Experimental studies of *Elaphostrongylus rangiferi* in reindeer *(Rangifer tarandus tarandus)*: Clinical observations

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Abstract: Clinical observations were made on 12 reindeer calves *(Rangifer tarandus tarandus)* experimentally infected with 200–1,000 infective larvae of *Elaphostrongylus rangiferi* and autopsied 2.5–196 days post inoculation (p.i). Seven experimental animals autopsied later than 20 days p.i. all developed neurologic signs starting 4-8 weeks p.i. In six of these animals, signs lasted until autopsy 0-12 weeks after onset. The seventh animal recovered completely after a disease period lasting five months. A dose-response relationship between the infective dose and severity of signs was observed. Clinical signs observed in all affected animals were paraparesis, tail paresis and posterior ataxia. Other signs included lowered head, general weakness, lameness, tetraparesis, scoliosis, anal hypotonia, head and neck turn, depression and reduced vision. The prepatent period was 4–4.5 months.

Key words: Cervidae, Protostrongylidae, neurologic disease.

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Introduction

Nematodes of the genus *Elaphostrongylus* parasitize the central nervous system (CNS) and skeletal muscles of ruminants in the family Cervidae. Four species have been described: *E. cervi* Cameron 1931 and *E. panticola* Lubimov 1945 from *Cervus* spp., *E. rangiferi* Mitskevich 1960 from reindeer *(Rangifer tarandus tarandus),* and *E. alces* Steen, Chabaud and Rehbinder 1989 from moose *(Alces alces).*

Naturally occurring neurologic disease due to *Elaphostrongylus* infection has been reported in reindeer, red deer *(Cervus elaphus)* and moose in Fennoscandinavia (Roneus & Nordkvist, 1962; Groholt, 1969; Bakken & Sparboe, 1973; Kummeneje, 1974; Borg, 1979; Holmström *et al.*, 1989; Steen & Roepstorff, 1990; Handeland & Norberg, 1992), and in reindeer and *Cervus* spp. in the former USSR (Pryadko *et al.*, 1963; Polyanskaya, 1965). The parasites have also been reported to cause disease in sheep and goats (Handeland, 1991; Handeland & Sparboe, 1991).

Neurologic disease in cervids following experimental *Elaphostrongylus* infections has been reported in red deer calves (Watson, 1983), one caribou *(Rangifer tarandus caribou)* and two moose calves (Lankester, 1977; Stuve & Skorping, 1987).

An infection experiment was carried out to

study the clinical disease and the life cycle and the pathogenesis of *E. rangiferi* in reindeer. Results of the life cycle and pathogenetic studies are given in Handeland (1994). This report presents clinical observations of the animals in the experiment.

Material and methods

Animals and inoculums

L_1 of *E. rangiferi* were obtained from naturally infected reindeer. Snails (*Arianta arbustorum*) were reared in the laboratory, exposed to larvae and examined as described in Skorping (1982). The number of snails necessary to give the predetermined infective dose was decided on the basis of the L_3 average of 18 (SD 15) found in 50 randomly picked snails.

Fourteen 3–3.5 month old reindeer calves (live weight 30–44 kg) were used in the experiment. Twelve of the calves were brought from a semi-domesticated herd in the Tromsø region on August 20, 1992, whereas the remaining two (Table 1; Calves 3 and 9) were born in captivity at the Department of Arctic Biology, University of Tromsø. All animals were kept indoors on a drained floor from the day of arrival of the semi-domesticated calves. During the experiment, all calves were fed a commercially available concentrate composed for reindeer (RF 80) ad libitum, with the exception of the first 3–4 weeks when the 12 semi-domesticated calves were also fed fresh grass.

On August 20, 12 calves were given an aqueous suspension of squashed snails containing approximately 200, 300 or 1,000 L_3 by stomach tube (Table 1). Two remaining calves were kept as controls.

Calf 5 refused to eat the concentrate, consumed only small amounts of grass and died from inanition 20 days post inoculation (p.i.). The remaining calves were anaesthetized with barbiturates, bled and autopsied 2.5–196 days p.i. (Table 1).

