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SHORT COMMUNICATIONS

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Cerebrospinal Nematodiasis in a Moose in Norway

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ABSTRACT: A case of cerebrospinal nematodiasis in a young adult moose (*Alces alces*) from Telemark county, southeastern Norway, is described. The moose was found by bird hunters during January, displaying signs of severe posterior paresis. It was killed and submitted for autopsy. The carcass was emaciated, and there were skin excoriations and subcutaneous edema over both metacarpi. Histopathologic examination revealed traumatic malacia throughout the spinal cord and meningeal accumulations of mononuclear inflammatory cells and eosinophils in brain and spinal cord. Two adult female nematodes were found in sections, respectively, of the subarachnoid and subdural spaces of the thoracic spinal cord. The nematode cross sections were similar with those of the two neurotropic *Elaphostrongylus* species, *E. rangiferi* and *E. cervi*. The moose originated from an area overlapping the grazing area of a large population of wild reindeer (*Rangifer tarandus tarandus*) living on the mountain plateau of Hardangervidda, suggesting the moose was infected with *E. rangiferi* from reindeer.

Key words: *Alces alces*, case report, cerebrospinal nematodiasis, *Elaphostrongylus rangiferi*, moose, paresis.

Nematodes of the genus *Elaphostrongylus* are parasites of Cervidae and may cause neurologic disorders. The genus includes *E. cervi* (= *E. panticola*) of red deer (*Cervus elaphus*) and other *Cervus* spp., *E. rangiferi* of reindeer (*Rangifer tarandus*) and *E. alces* of moose (*Alces alces*) (Steen et al., 1989; Gibbons et al., 1991). Norwegian red deer, reindeer, and moose are commonly infected (Halvorsen et al., 1980; Ottestad, 1983; Stuve, 1987). *Elaphostrongylus rangiferi* and *E. cervi* also infect and cause neurologic disease in abnormal hosts such as sheep and goats (Handeland, 1991; Handeland and Sparboe, 1991; Pusterla et al., 2001). Lankester and Fong (1998) described *E. rangiferi* from caribou (*R. tarandus caribou*) in the

skeletal muscles of healthy moose (*A. alces americanus*) in Newfoundland, Canada. This nematode has produced clinical disease in experimentally infected moose (Lankester, 1977). The present study presents a case report of clinical elaphostrongylosis in a moose that is believed to have resulted from natural cross-infection with *E. rangiferi* from reindeer.

On 24 January 1998, a young adult moose bull displaying signs of locomotor disturbances was found by bird hunters in the northern part of Fyresdal municipality (59°12'N, 08°06'E), Telemark county, southeastern Norway. The moose appeared bright and alert, but had great difficulty in rising and moving due to severe posterior paresis. The moose was killed by a shot to the caudal part of the head, and through the thoracic cavity. During evisceration, a greatly distended urinary bladder was observed. The eviscerated carcass, along with the heart, lungs, and kidneys, were transported to the laboratory for autopsy.

The skull was split in the sagittal plane, and the vertebral column was opened from the dorsal surface. The undamaged cranial part of the brain and the entire spinal cord were removed. Smears of bronchial mucus were examined microscopically for nematode larvae. The brain, spinal cord, and samples from the lungs, heart, and kidneys were fixed in 10% buffered formalin. After fixation, tissue blocks were embedded in paraffin, sectioned at 2–4 µm, and stained with hematoxylin and eosin (HE) for histologic examination. Based on the presence of eosinophilic meningitis and focal malacia, lesions suspicious of cerebrospinal nematodiasis, it

was decided to carry out extensive examination of the entire formalin-fixed central nervous system (CNS). The brain and spinal cord were sectioned every 5 mm and prepared for histologic examination. About 500 slides were studied.

The carcass was emaciated and there were skin excoriations and subcutaneous edema over both metacarpi. Neither significant gross lesions nor parasites were detected in cranial cavity, vertebral canal, meninges, or skeletal muscles. Lesions were not observed in lungs or kidneys, and nematode larvae were not found in bronchial mucus.

