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Meningeal Worm Invasion of the Brain of a Naturally Infected White-tailed Deer

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Introduction

The meningeal worm *(Pneumostrongylus tenuis)* is a common parasite of white-tailed deer in eastern North America.[1,2] The parasite develops in the neural parenchyma of the spinal cord, migrates to the subdural space and matures. The adult *P. tenuis* is most commonly found in the subdural space and venous sinuses of the cranium. Related signs of a neurologic disease in the wild deer are rare,[1,3] and heavy experimental infections produce only occasional transient lameness and limb weakness.[1] Meningeal worm infection of other cervids (moose, wapiti, caribou and mule deer) however, usually results in a fatal neurologic disease.[1]

This study provides evidence of meningeal worm migration through the cerebral parenchyma of a naturally infected white-tailed deer and considers pathogenetic mechanisms for the lesions.

Case History

The male white-tailed deer described in this report was born in Green Bay, Wisconsin about June 1, 1968 and was one of ten deer maintained for commercial purposes. *P. tenuis* larvae were found in feces collected from the pens used to confine these deer. It was not known which of these deer were infected. This animal was transported to Madison, Wisconsin in September 1968 to be used in a study of experimental Haemonchosis in white-tailed deer. Techniques and results of hematology and fecal examinations have been reported.[4] The deer under discussion was infected with 25,000 *Haemonchus contortus* larvae on November 6. The pre- and post-infection hemoglobin, packed cell volume and total serum protein were significantly lower than those reported for the other deer in the group. Starting on October 11, feces were collected from the rectum every two days. This deer began passing *P. tenuis* larvae on November 18, 1968 (146/gram feces). Two days later only 23/gram feces were found and on the day of death, November 22, 1968, none were found. Haemonchus eggs were not found in the feces of this animal since it died 16 days post inoculation.

During the last five days of life, the deer was observed to have had difficulty standing. When standing or attempting to get up, he would stumble and fall. There was a progressive loss of coordinated locomotor function. This deer was noticeably small at the beginning of the experiment and had additionally suffered a considerable weight loss, although he had been eating until about five days prior to its death. The brain and visceral tissues were collected shortly after death, fixed in 10% formalin, embedded in paraffin and stained with hematoxylin-eosin and Luxol-fast-blue/cresyl violet.

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Results

Necropsy Examination

Ten live adult *P. tenuis* were collected from the intracranial subdural space. One of these parasites was found over the cerebral cortex protruding from the leptomeninges. There were multiple extensive recent subarachnoid hemorrhages over the cerebrum (Figs. 1 & 2).

On coronal sections, the most striking finding was numerous recent curvilinear hemorrhages of an average width of 1 mm (Figs. 2 & 3). These were located preferentially within the immediate subcortical white matter, where they ran parallel to the cortical ribbon over distances up to 14 mm. Some of these hemorrhages extended through the cortex (Fig. 2) towards, or into the leptomeninges. Moderate recent subarachnoid hemorrhage filled the sulci (Fig. 2, arrows) and also the meningeal space over the gyri (Fig. 1). During sectioning, several threadlike parasites became evident. No distension or thrombosis of superficial veins, and no petechial hemorrhages or edematous softening of the parenchyma were noted. There was no yellowish discoloration of tissues suggestive of hemosiderin deposits. The deep central white matter surrounding the lateral ventricles, the medulla oblongata, the spinal cord, and the cerebral dura had not been collected. The basal ganglia, the anterior brainstem and the cerebellum revealed no distinct lesions.

Examination of the gastrointestinal tract for parasites revealed 15 Capillaria species in the small intestine, and 1 adult female Haemonchus and 18,129 4th stage Haemonchus larvae in the abomasum.

