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# NEUROLOGIC DISEASE IN WAPITI NATURALLY INFECTED WITH MENINGEAL WORMS

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*Abstract:* Four wapiti *(Cervus canadensis)* from Oklahoma which had shown clinical signs of neurologic disease were submitted for pathological examination. Both mature female and larval meningeal worms *(Parelaphostrongylus tenuis* Pryadko and Boev, 1971) were either observed in or recovered from the meninges and parenchyma of the brains. Lesions found in the brain of each wapiti included focal meningitis, perivascular cuffing, and focal gliosis; frequently these were associated with the meningeal worms. There appeared to be a direct correlation between the presence of meningeal worms and lesions observed histologically which resulted in neurologic disturbances in the wapiti.

## INTRODUCTION

The meningeal worm *(Parelaphostrongylus tenuis* Pryadko and Boev, 1971) (=*Pneumostrongylus tenuis* Dougherty, 1945) is a parasite of white-tailed deer *(Odocoileus virginianus)* in eastern North America.[4,9,10,18] Although this parasite seldom produces clinical signs in white-tailed deer, natural infection of meningeal worms in moose[3,14,15,19,20] *(Alces alces)*, in reindeer[5] *(Rangifer tarandus tarandus)*, and in domestic sheep,[13,16,21,22] and experimental infection in wapiti[7] *(Cervus canadensis)*, caribou[6] *(Rangifer tarandus)*, and mule deer[2] *(O. hemionus)* typically results in neurologic disease. First-stage larvae of *P. tenuis* have been reported from the feces of a wapiti in Minnesota,[11] but adult meningeal worms have not been recovered from naturally infected wapiti.

Although neurologic disease has not been reported in wapiti naturally infected with meningeal worms, Anderson et al.[7] have revealed some aspects of the pathogenesis of this parasite by experimentally infecting wapiti with this helminth. The major lesions consisted of microcavitations, particularly in the dorsal horns of gray matter of the spinal cord, and traumatic damage to surrounding neural tissue. The worms apparently migrated to the brain from the spinal cord by way of the dorsal horns of gray matter and the dorsal nerve roots, although major lesions were rarely observed in the brain.

This paper presents histopathologic evidence of meningeal worm invasion in four eastern Oklahoma wapiti exhibiting signs of neurologic disturbances and reports the results of an examination for meningeal worms in 16 hunter-killed wapiti from the Wichita Mountains Wildlife Refuge in southwestern Oklahoma.

## CASE HISTORIES

A wapiti-transplanting program was initiated by the Oklahoma Department of Wildlife Conservation in 1969. Between January 1969 and March 1971, 375 wapiti were trapped on the Wichita Mountains Wildlife Refuge and relocated in various areas in Oklahoma.

In June 1970, a wapiti released near Heavener, in southeastern Oklahoma, was observed "circling". Although this wapiti later died, it was not necropsied. In the ensuing months, four wapiti exhibiting ataxia and/or "circling" were submitted to the College of Veterinary

Medicine, Oklahoma State University, for examination: on 15 June 1970, a female yearling captured at Heavener was submitted with a history of "circling"; another female with clinical signs of ataxia and "circling" was submitted from Heavener on 6 July 1970; on 17 July 1970, a yearling male showing signs of adipsia and ataxia was submitted from Spavinaw Hills State Game Refuge, Eucha, in eastern Oklahoma; and on 24 February 1971, a yearling male with a history of "circling" and vision impairment was submitted from Spavinaw Hills State Game Refuge.

## MATERIALS AND METHODS

Necropsies were performed on the four wapiti. Tissues were obtained from the cerebral cortex, midbrain, thalamus, cerebellum, medulla oblongata, and spinal cord, and preserved in 10% buffered formalin. For histologic examination, all tissues were embedded in paraffin, sectioned at 7$\mu$, and stained with hematoxylin and eosin.

Heads of 16 other wapiti were collected on 7 December 1971 from the Wichita Mountains Wildlife Refuge, Lawton, Oklahoma, from cooperating hunters by personnel of the Oklahoma Department of Wildlife Conservation. The heads were refrigerated immediately and maintained in this state until examined. For examination, each head was skinned and tissues overlying the skull were removed. The skull cap was removed by making incisions with a Stryker Autopsy Saw (Lipshaw Mfg. Co., Detroit, Michigan) across the frontal area posterior to the orbits, and sawing through the lateral aspect of the frontals and parietals to the foramen magnum. The brain was removed and examined for parasites under a dissecting microscope. Tissue sections were obtained from each brain, preserved in 10% buffered formalin, and prepared for histologic examination.

## RESULTS

### Necropsy Examination

One female nematode was recovered from the pons and one from the cerebellum of one of the wapiti that was submitted for pathological examination. One to three female nematodes were recovered from the brain parenchyma of each of the other three wapiti examined. A slight greenish coloration was observed in the nematodes. No other gross lesions which could be associated with a central nervous system (CNS) disturbance were observed in these wapiti.

