0.01$) than calves without neurological signs. The results of this same study suggest that disturbance of serum Na, K, Ca, P, or Mg concentration, vitamin A deficiency, thiamine deficiency, anemia, lead intoxication, uremia, *Haemophilus somnus* meningoencephalitis, severity of coccidial infection, gross alterations in intestinal bacterial flora and hepatopathy are not involved in the pathogenesis of "nervous" coccidiosis.[6]

In a subsequent study[8,9] these same authors reported a neurotoxin to be present in the serum of "nervous" coccidiosis calves that is not found in the serum of control calves or calves with only enteric coccidiosis. Mouse inoculation was used to test serum, cerebrospinal fluid and intestinal contents for the presence of a toxin. Severe neurological signs such as loss of righting reflex, seizures and death occurred only in the mice given the serum from calves with nervous coccidiosis. Inter-

estingly enough, copper deficient mice injected with the serum from the nervous coccidiosis calves did not have the neurological signs demonstrated by mice on normal dietary levels of copper.[8] This observation suggests that the copper deficiency observed in the nervous coccidiosis calves may not be primary in the production of this disease if the toxin demonstrated in the sera of these calves is related to the pathogenesis of nervous 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.

It appears that multiple etiologies may 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 brain lesions are the only constant features of this disease.

References

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