NATURALLY OCCURRING NEUROLOGIC DISEASE IN A FALLOW DEER INFECTED WITH MENINGEAL WORMS

Authors: KISTNER, T. P., JOHNSON, G. R., and RILLING, G. A.

Source: Journal of Wildlife Diseases, 13(1) : 55-58

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-13.1.55

NATURALLY OCCURRING NEUROLOGIC DISEASE IN A FALLOW DEER INFECTED WITH MENINGEAL WORMS

T. P. KISTNER,[1] G. R. JOHNSON[2] and G. A. RILLING[3]

Abstract: Neurologic disease resulting from infection with *Parelaphostrongylus tenuis* was diagnosed in a fallow deer (*Dama dama*) from Georgia, with clinical signs and histopathologic lesions similar to those reported for other accidental hosts of *P. tenuis*. Early fifth stage parasites were found in the spinal meninges and immature parasites were found in the neural parenchyma, but none were recovered from the brain and cranial meninges.

INTRODUCTION

Neurologic disease resulting from natural infection with the meningeal worm (*Parelaphostrongylus tenuis*) has been reported in caribou[12] (*Rangifer tarandus*), moose[3,4,12,16,21,22] (*Alces alces*), reindeer[5,6] (*Rangifer tarandus tarandus*), wapiti[13] (*Cervus canadensis*), white-tailed deer[14,18] (*Odocoileus virginianus*), and domestic sheep[15,17,23,24] (*Ovis aries*). Neurologic disease resulting from experimental infections with *P. tenuis* has been produced in caribou[9] (*Rangifer tarandus terraenovae*), moose,[2,4] mule deer[7] (*Odocoileus hemionus hemionus*), wapiti,[7] domestic sheep[8] and domestic goats[10,11] (*Capra hircus*).

Despite the number of exotic ungulates introduced into North America, the pathogenicity of *P. tenuis* has not been determined for these animals with the exception of reindeer,[5,6] domestic goats[10,11] and domestic sheep.[8,15,17,23,24] With the increasing numbers of drive-through wild animal parks or preserves featuring exotic ungulates, the pathogenicity of *P. tenuis* should be determined for these animals. This paper presents the pathologic features of *P. tenuis* infection in a fallow deer, and is intended to serve as an alert that the meningeal worm may result in significant loss if susceptible animals are transplanted to naturally infected areas.

CASE REPORT

The subject of this report is a mature male fallow deer (*Dama dama*), one of seven maintained in captivity for three years at a game ranch in De Kalb County, Georgia. They were held in a 4 ha enclosure together with American Bison (*Bison bison*), cattle (*Bos taurus*), domestic goats and white-tailed deer. The presence of *P. tenuis* in the white-tailed deer within this enclosure was established in 1967.[13]

During the fall of 1971, two mature fallow deer became ill and subsequently died. Signs consisted of suddenly developing weakness in the hind quarters, which rapidly progressed to paraplegia. An ascending paralysis culminated in total locomotor paralysis within a few days. In both cases, initial signs appeared the day following strenuous exercise associated with attempts to capture the deer. The first deer died after having been paralyzed for several days and was incinerated without being examined postmortem. The deer described in this report was presented while alive.

[1] School of Veterinary Medicine, Oregon State University, Corvallis, Oregon 97331, USA.
[2] The Procter & Gamble Company, Professional & Regulatory Service Division, P.O. Box 39175, Cincinnati, Ohio 45239, USA.
[3] Stone Mountain Game Ranch, Stone Mountain, Georgia 30083, USA.
Oregon Agricultural Experiment Station Technical Paper No. 4175.

The animal had a flaccid paralysis of all four legs and decubital ulcers on the left shoulder and left hip. Ausculation revealed extensive bilateral pneumonia. The buck was treated with antibiotics and placed in a bedded stall where it was found dead the next morning.

At necropsy, significant gross visceral lesions consisted of bilateral pneumonia and sequelae associated with secondary septicemia. Gross neurologic lesions consisted of engorgement of the cerebral blood vessels and subdural hemorrhage at a level of the fifth and sixth cervical vertebrae. The brain and spinal cord were removed and examined using the methods of Prestwood and Smith[19] and Anderson,[1] with some modification in the latter investigator's procedure. Gross examination of the brain failed to reveal parasites. One male and two female fifth stage *P. tenuis*[1] were recovered from the subdural space of the unpreserved spinal cord; subsequent examination confirmed identification of the male worm. Representative portions of the brain and spinal cord were collected and preserved in 10% formalin for histopathologic examination.