Clinical observations

During the first six weeks p.i., weekly faecal samples of 3 g were examined for L_1 after baermannisation. Thereafter, this examination was performed twice a week. Faecal sampling started at day 0 of the experiment.

All reindeer calves were studied daily while standing and while exercised indoors. Every fortnight, the animals were studied while exercised in an outdoor enclosure. Any abnormali-

Table 1. Sex, infection dose, time of autopsy, and first observation, duration and nature of neurologic signs of two control and 12 experimental reindeer calves inoculated with *Elaphostrongylus rangiferi*.

Calf No.	Sex	No. of larvae	Autopsy[A]	Neurologic signs			
				First observation[A,B]	Duration[C]	Brain disturbances	Locomotor disturbances
1	M	1,000	2.5 dpi	–	–	–	–
2	M	1,000	4 dpi	–	–	–	–
3	F	1,000	6 dpi	–	–	–	–
4	M	1,000	12 dpi	–	–	–	–
5	M	200	20 dpi	–	–	–	–
6	M	1,000	4.5 wpi	4.5 wpi	u.a.	–	+
7	M	1,000	6.5 wpi	4.5 wpi	u.a.	–	+
8	M	1,000	8.5 wpi	5 wpi	u.a.	–	+
9	M	1,000	10.5 wpi	5 wpi	u.a.	+	+
10	F	1,000	15 wpi	4 wpi	u.a.	–	+
11	M	200	20 wpi	8 wpi	u.a.	–	+
12	M	300	28 wpi	5.5 wpi	5 months	+	+
13	M	0	21 wpi	–	–	–	–
14	M	0	26 wpi	–	–	–	–

[A] dpi = days post inoculation; wpi = weeks post inoculation

[B] The first neurologie signs observed in all animals were disturbances of locomotion

[C] u.a. = until autopsy

ties in their behaviour, apparent mental status, vision, posture and gait were recorded.

Results

Parasitological findings

The first detection of L_1 of *E. rangiferi* was obtained in the faeces of calves 11 and 12 at 4.5 and 4 months p.i., respectively. None of the other calves passed L_1 in the faeces during the experiment.

Neurologic signs

A summary of the neurologic signs of individual animals in the experiment is given in Table 1.

Calves 6–10 (1000 L_3):

Calves 6–10 showed signs of locomotor disturbance starting 4–5 weeks p.i. The initial signs were asymmetric paraparesis, posterior ataxia, and incompletely raised tails while moving.

From 5–6 weeks p.i., calves 7–10 showed a reluctance to rise and move. Lowered pelvis (Fig. 1) and head positions were observed both when standing and moving. While standing, the hocks frequently were rotated inwards (Fig. 2) and while moving, a tendency to knuckle over in the pelvic limbs was noticed.

From about 8 weeks p.i., calves 8–10 were observed to collapse on their hindquarters while turning, and their hocks sometimes bowed outwards when walking.

Thereafter, calves 9 and 10 gradually developed a stiff, short-spaced and cautious locomotor pattern that also seemed to affect the thoracic limbs. Both animals gave an impression of general weakness. Calf 9 also developed signs of reduced vision.

From about 12 weeks p.i., calf 10 readily collapsed on its hindquarters when moving. Locomotion was slow and cripple-like with the pelvis drifting slightly to the right resulting in scoliosis. The pelvic limb paresis became profound (Fig. 3). Extreme bowing of the hocks to the right could be observed during standing (Fig. 4), and from this position the calf frequently collapsed on its hindquarters. Sometimes the calf barely managed to rise. It soon became exhausted during exercise and could easily be caught. The tail was incompletely raised during defecation and reduced anal muscular tonus was observed. The perineal region and tail became smeared with faeces.

Calves 11–12 (200–300 L_3):

In calf 11, a slightly lowered pelvis when standing, slight posterior ataxia while turning, and incompletely raised tail when moving, were observed from about 8 weeks p.i. These signs were variably present throughout the rest of the experiment.