Gross examination of the brain of the 16 hunter-killed wapiti revealed no lesions or parasites.

### Histopathologic Observations

Histologic lesions apparently related to damage by nematodes were observed in each of the four wapiti submitted for necropsy. Lesions in the meninges were characterized by focal, disseminated areas of lymphocyte infiltration interspersed with macrophages, eosinophils, and sometimes multinucleated giant cells. Immature larvae were found within the meninges and were within or closely associated with areas of meningitis (Fig. 1). Hemosiderin deposits were dispersed freely within the inflammatory reaction or within macrophages. Occasionally, fibroplasia was also present. Adult nematodes were seen within the meninges, but did not appear to elicit an inflammatory response (Fig. 2). Meningitis was also found in one of two spinal cords examined.

Lesions observed in the cerebrum, cerebellum, and spinal cord included mild perivascular cuffing and mild focal gliosis. No predilection site was noted. Linear hemosiderin accumulations near reactive glial cells marked probable nematode migratory tracts (Fig. 3). Nematode larvae, observed in the parenchyma of three of the four brains, usually elicited very little inflammation (Fig. 4). Although groups of several larvae usually were not associated with inflammation, perivascular cuffs of lymphocytes were occasionally situated near these groups. Adult nematodes were not found within the brain. Protozoan cysts, presumably *Toxoplasma gondi*, were also present in one of the brains examined, but there was no inflammatory response associated with them.

FIGURE 1. Meningeal worm larvae (arrows) in sulcus of cerebral cortex, associated with a mild, focal meningitis. Some larvae are degenerating. H & E, x 64.

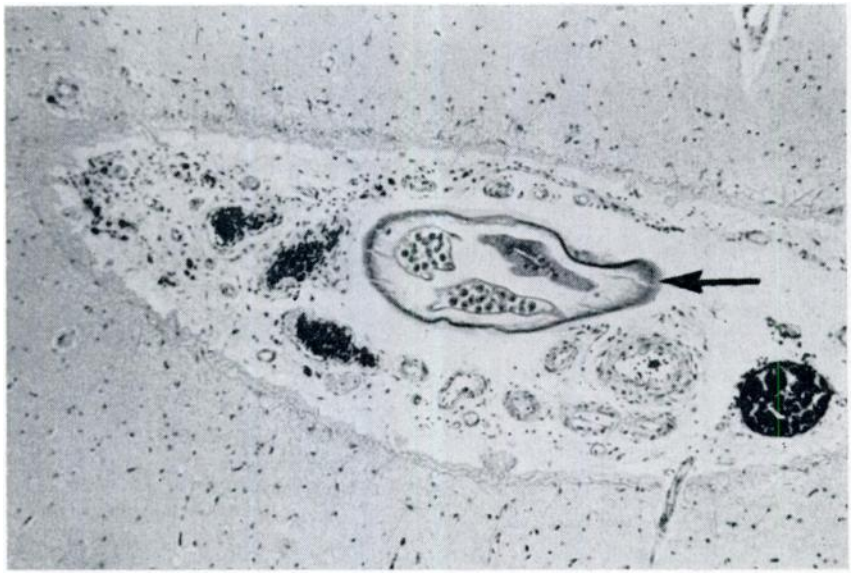

FIGURE 2. Adult female meningeal worm (arrow) in meninges of cerebral cortex. No significant inflammatory reaction is present. H & E, x 25.2.

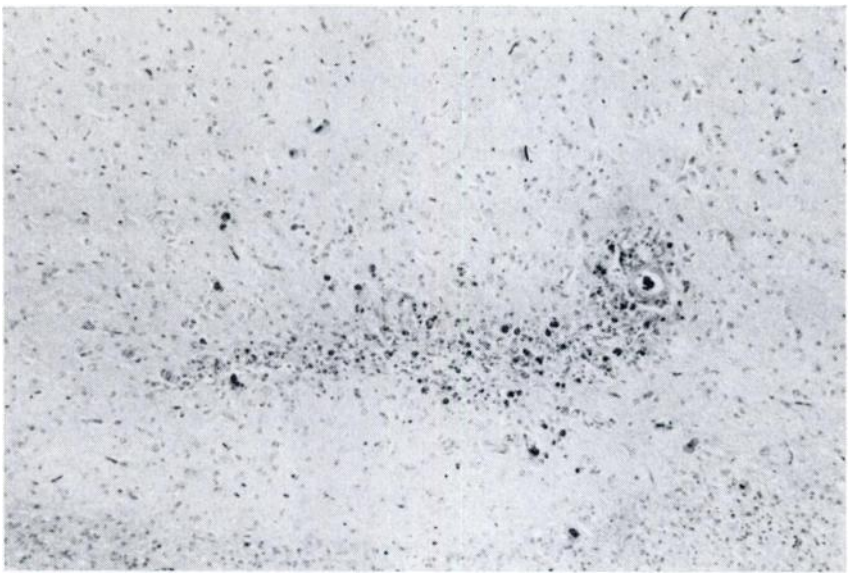

FIGURE 3. Focal gliosis and accumulation of hemosiderin in the cerebrum near the lateral ventricle, marking a probable migratory path of meningeal worm larvae. H & E, x 25.2.