Histopathologic examination of hematoxylin and eosin stained sections of formalin-fixed spinal cord and associated meninges revealed lesions in all segments of the cord from the cervical through the lumbar. The meningeal lesions were characterized by marked thickening and chronic inflammation of the dura, subdural hemorrhage, marked lymphocytic infiltration, and mineralization (Fig. 1). It could not be determined whether the calcified areas represented mineralized parasites, parasite ova, or resulted solely from the inflammatory response to the parasites.

The spinal cord contained numerous lesions thought to be caused by direct or secondary effects of migrating parasites. The lesions included hemorrhage, microcavitations, neuronal necrosis and perivascular cuffing composed principally of lymphocytes and eosinophils. Parasites were present in some of the lesions. Sectioned parasites *in situ* were identified as *P. tenuis*[1,7,17] (Fig. 2).

The inflammatory response observed in sections of the cord and meninges also was present in the perineural sheaths of the dorsal and ventral nerve rootlets. No

FIGURE 1. Photomicrograph depicting mineralization and mononuclear cell infiltration of lumbar meninges (x25.2).

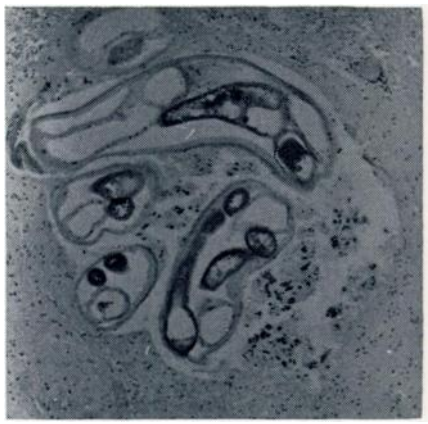

FIGURE 2. Cross section of **P. tenuis** larvae in gray matter of lumbar spinal cord (x25.2).

lesions or parasites were seen in sections of the brain and associated meninges. Mature parasites, eggs or larvae were not isolated from the brain, nor were eggs or larvae found in lung sections.

DISCUSSION

This is believed to be the first reported case of neurologic disease attributable to *P. tenuis* infection in fallow deer. The clinical signs and lesions were similar to those reported for other accidental cervid hosts of *P. tenuis*,[1,2,5,7,9,11,15] but the conditions associated with the occurrence of disease were unusual.

The histopathologic lesions of the spinal dura consisted predominantly of thickening and calcification, but it was impossible to determine if the lesions resulted from relatively recent, chronic or repeated infections.[8,20] The presence of fifth stage worms suggested that the infection was of sufficient duration to have permitted development of the lesions in the dura (R. C. Anderson, 1976, pers. comm.).

Since gravid adult worms were not found in the cranium, and eggs or larvae were not found in the cranium or lungs, it was not determined if *P. tenuis* infection in fallow deer is similar to the infection in sheep, in that neither animal is a suitable definitive host for the parasite, or whether patency would have occurred had the deer survived.

This case is considered unusual because the deer showed no visible abnormalities or behavior for three years. Accidental hosts of *P. tenuis* frequently succumb to neurologic disease shortly after introduction into an infected area.[5,12,13] The overgrazed condition of the pen precluded optimum snail habitat, hence, a low prevalence of infected molluscs may offer a plausible explanation for the long survival of fallow deer and goats under these circumstances (R. C. Anderson, 1976, pers. comm.). Although facets of this case were not resolved, the deer was nevertheless paralysed as a result of *P. tenuis* infection. It therefore seems inadvisable to place fallow deer in areas infected with *P. tenuis*. Similarly, it would appear inadvisable to hold other exotic ungulates in similar areas until the susceptibility of each species is determined. If establishment of a wild animal reserve is planned in an area infected with *P. tenuis*, terrestrial snail eradication and exclusion of native white-tailed deer may offer solutions to this potential problem.

Acknowledgements

Appreciation is expressed to Drs. R. C. Anderson, J. F. Bone, E. O. Dickinson and A. K. Prestwood for critical review and helpful suggestions regarding the manuscript.