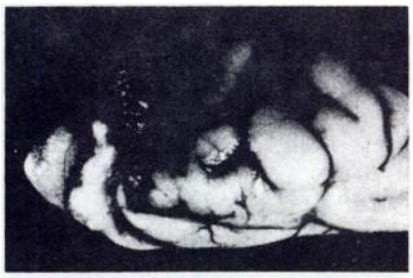

FIGURE 1. *Recent subarachnoid hemorrhage over the cerebral convexity.*

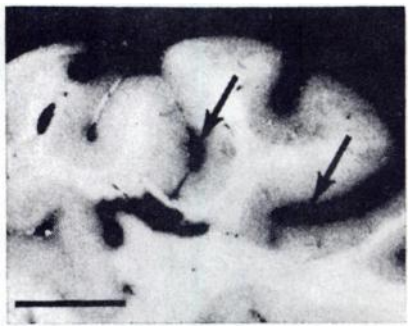

FIGURE 2. *Curvilinear hemorrhages in subcortical white matter and focal hemorrhage in the cortex. Arrows indicate widening of two sulci by subarachnoid hemorrhage. Bar equals 10 mm.*

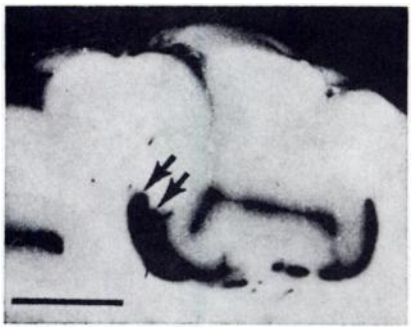

FIGURE 3. *Curvilinear hemorrhages in subcortical white matter following cortical ribbon. Arrows indicate two parallel hemorrhages. Bar equals 10 mm.*

Histopathologic Observations

Sectioned young adult parasites were found in the cerebrum only. They were located in the periventricular white matter, in the subcortical white matter (Fig. 6), in the cortex (Fig. 4), and at the bottom of a sulcus. None were located within the lumen of blood vessels. Several worms, however, were found adjacent to small arteries of the cerebral

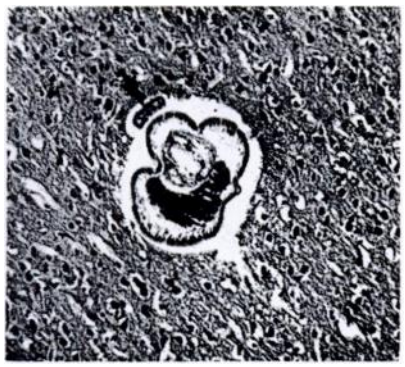

FIGURE 4. P. tenuis *in the cerebral cortex adjacent to a small artery (arrow). X 95.*

cortex (Fig. 4) or subcortex. Groups of eggs, presumably *P. tenuis,* were found in cerebral sulci (Fig. 5), but there were none in the neural parenchyma. Occasional larvae were found in the subpial portion of the molecular layer of the cerebral cortex.

Cerebral hemorrhages, in many instances, could be associated with the presence of parasites in the parenchyma. However, not all parasites were surrounded by hemorrhage. When sectioned longitudinally, curvilinear hemorrhages seemed to follow the course of the subcortical association fibers (Meynert's U-fibers). In some instances, an additional relationship to small arteries was noted. In the cortex, hemorrhages had produced marked focal disruption of all nerve cell layers.

No infiltrates with white blood cells or with glial macrophages were found as a reaction to the adult parasites. One small collection of lymphocytes and plasma cells was located within the pia close to a larvae in the cortex, and a second infiltrate was located randomly.

Where worms were surrounded by parenchyma rather than hemorrhage, only minimal tissue damage was noted, such as cell compression and dislodgement of small pieces. No thrombosis, no edema, no siderotic pigmentation and no astroglial reaction were seen.

In the cerebellum, scattered small recent hemorrhages were associated with minor focal edema of the white matter.

Numerous larvae and eggs were found in the alveolar septa of the lungs, where they were surrounded by fibroblasts. There were areas of congestion and edema with a moderate cellular infiltrate composed primarily of eosinophils. Randomly sampled lymph nodes contained large numbers of larvae, most of which were found in the marginal sinuses.