There were meningeal accumulations of plasma cells, lymphocytes, and macrophages, and variable numbers of eosinophils (Fig. 1) as well as lymphohistiocytic granulomas in the CNS. Foci of traumatic malacia occurred throughout white matter of cervical, thoracic, and cranial lumbar spinal cord (Fig. 2). Two adult female nematodes were sectioned in, respectively, the subarachnoid (Figs. 3–4) and subdural spaces of thoracic spinal cord. Morphologic characteristics of the nematodes in sections were as follows: thin cuticle; hypodermal cords divided into sublaterals, often associated with a large nucleus; coelomyarian musculature; and presence of a double genital tract. Nematode cross sections were typical of adult *E. rangiferi* or *E. cervi* as occurring in the CNS of infected animals (Handeland, 1991; Handeland et al., 2000a). No nematode eggs or inflammatory processes in the epidural tissue along the vertebral canal, typical for *E. alces* infection (Steen and Reh binder, 1986), were observed. *Elaphostrongylus* eggs were not seen in lung.

Elaphostrongylus rangiferi and *E. cervi* develop in the CNS (neurotropic nematodes), preferentially in the subarachnoid spaces, and thereafter migrate into the skeletal muscles (Hemmingsen et al., 1993; Handeland, 1994; Handeland et al., 200b). In contrast, *E. alces* is a non-neurotropic nematode that seems to develop in the epidural space of the caudal verte-

bral canal (Handeland and Gibbons, 2001). The findings in the present moose of nematodes and associated lesions in the CNS and not in the epidural space of the vertebral canal, are suggestive of cross-infection with one of the two neurotropic *Elaphostrongylus* species, most likely *E. rangiferi*. The moose originated from an area overlapping with the grazing area of the large population of wild reindeer living on the mountain plateau of Hardangervidda. Red deer are very rare in this region of the country, and have never been observed in the area where the moose was found. The poor condition of the carcass suggested the disease had been present for some time, possibly as far back as early winter. This is about the time that cerebrospinal elaphostrongylosis caused by *E. rangiferi* occur in small ruminants in Norway (Handeland, 1991; Handeland and Sparboe, 1991).

The two adult nematodes found in the CNS were located in the meninges of the spinal cord. However, there were foci of traumatic malacia indicating parenchymal migration of nematodes in all areas of the spinal cord. These lesions most probably were a central cause of clinical signs. Traumatic malacia caused by adult nematodes migrating in the brain and spinal cord parenchyma are typical findings in naturally occurring cerebrospinal elaphostrongylosis in small ruminants (Handeland, 1991; Handeland and Sparboe, 1991). Experimental studies of *E. rangiferi* and *E. cervi* in small ruminants have revealed that these nematodes are likely to represent hematogenously spread larvae that have developed in the CNS parenchyma or inner cerebrospinal fluid system, causing parenchymal laceration when they start search for a sexual partner and embark upon their migration towards the meninges and skeletal muscles (Handeland and Skorp ing, 1992; Handeland et al., 2000a). The present findings may indicate the same pathogenesis in clinical infection with these nematodes in moose. This is further supported by Lankester (1977),

