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J Vet Diagn Invest 10:82–84 (1998)

Probable elaeophorosis in a moose (*Alces alces*) from eastern Washington state

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On October 20, 1995, a free-ranging yearling female moose (*Alces alces*) was presented to the Washington State University College of Veterinary Medicine, Pullman, for evaluation of blindness and ataxia. Two days previously, the moose had been found blind and staggering on the Colville Indian Reservation (48°15'N, 119°10'W) in eastern Washington. The moose was in fair body condition with a rectal temperature of 100.5 F (38.1 C). When walking, the moose had a hypermetric gait and would stumble into objects. A head tilt to the right was observed. The eyes were normal with the exception of mild scleral injection and miotic pupils. The menace response was absent, with intact corneal and palpebral reflexes. A 6- × 6-cm reddened, alopecic area was noted behind the right ear. A painful response was elicited upon extension of the neck at the base of the skull and on palpation of the temporal region. Head trauma was considered a likely initial diagnosis; no abnormalities were noted on lateral radiographs of the skull and craniocervical spine. Thirty milligrams of dexamethasone^a and 150 mg of flunixin meglumine^b were administered intramuscularly, and the moose was hospitalized for further evaluation of neurologic signs. Overnight observations included head pressing, aimless wandering, and circling to the right and episodes of hyperventilation and vocalization. No improvement was noted the following morning. Attempts by the moose to eat and drink were unsuccessful, and euthanasia was performed with 24 g sodium pentobarbital^c given intravenously.

At necropsy, the moose weighed 140 kg and was in good postmortem condition. Two 5- × 5-cm areas of subcutaneous hemorrhage were noted next to the sternum in addition to a 12- × 4-cm area of subcutaneous hemorrhage between the eyes. The alopecic area behind the right ear was overlying normal subcutaneous tissue. A prominent leptomeningeal vascular pattern was noted over the right cerebral hemisphere. Evidence of trauma was considered minimal, with areas of subcutaneous trauma attributed to minor trauma associated with ataxia and blindness. Representative samples of brain, eyes, skin, heart, mesenteric lymph nodes, spleen, lungs, thyroid glands, gastrointestinal tract, adrenal glands, liver, kidney, ovary, and bladder were preserved in 10% formalin, sectioned at 6 μm, and stained with hematoxylin and eosin (HE) for histologic evaluation.

Histopathologic findings were predominantly within the brain. The cerebrum was characterized by multifocal malacia and vasculitis (Fig. 1). The most severe lesions were noted in the middle and caudal cerebrum. Many small blood vessel walls were edematous and had fibrinoid degeneration, and the walls were expanded by inflammatory cells, which were predominantly lymphocytes with smaller numbers of neutrophils and eosinophils. Many arterioles of the leptomeninges and left and right cerebrum had mild to extensive expansion of the tunica intima by eosinophilic spindle cells interpreted to be smooth muscle (Masson's trichrome stain) (Fig. 2). Blood vessels surrounding areas of malacia were often thrombosed. A large artery adjacent to a mesenteric lymph node contained a cross section of a nematode, which was approximately 600 μm in diameter (Fig. 3). Other histologic findings included a small focal area of myocardial fibrosis and an eosinophilic ulcerative dermatitis with granulation tissue replacement associated with the reddened, alopecic area behind the right ear. The eyes were histologically normal.

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Received for publication January 13, 1997.

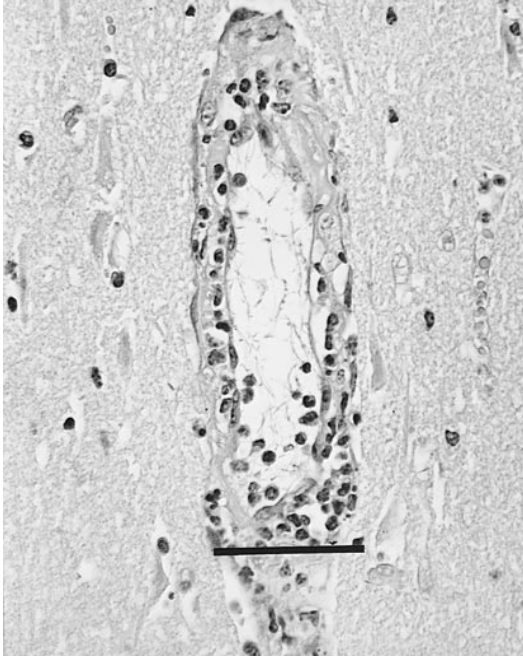


Figure 1. Cerebral artery of a moose, with vasculitis and fibrinoid degeneration. HE. Bar = 60 μm .