LITERATURE CITED

1. ANDERSON, R. C. 1963. The incidence, development, and experimental transmission of *Pneumostrongylus tenuis* Dougherty (Metastrongyloidea: Protostrongylidae) of the meninges of the white-tailed deer (*Odocoileus virginianus borealis*) in Ontario. Can. J. Zool. 41: 775-792.
2. ————. 1964. Neurologic disease in moose infected experimentally with *Pneumostrongylus tenuis* from white-tailed deer. Path. Vet. 1: 289-322.
3. ————. 1965. An examination of wild moose exhibiting neurologic signs in Ontario. Can. J. Zool. 43: 635-639.
4. ————. 1965. Cerebrospinal nematodiasis (*Pneumostrongylus tenuis*) in North American cervids. Trans. N. Amer. Wildl. Nat. Res. Conf. 30: 156-167.
5. ————. 1971. Neurologic disease in reindeer (*Rangifer tarandus tarandus*) introduced into Ontario. Can. J. Zool. 49: 159-166.
6. ————. 1972. The ecological relationships of meningeal worm and native cervids in North America. J. Wildl. Dis. 8: 304-310.

7. ————, M. W. LANKESTER and U. R. STRELIVE. 1966. Further experimental studies of *Pneumostrongylus tenuis* in cervids. Can. J. Zool. 44: 851-861.

8. ———— and U. R. STRELIVE. 1966. Experimental cerebrospinal nematodiasis (*Pneumostrongylus tenuis*) in sheep. Can. J. Zool. 44: 889-894.

9. ————. 1968. The experimental transmission of *Pneumostrongylus tenuis* to caribou (*Rangifer tarandus terraenovae*). Can. J. Zool. 46: 503-510.

10. ————. 1969. The effect of *Pneumostrongylus tenuis* (Nematoda: Metastrongyloidea) on kids. Can. J. Comp. Med. 33: 280-286.

11. ————. 1972. Experimental cerebrospinal nematodiasis in kids. J. Parasit. 58: 816.

12. BEHREND, D. F. and J. F. WITTER. 1968. *Pneumostrongylus tenuis* in white-tailed deer in Maine. J. Wildl. Manage. 32: 963-966.

13. CARPENTER, J. W., HELEN E. JORDAN and B. C. WARD. 1973. Neurologic disease in wapiti naturally infected with meningeal worms. J. Wildl. Dis. 9: 148-153.

14. ECHROADE, R. J., G. M. ZURHEIN and W. FOREYT. 1970. Meningeal worm invasion of the brain of a naturally infected white-tailed deer. J. Wildl. Dis. 6: 430-436.

15. KENNEDY, P. C., J. H. WHITLOCK and S. J. ROBERTS. 1952. Neurofilariosis, a paralytic disease of sheep. I. Introduction, symptomatology, and pathology. Cornell Vet. 42: 118-124.

16. LOKEN, K. I., J. C. SCHLOTTHAUER, H. J. KURTZ and R. D. KARNS. 1965. *Pneumostrongylus tenuis* in Minnesota moose (*Alces alces*). Bull. Wildl. Dis. Ass. 1: 7.

17. NIELSEN, S. W. and J. AFTOSMIS. 1964. Spinal nematodiasis in two sheep. J. Am. vet. med. Ass. 144: 155-158.

18. PRESTWOOD, ANNIE K. 1970. Neurologic disease in a white-tailed deer massively infected with meningeal worm. J. Wildl. Dis. 6: 84-86.

19. ———— and J. F. SMITH. 1969. Distribution of meningeal worm (*Pneumostrongylus tenuis*) in deer in the southeastern United States. J. Parasit. 55: 720-725.

20. SMITH, H. A., T. C. JONES and R. C. HUNT. 1972. *Veterinary Pathology*. Lea and Febiger. 1521 pp.

21. SMITH, H. J. and R. M. ARCHIBALD. 1967. Moose sickness, a neurological disease of moose infected with the common cervine parasite, *Elaphostrongylus tenuis*. Can. Vet. J. 8: 173-177.

22. ————, ———— and A. H. CORNER. 1964. Elaphostrongylosis in maritime moose and deer. Can. Vet. J. 5: 287-296.

23. WHITLOCK, J. H. 1952. Neurofilariosis, a paralytic disease of sheep. II. *Neurofilaria cornellensis* n.g.n. sp. (Nematoda, Filarioidea), a new nematode parasite from the spinal cord of sheep. Cornell Vet. 42: 125-132.

24. ————. 1959. *Elaphostrongylus,* the proper designation of *Neurofilaria*. Cornell Vet. 49: 328-339.

Received for publication 17 February 1976