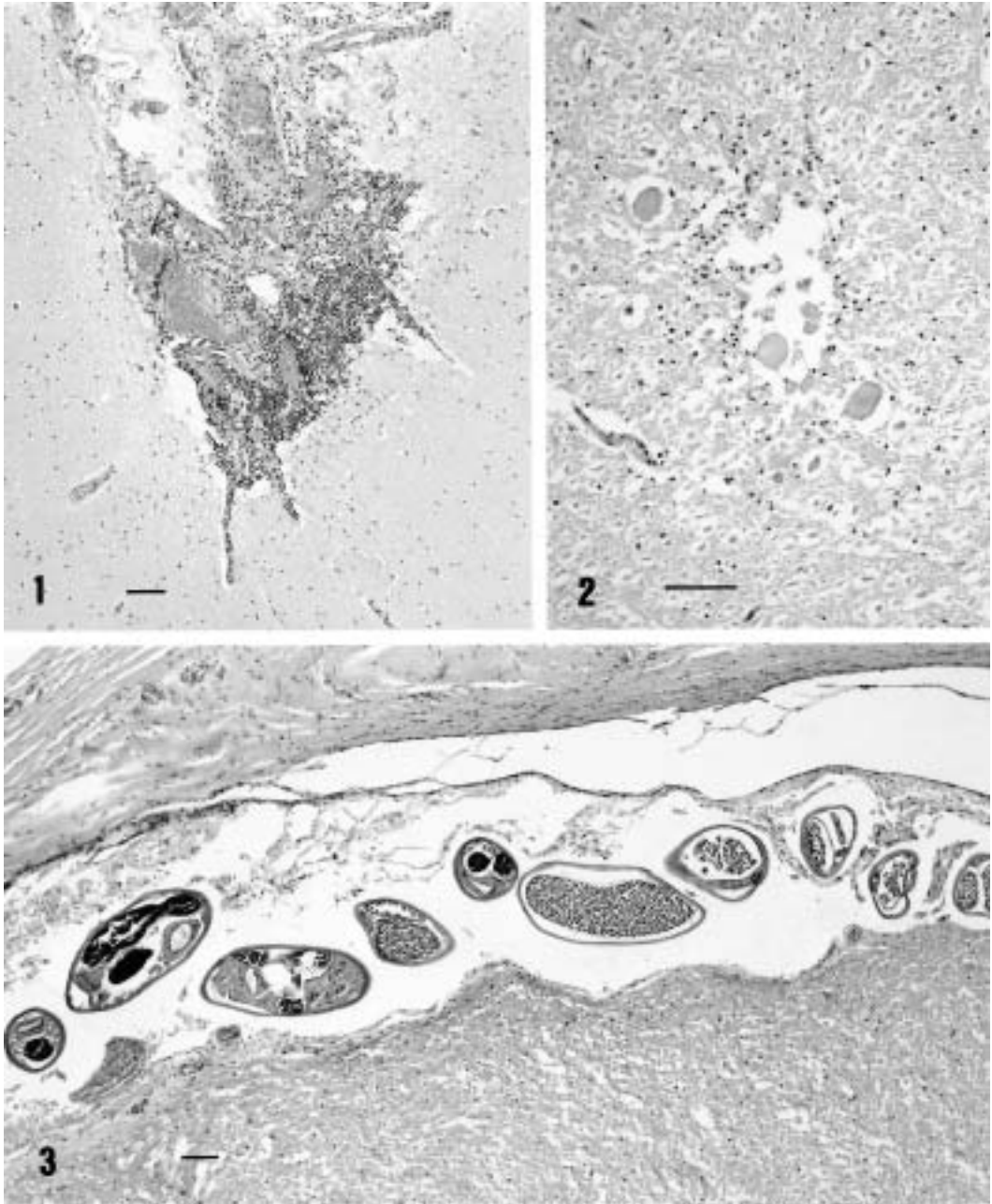


FIGURE 1. Cerebrospinal nematodiasis in a moose. Accumulation of inflammatory cells in a cerebral sulcus. HE stain. Bar=100 μ m.

FIGURE 2. Focus of traumatic malacia in white matter of the spinal cord of a moose with cerebrospinal nematodiasis. Axonal swelling and macrophage proliferation are present. HE stain. Bar=100 μ m.

FIGURE 3. Cross-sections of a mature *Elaphostrongylus* female in the subarachnoid space of the thoracic spinal cord of a moose with cerebrospinal nematodiasis. Ova are visible in the uterus. HE stain. Bar=100 μ m.

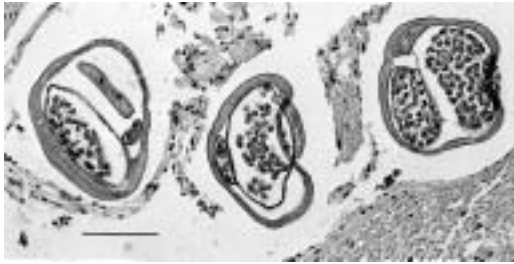


FIGURE 4. Closer view of nematode cross-sections shown in Figure 3.

who produced grave neurologic signs in a moose calf experimentally infected with *E. rangiferi* (called *E. cervi*) from caribou, and recovered two nematodes from the brain parenchyma.

The *Elaphostrongylus* infection in the present moose was not patent. Nor have patent infections been induced in sheep and goats experimentally infected with *E. rangiferi* or *E. cervi* (Handeland and Skorping, 1993; Handeland et al., 1993; Handeland et al., 2000a). In small ruminants, this seems to be due to mortality of larvae during development in the CNS, where only occasional nematodes reach the adult stage. A similar die-off not unlikely occur in moose when infected with these nematodes. The lymphohistiocytic granulomas found in the CNS of the moose could represent remains of parasitic granulomas that had formed around dead nematodes.