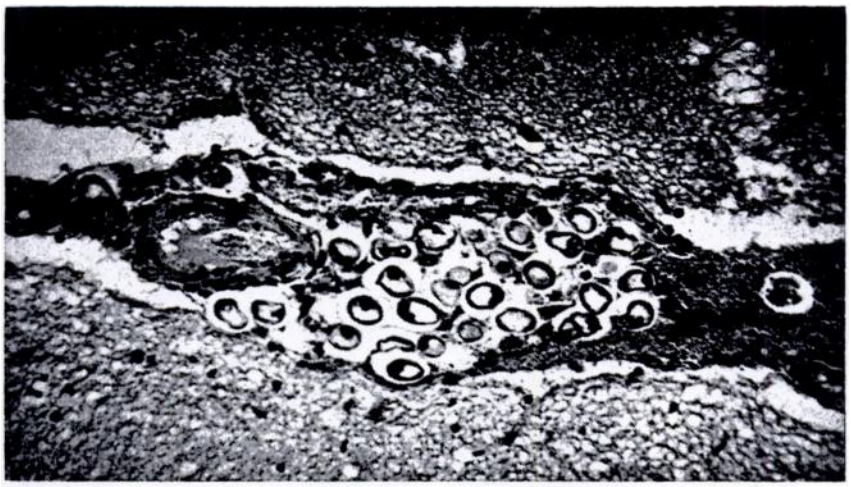

FIGURE 5. *Parasite eggs in the leptomeninges of a cerebral sulcus. X 200.*

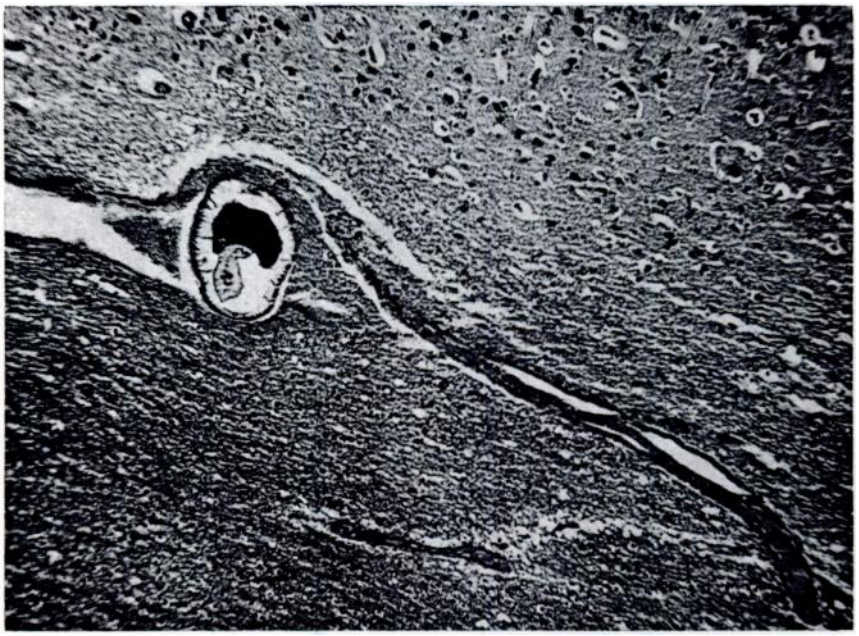

FIGURE 6. *Young adult* P. tenuis *in the subcortical white matter next to a small artery surrounded by hemorrhage. X 95.*

Occasional foreign body giant cells and eosinophils were observed in and around these larvae. In the liver, focal coagulative necrosis of the parenchyma, associated with hemorrhage and with eosinophilic infiltrates, was found. The lesions were not extensive. Neither parasites, larvae or eggs were seen.

Discussion

The type and the extent of the cerebral lesions of this deer suggest some relevance for the clinical illness as well as for the death of the animal. However, the incompleteness of the submitted material precludes final judgment on the motor pathways as a whole, and on vital centers. The runted condition of the animal, the abnormal blood chemistry, the anemia, the second parasitic infection, and the final period of starvation presumably all contributed to the demise.

