Polioencephalomalacia in a captive fallow deer

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Polioencephalomalacia (PEM) is a noninfectious disease affecting primarily the gray matter of the brain. Polioencephalomalacia, or cerebral cortical necrosis (CCN), has been described worldwide in cattle and sheep. There have been relatively few reported occurrences of PEM in cervid species; it has been described in three adult white-tailed deer (Odocoileus virginianus) (1,2) and one very young fallow deer (Dama dama) fawn (3). The clinical signs described have been very similar to those seen in domestic species and the diagnosis involved histological evidence of laminar neuronal necrosis in the cerebral cortex. Grossly, various lesions have been noted in the central nervous system (CNS): flattened gyri, swelling and caudal displacement of the brain, softening and yellow discoloration of the cerebral cortex, focal areas of cavitation, and bilateral petechial hemorrhage within the brain.

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The raising of deer in captivity is a quickly growing specialty, no doubt due to interest in agricultural diversification. Because of the early stage of this industry in Canada, there has not been much opportunity to study problems encountered by deer farmers. We describe herein a case of PEM in a fallow deer.

A two and one-half year old, 35 kg fallow doe was one of a mob of 19 deer being raised on a farm in Saskatchewan. Throughout the winter the deer were fed a ration of alfalfa hay and barley supplement. The barley had been fed at 0.5 kg per animal per day until one month prior to the onset of the problem described herein. At that time, a doe died of what was diagnosed at necropsy to be grain overload. The owners were advised to reduce the amount of barley fed.

The owners noticed that a doe was ostracized by the rest of the mob on the day before she was submitted for examination. She had a slight head bob at that time. The following day she was slightly ataxic and was seen running into a fence as though blind. When she arrived at the large animal clinic at the Western College of Veterinary Medicine, she was not showing normal escape behavior. On physical examination, she was in good body condition. The mucous membranes were very congested, with a capillary refill time of less than one second. She had head and neck tremors and was making smacking, chewing movements. Her gait was very ataxic but she was strong and resisted being held. There was no menace response, the pupillary light reflex was intact, and she blinked when a light was shone into her eyes. Results of the neurological examination, along with the head and neck tremors, were consistent with cerebellar dysfunction (4).

Supportive therapy included twice daily treatment with oral electrolytes administered by esophageal intubation, as her swallowing reflex was compromised during the head tremors. The differential diagnoses included cerebral abscess, cerebrospinal nematodiasis, encephalitis (bacterial or viral), polioencephalomalacia, lead poisoning, rabies, and spongiform encephalopathy (5).

Blood was collected at the time of admission. The only significant findings were an increased packed cell volume and total protein, reflecting the fact that the deer had not been able to drink. There was elevation of aspartate aminotransferase (AST) and creatine kinase (CK), probably due to damage from excessive muscular activity and from bumping and falling because of ataxia.

On the second day after admission, a cerebrospinal tap was performed under xylazine sedation (0.3 mg/kg IV) (Bayvet Division, Chemagro Limited, Etobicoke, Ontario). This procedure involved location of the lumbosacral (LS) cistern on the dorsal midline just caudal to a line joining the crests of the ilia. A 20 ga 5 cm spinal needle was inserted, at 45° to 60° from the horizontal, into the LS cistern. The attached syringe filled immediately to 3 mL, indicating considerable cerebrospinal fluid (CSF) pressure. The CSF was examined cytologically and cultured for bacteria.

The doe was treated with dexamethasone (0.04 mg/kg IM) (rogar/STB, Pfizer Animal Health Division, Calgary, Alberta) and thiamine hydrochloride (1.4 mg/kg IM) (Langford Inc., Guelph, Ontario). The use of oral electrolytes was continued. On the morning of the third day, the doe was found in lateral recumbency with severe opisthotonus. She was given thiamine HCl (250 mg IV slowly, and 350 mg IM). The CSF collected on day 2 had a cellular component within normal limits, (WBC 3.3 × 10⁶/L, RBC 4.4 × 10⁶/L); the protein was moderately elevated (1.48 g/L). This was considered to be a nonspecific finding. Bacterial culture was negative. The deer died on day 3 and was sent for necropsy.

The doe was in good body condition and was pregnant with a four-month-old fetus. Grossly abnormal findings were limited to the CNS. There was cerebral edema with coning of the cerebellum through the foramen magnum and some flattening of the lateral gyri of the cerebral hemispheres. Histological examination of various tissues revealed congestion and hemorrhage of the intestinal tract, pulmonary congestion and edema, and renal and hepatic congestion, consistent with terminal shock.
tissue edema of the brain. Gray matter cortex. Limits. Described and deeper and acute necrosis of the relatively shrunken and more of the matter, and affected with laminar of the pyknotic nuclei (arrows) and with increased perineuronal spaces. Bar = 5 μm. H & E stain.

Microscopic lesions in the brain were similar to those described in other species with PEM (1,6–8). There was acute necrosis with laminar distribution in the cerebral cortex. There were lines of vacuolation of the neuropil in the gray matter, following the corona radiata. In these areas there was marked increase of the perivascular spaces. Affected neurons were shrunken, angular, and hyperchromatic, with perineuronal vacuolation (Figure 1). This laminar necrosis of the gray matter tended to be more distinct in the middle and deeper layers of the gyri. In some regions, there were two affected laminae separated by a strip of relatively normal tissue. There was laminar status spongiosus of the neuropil, indicative of the severe tissue edema which had been noted on gross examination of the brain.

Brain lead levels (0.002 μmol/g) were within normal limits.

The gross and histological findings were compatible with a diagnosis of PEM. The clinical development of the disease and the CNS lesions were also similar to those described in other species (1,6–8).

The etiology of PEM is multifactorial, but is ultimately associated with decreased availability of thiamine in the CNS (6,9–11). Daily requirements for thiamine vary with species, metabolic activity, feed intake, and dietary composition. Requirements increase during pregnancy and lactation, and when the carbohydrate level of the diet is high (6). In the cases of PEM described in cervids, one white-tailed deer had a rumen full of corn (1), whereas two others had small amounts of wheat and barley in their rumens (2). Several cases of PEM have been seen in pronghorn antelope (Antilocapra americana) and these animals have had grain in their rumens at the time of death (12). Descriptions of goats with the disease have included histories of supplemental feed high in carbohydrates (8). The feed supplement of the deer in this case had been barley, a high energy feed.

Although there was no confirmation of thiamine deficiency or any conclusive etiological diagnosis in this case, the rest of the deer in the mob were treated with parenteral thiamine HC1, the barley component of the diet was replaced with a balanced herbivore ration, and no furthur problems were encountered.

References