The posterior fragment of the nematode observed in a blood vessel adjacent to a mesenteric lymph node was recovered from the paraffin block used to prepare the histologic sections. The blood vessel was cut out of the block with a razor blade, deparaffinized in xylene, and mounted

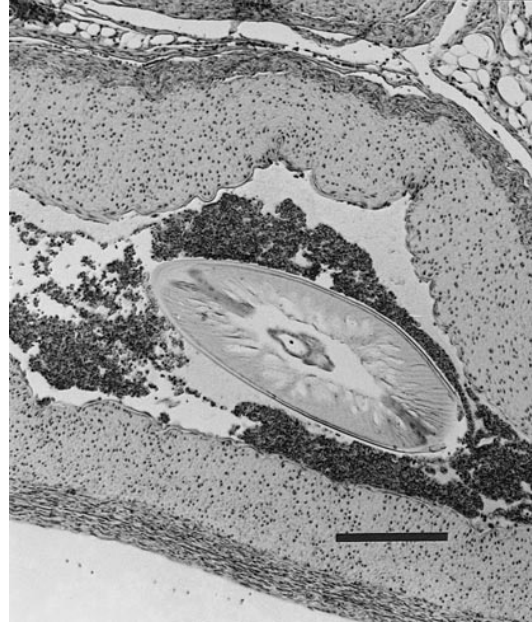


Figure 3. Immature *Elaeophora* in cross section of an artery of a moose, adjacent to a mesenteric lymph node. HE. Bar = 200 μm .

on a glass microscope slide in glycerol. Microscopically, the nematode was not sheathed and had a blunt tail, and the cuticle had prominent transverse striations (Fig. 4) characteristic of *Elaeophora*.³ The transverse striations were in bands 3–4 μm wide, and each band was separated by approximately 14 μm .

Based on the clinical signs, histopathologic findings, and the recovery of an immature nematode morphologically con-

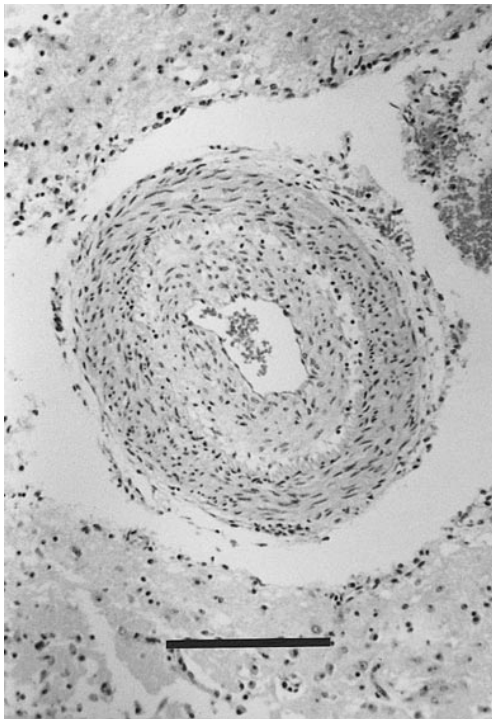


Figure 2. Leptomenigeal artery of a moose, with extensive intimal proliferation. HE. Bar = 200 μm .

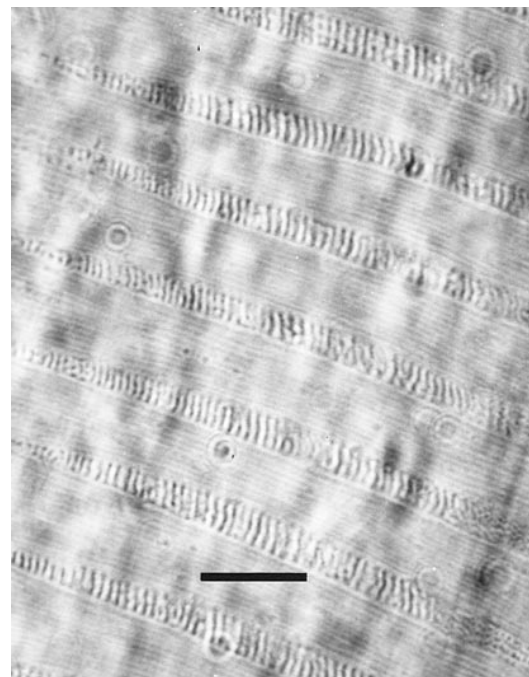


Figure 4. Posterior section of immature *Elaeophora*, illustrating the prominent cuticular striations (arrows). Bar = 20 μm .