The reason for the presence of *P. tenuis* in the cerebrum, presumably by ascension from the spinal cord remains obscure. This is a rare event in the white-tailed deer, while involvement of the medulla oblongata is not uncommon.[1] In contrast to this, meningeal worm infection of other cervids results in more frequent invasion of the entire brain, in addition to extensive damage to the anterior horns of the spinal cord.[1]

Anderson[1] made a comparison between the lesions produced in white-tailed deer and in other cervids. One of the factors which he suggests that may influence the extent of tissue injury is the host susceptibility to neural invasion. He concluded that the absence of significant neural invasion in the white-tailed deer was related to a high mortality of infective larvae outside the central nervous system. The duration of presence of the parasite within the central nervous system is another influential factor suggested by Anderson. The development of *P. tenuis* in the spinal cord of white-tailed deer

continues up to about 30-40 days post-infection.[5] After 40 days, parasites were found in the subdural spaces, but not in the parenchyma. In this deer, a prolonged neural invasion is suggested from the presence of migrating parasites in the parenchyma at least 70 days post-infection. This minimal time estimate is based on the transfer time of the animal to experimental facilities after which infection could not have occurred.

In light of the factors suggested above, the invasion of the cerebrum of this white-tailed deer may be related to an altered host-parasite relation due to the poor physical condition of the deer. This altered relation may have permitted numerous larvae to invade the neuroparenchyma and to remain there for an unusually long period of time.

The intracerebral hemorrhages in this deer are of a very unusual morphology and, unlike hemorrhages in venous or in venous sinus thrombosis, or in ischemic infarctions, they are not associated with edema and tissue necrosis. Due to their almost constant width, they are reminiscent of hemorrhages following surgical needle punctures. However, in their course they seem to correspond to the distribution pattern of small arteries (Fig. 7 & 8). By light microscopy, worms were seen next to vessels, which were sometimes collapsed, and were seen surrounded by hemorrhage. It would appear, that the parasites, when approaching the cortex, made use of the arteries as a guidance system towards the brain surface. During their progression, they tore off arterial branches and thus instigated cylindrical hemorrhages. These might subsequently extend into the leptomeninges via the perivascular Virchow-Robin spaces. However, the motion of the parasites within the pia, especially of the narrow sulci, might also produce local hemorrhages of a mechanism as outlined above.

Recently, Prestwood[3] found masses of adult *P. tenuis* in the cranium of a pregnant doe which had been in poor condition and had shown neurologic illness. At necropsy, worms were principally

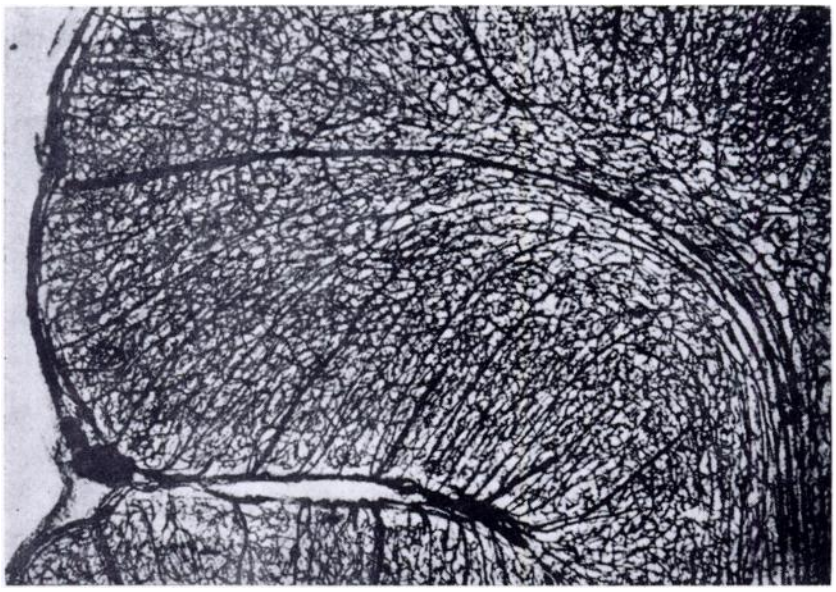

FIGURE 7. *Dye injection study of cerebral cortex of cat. Note typical course of long arterial branch through cortex and subjacent white matter. Vessel enters at tip of gyrus and sends many branches to the cortex.*[6] *(compare with Fig. 2).*

located within the subdural space and within venous sinuses. The major finding in the brain was an extensive discoloration of the leptomeninges and of the outer zone of the cerebral cortex by a strongly iron positive pigment. From this description and from the two light micrographs, this was a case of marginal or superficial siderosis of the central nervous system. This condition is a well established pathologic entity in the human,[7] known to occur after repeated hemorrhages into the subarachnoid space and to be associated with a progressive neurologic illness.[8] The lesion has been reproduced in dogs,[9] in cats[10] and in rabbits[11] by repeated injections of either whole blood, or suspensions of erythrocytes or iron compounds into subarachnoid cisternal spaces.