LITERATURE CITED

- GIBBONS, L. M., O. HALVORSEN, AND G. STUVE. 1991. Revision of the genus *Elaphostrongylus* Cameron (Nematoda, Metastrongyloidea) with particular reference to species of the genus occurring in Norwegian cervids. *Zoologica Scripta* 20: 15–26.
- HALVORSEN, O., J. ANDERSEN, A. SKORPING, AND G. LORENTZEN. 1980. Infection in reindeer with the nematode *Elaphostrongylus rangiferi* Mtskevich in relation to climate and distribution of intermediate hosts. *Proceedings of the International Reindeer/Caribou Symposium* 2: 449–455.
- HANDELAND, K. 1991. Cerebrospinal elaphostrongylosis in sheep in northern Norway. *Journal of Veterinary Medicine Series B* 38: 773–780.
- . 1994. Experimental studies of *Elaphostrongylus rangiferi* in reindeer (*Rangifer tarandus*) II. Life cycle, pathogenesis, and pathology. *Journal of Veterinary Medicine Series B* 41: 351–365.
- , AND L. M. GIBBONS. 2001. Aspects of the life cycle and pathogenesis of *Elaphostrongylus alces* in moose (*Alces alces*). *Journal of Parasitology* 87: 1054–1057.
- , AND O. SPARBOE. 1991. Cerebrospinal elaphostrongylosis in dairy goats in northern Norway. *Journal of Veterinary Medicine Series B* 38: 755–763.
- , AND A. SKORPING. 1992. Experimental cerebrospinal elaphostrongylosis (*Elaphostrongylus rangiferi*) in goats: II. Pathological findings. *Journal of Veterinary Medicine Series B* 39: 713–722.
- , AND ———. 1993. Experimental cerebrospinal elaphostrongylosis (*Elaphostrongylus rangiferi*) in goats: I. Clinical observations. *Journal of Veterinary Medicine Series B* 40: 141–147.
- , ———, AND T. SLETTBAKK. 1993. Experimental cerebrospinal elaphostrongylosis (*Elaphostrongylus rangiferi*) in sheep. *Journal of Veterinary Medicine Series B* 40: 181–189.
- , L. M. GIBBONS, AND A. SKORPING. 2000a. Experimental *Elaphostrongylus cervi* infection in sheep and goats. *Journal of Comparative Pathology* 123: 248–257.
- , ———, AND ———. 2000b. Aspects of the life cycle and pathogenesis of *Elaphostrongylus cervi* in red deer (*Cervus elaphus*). *Journal of Parasitology* 86: 1061–1066.
- HEMMINGSSEN, W., O. HALVORSEN, AND A. SKORPING. 1993. Migration of adult *Elaphostrongylus rangiferi* (Nematoda: Protostrongylidae) from the spinal subdural space to the muscles of reindeer (*Rangifer tarandus*). *Journal of Parasitology* 79: 728–732.
- LANKESTER, M. W. 1977. Neurologic disease in moose caused by *Elaphostrongylus cervi* Cameron 1931 from caribou. *Proceedings of the North American Moose Conference and Workshop* 13: 177–190.
- , AND D. FONG. 1998. Protostrongylid nematodes in caribou (*Rangifer tarandus caribou*) and moose (*Alces alces*) of Newfoundland. *Rangifer, Special Issue* 10: 73–83.
- OTTESTAD, A. K. 1983. The occurrence of *Elaphostrongylus cervi* in a population of red deer (*Cervus elaphus*). *Candidatus Realium Thesis*, University of Tromsø, Norway, 64 pp. [In Norwegian.]
- PUSTERLA, N., P. CAPLAZI, H. HERTZBERG, F. EHRENSPERGER, AND U. BRAUN. 2001. *Elaphostrongylus cervi* infection in a Swiss goat. *Veterinary Record* 148: 382–383.
- STEEN, M., A. G. CHABAUD, AND C. REHBINDER. 1989. Species of the genus *Elaphostrongylus* parasite of Swedish cervidae. A description of *E. alces* n. sp. *Annales de Parasitologie Humaine et Comparee* 64: 134–142.

- , AND C. REHBINDER. 1986. Nervous tissue lesions caused by elaphostrongylosis in wild Swedish moose. *Acta Veterinaria Scandinavica* 27: 326–342.
- STUVE, G. 1987. *Elaphostrongylus cervi* infection in moose (*Alces alces*). Prevalence and pathological changes in relation to age and season. *Acta Veterinaria Scandinavica* 28: 157–164.

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