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ELAEOPHOROSIS IN MOOSE FROM MONTANA*

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Abstract: The ruminant arterial worm *Elaeophora schneideri* is reported for the first time from the moose, *Alces alces*, in Montana. One of four infected animals examined during 1971 was blind and had extensive intimal proliferation in the leptomeningeal arteries, areas of cerebrocortical neuronal necrosis, and numerous hemorrhages in the brain. Similarities were noted in the lesions of elaeophorosis in moose and wapiti.

INTRODUCTION

The ruminant arterial worm *Elaeophora schneideri* Wehr and Dikmans, 1935 has been recognized as a cause of dermatosis in domestic sheep on summer ranges in the mountains of New Mexico, Arizona, Colorado and Utah for more than 30 years.[3] Wapiti *(Cervus canadensis)* are also susceptible, with blindness, damage to the central nervous system and facial necrosis resulting from parasites developing in the cephalic arterial system.[1] Asymptomatic infections have been noted in deer *(Odocoileus* spp.) from Arizona, New Mexico, Colorado, Utah, California and British Columbia.[2] The present report constitutes the first records of *E. schneideri* in the moose *(Alces alces)*.

HISTORY AND POST-MORTEM OBSERVATIONS

In August, 1971, an apparently blind yearling female moose was observed staggering and walking in circles along the Boulder River in the Absaroka Mountains approximately 64 km south of Big Timber, Sweetgrass County, Montana. It died shortly thereafter, and was brought to Bozeman by personnel of the Montana Fish and Game Department. At necropsy, eight immature *Elaeophora schneideri* were found in the right common carotid artery near its terminal junction. The brain, eyes, optic nerves and carotid arteries were fixed in 10% buffered formalin and sectioned for histopathologic study.

Numerous fifth stage nematodes were present, bilaterally, in arteries of the optic nerve sheath (Fig. 1) and sclera. A large vessel posterior to one eye was thrombosed. Swelling and vacuolation of muscle fibers was noted in some of the extrinsic muscles of the eye. There was no evidence of damage to the optic nerve or retina. Several nematodes were present in the leptomingeal arteries, and in many arteries circumferential fibrous intimal proliferation had occurred (Fig. 2). One large cerebral vessel was thrombosed. Changes in the brain parenchyma were confined to the cerebrum and consisted of large areas of cerebrocortical neuronal necrosis. A malacic area was present in the subcortical white substance, and numerous hemorrhages were observed.

Intimal proliferation had not occurred in the common carotid artery nor in its terminal branches nor in the arteries of the eye. Failure to find lesions in the optic nerves and retinas and the widespread neuronal necrosis in the cerebral cortex indicated that blindness in this animal probably originated in the central nervous system.

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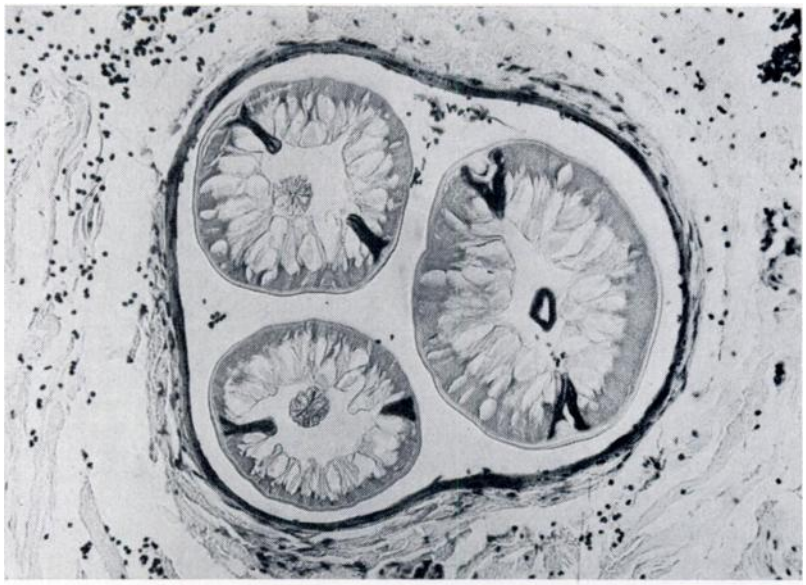

FIGURE 1. **Elaeophora schneideri** in a vessel of the optic nerve sheath. Mild hemorrhage surrounds the vessel, which is noticeably dilated. Azure eosinate. X 150.