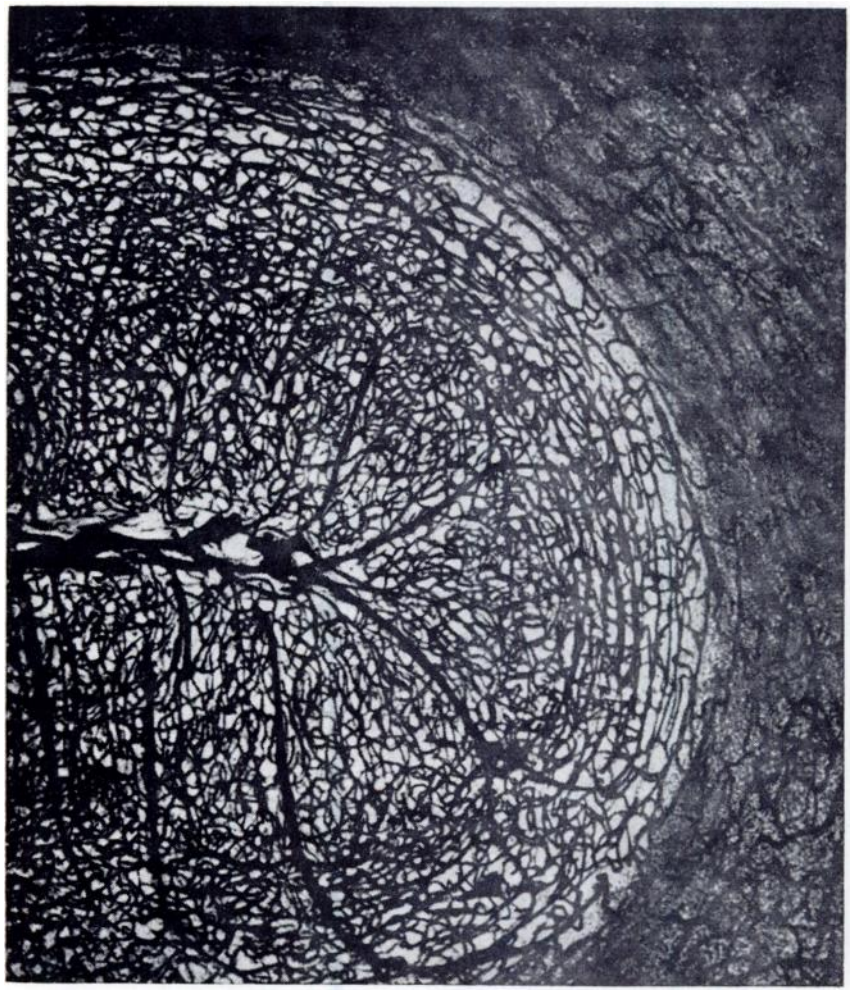

FIGURE 8. *Dye injection study of cerebral cortex along the depth of a sulcus. Typical semi-circular course of cerebral vessels which follow the U-fiber system of Meynert along the border between cortex and white matter.*[6] *(Compare with Figs. 2, 3)*

Prestwood[8] suggests that the pigmentation was the result of mechanical interference with the cerebral blood flow due to the presence of worms within the venous sinuses. However, no other features of stagnant anoxia, such as atrophy of the cortex or old malacias, were mentioned. When considering the hemorrhagic manifestations in this case, we wonder whether a superficially located, subacute to chronic cerebral lesion could be produced in a particularly susceptible deer after migration of excessive numbers of parasites through the neural parenchyma and the pia.

Acknowledgments

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