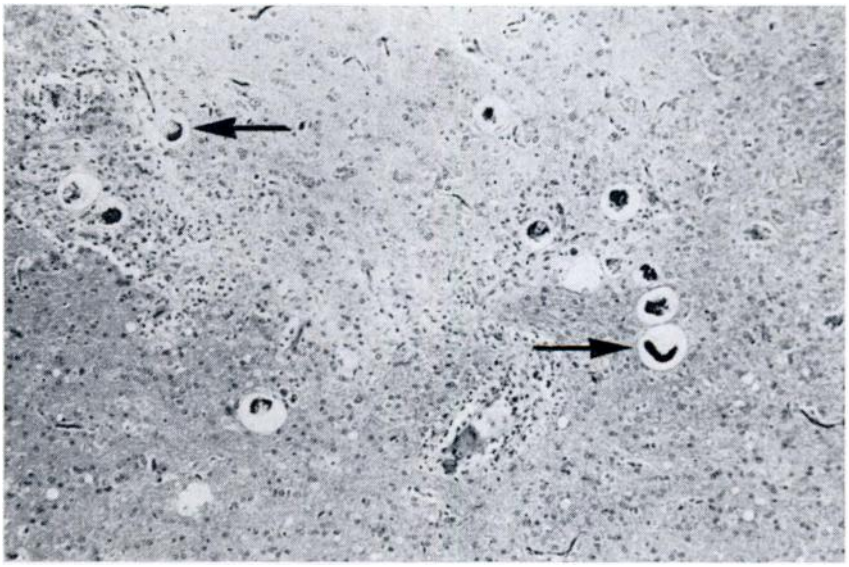

FIGURE 4. Meningeal worm larvae (arrows) in microcavitations in the cerebral cortex. A moderate glial reaction is also present. H & E, x 25.2.

Nematode parasites were not observed in any of the brains of the 16 hunter-killed wapiti. Very mild, focal meningitis, small perivascular cuffs, and focal accumulations of glial cells were seen in several brains, although no eosinophils nor other evidence of nematodes were found in these brains.

## DISCUSSION

Natural infection of meningeal worms usually does not elicit serious or prolonged neurologic disturbance in the white-tailed deer,[1,2] although neurologic disorders have been reported.[10,17] Study of experimentally infected cervids indicates that this parasite develops in moose, wapiti, and mule deer similarly to the way it develops in the white-tailed deer, although in the white-tailed deer it causes little damage to the CNS.[7]

Clinical signs of "circling", ataxia, impairment of vision, and adipsia observed in the wapiti obtained from eastern Oklahoma were associated with histologic lesions in the CNS. Meningitis was found in one of two spinal cords examined, although parasites were not observed. Other evidence of nematode-induced trauma to the spinal cord as described by Anderson et al.[7] in experimentally infected wapiti was not observed.

Examination of 16 wapiti obtained from the Wichita Mountains Wildlife Refuge revealed no evidence of meningeal worms. This may indicate that this parasite is not present in southwestern Oklahoma. However, these wapiti are live-trapped and released into areas of eastern Oklahoma where meningeal worms have been reported.[9]

The successful development of meningeal worms in wapiti raises the possibility that this helminth could become established in herds after an initial exposure to the infection in areas concomitant with infected white-tailed deer.[7] This should be an important consideration in Oklahoma when wapiti are introduced into areas in eastern Oklahoma where white-tailed deer are infected with meningeal worms. Management implications for wapiti in Oklahoma are similar to those proposed for moose in Minnesota by Karns:[12] an increase in the number of infected deer on range managed for wapiti would likely increase the number of infected intermediate hosts and consequently increase the probability of infecting wapiti; it seems advisable, therefore, that on areas where meningeal worms are indigenous, and where the areas will be managed primarily for wapiti, deer should be kept at a low population level.

A similar situation has also occurred in Canada when reindeer translocated from Norway to an area endemic with meningeal worms in Ontario developed neurologic disease.[6] This disease was due to a small number of meningeal worms in the CNS and was considered the direct cause of the failure of this attempted introduction. Anderson concluded that it would also be impossible to reintroduce woodland caribou, a close relative of the reindeer, into areas in which they were previously indigenous and which were now occupied by white-tailed deer with a high prevalence of meningeal worms.[4] Parelaphostrongylosis, therefore, may have been the reason why introductions of some cervids into certain parts of continental North America have been failures.[6]

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