sistent with *Elaeophora*, a diagnosis of elaeophorosis, probably caused by *E. schneideri*, was made. Blindness, circling, and ataxia as seen in this moose are characteristic clinical signs of infection with *E. schneideri* in moose from Montana⁵ and Colorado.⁴ Histopathologic findings in moose elaeophorosis are similar to those of elk (*Cervus elaphus*)⁵ and include regions of cerebrocortical necrosis, intimal proliferation within leptomenigeal arteries, and thrombosis of cerebral vessels. The vasculitis reported in this moose has been described in elk with elaeophorosis.¹ Both previous reports of elaeophorosis in moose contained descriptions of nematodes within histologic sections of brain, and nematodes were recovered from the carotid arteries.^{4,5} Despite examination of numerous brain sections and other tissues, only the nematode within an artery surrounding a mesenteric lymph node was observed; the carotid arteries were not examined grossly. The observation of the larval nematode within a mesenteric vessel is not surprising based on observations in elk with elaeophorosis in which parasites were observed within multiple organ systems. Postmortem migration of nematodes was also thought to occur.¹ The cause of the observed eosinophilic dermatitis was not determined and although a localized reaction to circulating *Elaeophora* microfilariae was considered, no microfilariae were observed on histologic examination.

This is the first report of elaeophorosis in a moose from Washington state and the first description of this parasite from the eastern portion of the state. The only previous report of elaeophorosis reported from Washington state was *E. schneideri* from a black-tailed deer (*Odocoileus hemionus columbianus*) from the western portion of the state.² Al-

though *E. schneideri* is considered rare in Washington,² moose may be a sensitive indicator species for the presence of the parasite, especially in areas where the parasite has not been diagnosed previously.

Acknowledgements. We thank Dr. Melinda Wilkerson, Washington Animal Disease Diagnostic Laboratory, for assistance with histopathology, Dr. Scott Lyell Gardner, University of Nebraska State Museum, for assistance in examination of the parasite, and Steve Judd, the Colville Indian Tribe, for bringing this moose to our attention.

Sources and manufacturers

- a. Phoenix Pharmaceutical, St. Joseph, MO.
- b. Professional Pharmaceuticals, Omaha, NE.
- c. Beuthanasia-D Special, Schering-Plough Animal Health, Kenilworth, NJ.

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J Vet Diagn Invest 10:84–86 (1998)

Development of a serum plate agglutination test to detect antibodies to *Ornithobacterium rhinotracheale*

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Ornithobacterium rhinotracheale is a pleomorphic gram-negative rod-shaped bacterium that has been isolated from chickens and turkeys manifesting severe respiratory problems in Germany, South Africa, The Netherlands, France, Israel, Belgium, Hungary, and the United Kingdom.^{5,8–11} In 1993, an organism with similar characteristics was isolated for the first time in California from chickens and turkeys exhibiting respiratory problems.⁴ In late 1995, *O. rhinotracheale* was associated with increased mortality and condemnation due to airsacculitis in market age tom turkeys in Minnesota and Wisconsin.¹

Clinical signs seen in birds infected with *O. rhinotracheale* include coughing, nasal discharge, arthritis, prostration, drop in egg production, growth suppression, and mortality.^{4,6,9} The gross pathologic changes in turkeys with *O. rhinotracheale* infection are edema and consolidation of lungs with fibrinopurulent exudate, pericarditis, hepatomegaly, and airsacculitis, whereas in chickens pneumonia and airsacculitis with exudate have been observed.^{2,6,8} In some cases, mortality may be $\leq 10\%$.² In addition, condemnation at the processing plant because of *O. rhinotracheale* infection has resulted in economic losses to producers.

Infected birds currently are diagnosed by the isolation of the organism, mainly from the lungs and trachea. *Ornithobacterium rhinotracheale* grows on blood agar at 37 C in the presence of 5% CO₂, does not grow on MacConkey agar,

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Received for publication January 13, 1997.