An Atypical Case of Polioencephalomalicia

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Polioencephalomalacia (PEM) is a significant feedlot cattle problem here in the midwest. Differentiation of this disease from other diseases that produce central nervous signs such as acute lead poisoning (ALP), thromboembolic meningoencephalomyelitis, hypomagnesemia, and rabies is important to the practitioner as he has to decide on the best course of therapy for recovery. (2,3) Because the following atypical case of PEM looked so much like ALP to the group of clinicians (bovine clinicians, toxicologists, and pathologists) who observed it, we feel it is worthwhile to report.

On October 14, 1970, three Angus cattle, one twelve year old cow and two yearling heifers all weighing about 1100 pounds were admitted to the Iowa State University Veterinary Clinic exhibiting central nervous signs. On October 9, 1970, the herd of Angus cattle that these animals were from was taken from a poor quality pasture to a good quality pasture. Two days later on October 11, the twelve year old cow was found to be blind. Further investigation revealed about ten more cows out of a herd of 150 were exhibiting central nervous signs. The attending veterinarian tentatively diagnosed ALP and quizzed the owner as to possible sources of lead such as dumps on the pasture, paint, and painted buildings; but no sources were discovered. There had been no deaths at the time these three cows were admitted to the clinic.

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Clinical Signs

All three animals were blind and remained so throughout the course of the disease. They showed head bobbing and were grinding their teeth. The older cow had fallen in the trailer during the trip to the clinic and was unable to rise. The other two were reluctant to move and showed head pressing. One of the heifers periodically went into convulsions.

Disease Course

Upon entry into the clinic the aged cow was recumbent and considering the gravity of the situation, it was decided to euthanatize and perform a necropsy on this cow. The two remaining heifers were examined clinically and a tentative diagnosis of lead poisoning was made. CaEDTA therapy was initiated at the rate of 250 cc every 12 hours for the first 36 hours.

On the 2nd day both seemed more alert, less reluctant to move, and showed less head hobbing. The following day they continued to show improvement, characterized by absence of convulsions, head bobbing and grinding of the teeth.

On October 17th, the histopathology report on the cow came back with a diagnosis of PEM. At this time therapy with thiamine hydrochloride was instituted and oral fluids were given to combat dehydration. The thiamine hydrochloride was given intramuscularly at the rate of 1000 milligrams per day for 3 days.

There was no appreciable change in their condition until October 22, when one of the heifers showed pharyngeal paraly-

sis. Therapy on this animal was changed to include intravenous fluids. The temperatures of these heifers were within the normal ranges throughout the course of the disease until October 23rd. On this day the heifer with the pharyngeal paralysis showed a temperature of 104° F.; labored breathing, moist râles, and ingesta was seen in the nares. At this time since there was no clinical improvement in either animal it was decided both animals should be sent to post mortem.

Necropsy Findings

Gross examination of the older cow showed no significant lesions. The other two being further along in the course of the disease showed some slight malacia of the cerebrum. The two heifers had froth in their tracheas, and their lungs had dark red edematous areas of consolidation in the ventral lobes. These two showed focal hemorrhages of the serosa of the intestines, fibrinous peritonitis, and adhesions.

Histopathology

The old cow had lipofuscin in the cardiac muscle with mild areas of interstitial inflammation and sarcosporidia. The kidneys showed tubular degeneration and regeneration with protein in the tubules. Microscopic sections of the brain revealed many significant findings: degeneration of the Purkinji cells in the cerebellum; neuronal degeneration; and mild gliosis in a rather uniform layer beneath the cerebral cortex. The other two showed some mild bile duct hyperplasia, but more important showed permanent damage to the brain in the form of laminar edema and malacia of the cortex with vasculitis, gitter cells concentrated around the vessels, and reactive astrocytes (lesions of PEM).

Discussion

Rabies was ruled out by the clinical signs, histopathology, and negative florescent antibody test. Hypomagnesemia was ruled out due to the time of year but

Laboratory findings—

October 14			
RBC	6.5 million,	6.7 million,	7.1 million
WBC	6,000	12,000	9,000
PCV	34.5%	33%	34.5%
Hb	12.8 mg%	11.8 mg%	11.8 mg%
Differential			
Segs.	77%	59%	43%
Bands	0	5%	1%
Lymphs	22%	36%	56%
Monos	1%	0	0
October 23			
RBC		7.8 million,	10.0 million
WBC		4,900	5,900
PCV		35%	44%
Hb		12.0 mg%	14.8 mg%
Differential			
Segs.		13%	11%
Bands		0	12%
Lymphs		86%	75%
Monos		1%	2%

Lead levels—0.10, 0.085, and 0.07 ug/ml. These are not significant unless they are 0.3 ug/ml or above.

mainly due to the histopathology. Thromboembolic meningoencephalomyelitis was ruled out by the clinical signs, lack of febrile response early in the disease, and histopathology. Due to the lack of severity of clinical signs this case of PEM was mistakenly diagnosed as ALP by the referring veterinarian and ourselves. It may have been coincidental, but it seemed the most response came at the time of the CaEDTA therapy. This tended to support the ALP diagnosis. The lack of response to the thiamine could well have been due to the late diagnosis of PEM and thus late initiation of treatment. The low blood lead levels obtained from samples submitted to the Iowa State Diagnostic Laboratory and the National Animal Disease Laboratory ruled out ALP. When the histopathology results were obtained, PEM was the definitive diagnosis. A case like this points out the necessity of utilizing state diagnostic laboratories to either confirm or disprove a clinical diagnosis even though the clinical signs may be typical of a disease. As professionals and future professionals we have an obligation to strive for perfection.

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