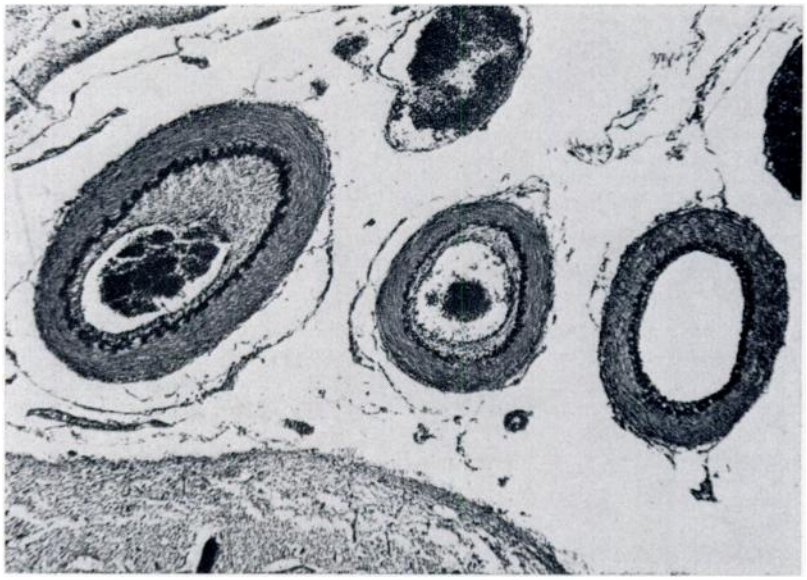

FIGURE 2. Intimal proliferation in leptomeningeal arteries. The vessel on the right is only slightly affected. Verhoeff-Van Gieson. X 60.

Three additional *Elaeophora* - infected moose have been collected in neighboring localities since the original case in Sweetgrass County. A mature cow taken in Park County in November, 1971, had 28 *E. schneideri* in its carotid arteries. No evidence was seen of clinical elaeophorosis. A cow moose shot by hunters in the Bridger Mountains in Gallatin County in November, 1971, harbored 11 adult worms, including gravid females containing ensheathed microfilariae. A 6 - 7 month-old female calf moose accompanying this cow was infected with one adult *E. schneideri*. No lesions were found in the common carotid artery of the cow, but some intimal proliferation had occurred in this vessel in the calf. Neither animal exhibited any obvious signs of impaired vision or abnormal behavior, although gross signs of neurologic disease might be difficult to detect under field conditions.

DISCUSSION

The tissue changes observed in the blind animal suggest that the histopathologic features of elaeophorosis in the moose resemble those described in wapiti.[1] No external facial lesions were noted in this moose, but the absence of opacity in the refractive media of the eyes was suggestive of the clear-eyed blindness characteristic of wapiti infections. Based on the degenerative changes in the brain and loss of vision in this animal, elaeophorosis in *A. alces* may resemble the disease in wapiti. As such, *E. schneideri* infections may have been involved in a number of recent cases of blindness observed in Montana moose.

The area in which *E. schneideri* presently is known to occur in moose extends for approximately 120 km along the upper Yellowstone River drainage in southwestern Montana. This region of mountain valleys and upland coniferous forests is somewhat similar ecologically to wildlife range in the southern Rocky Mountains where elaeophorosis is recognized as a serious disease in wapiti. An extensive mule deer population and isolated herds of wapiti exist throughout this portion of Montana, but the role of these species locally in the epidemiology of *E. schneideri* infection has not been investigated. All infected moose to date have originated from forested areas at elevations above 1680 m.

Acknowledgements

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