In calf 12, a moderate lameness on the left pelvic limb appeared from 5.5 week p.i. During the following week, incomplete tail elevation when moving, slight posterior ataxia and slightly lowered head and pelvis were also recognized. These signs remained stable or increased slightly during the period 7–20 weeks p.i. However, a passing remission of the pelvic limb lameness was observed 9–11 weeks p.i. From about 12 weeks p.i. the calf also showed signs of depression, and at 13 weeks p.i. a two day period with a right head and neck turn was observed.

From about 20 weeks p.i., all clinical signs of calf 12 gradually declined until they had completely disappeared at about 27 weeks p.i.

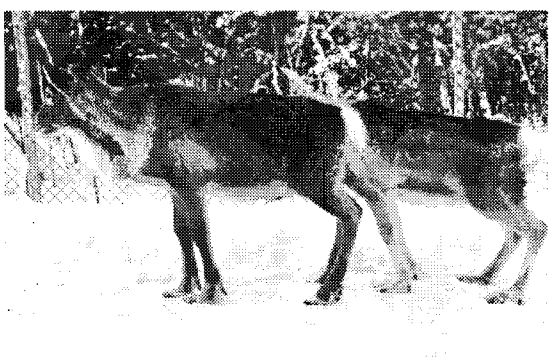

Fig. 1. Calves 9 (left) and 10 at 7.5 weeks post inoculation with *Elaphostrongylus rangiferi*. Slightly to moderately lowered pelvis due to pelvic limb paresis; the bulk of the animal's weight is placed on the thoracic limbs.

Fig. 2. Calves 9 (right) and 10 at 7.5 weeks post inoculation with *Elaphostrongylus rangiferi*. Inward rotation of the hocks because of pelvic limb paresis.

Fig. 3. Calf 10 at 13.5 weeks post inoculation with *Elaphostrongylus rangiferi*. Profound pelvic limb paresis. Markedly lowered head and pelvis, pelvic limbs widely spaced, and the bulk of the animal's weight is placed on the thoracic limbs.

Fig. 4. Calf 10 at 13.5 weeks post inoculation with *Elaphostrongylus rangiferi*. Right rotation of the hocks because of pelvic limb paresis.

Discussion

All seven infected reindeer calves autopsied later than day 20 p.i. developed signs of neurlogic disturbances. In infection experiments with *E. rangiferi* in goats and sheep using similar inoculums to the present study, only six of 17 goat kids and none of seven lambs autopsied later than 20 days p.i. developed signs of neurologic disturbances (Handeland & Skorping, 1993; Handeland *et al*, 1993). Thus, *E. rangiferi* seems to be most pathogenic in reindeer, intermediately pathogenic in goats, and least pathogenic in sheep. The pruritus that commonly occurred in the experimentally infected goats and sheep was not observed in reindeer.

Asymmetric paraparesis, tail paresis and posterior ataxia were the most common neurologic signs, observed in all affected calves in the present study. Posterior locomotor disturbances were also the most frequently reported signs in naturally *E. rangiferi* infected reindeer (Roneus & Nordkvist, 1962; Bakken & Sparboe, 1973; Kummeneje, 1974), goats (Handeland & Sparboe, 1991) and sheep (Handeland, 1991), as well as in experimentally *E. rangiferi* infected goats (Handeland & Skorping, 1993). These were also the most frequently reported signs in *E. alces* infected moose (Stuve & Skorping, 1987; Steen & Roepstorff, 1990) and *E. cervi* infected *Cervus* spp. (Pryadko *et al.*, 1963; Borg, 1979; Watson, 1983) and guinea pigs (Watson & Gill, 1985; Demiaszkiewicz, 1989). Thus, posterior locomotor disturbances appear to be the main clinical manifestation of *Elaphostrongylus* infections.

The neurologic signs of the reindeer calves started 4–8 weeks p.i. Signs appeared earlier and were more severe in calves receiving the larger compared to smaller numbers of L_3. This probably reflects the presence of a dose-response relationship between the infective dose and time of occurrence and severity of neurologic signs.

Six of the seven affected calves were killed while showing clinical signs, 4.5–20 weeks p.i. and 0–12 weeks after onset of signs. The seventh calf recovered about six months p.i., after a disease period lasting five months. Stuve and Skorping (1987) reported the recovery from neurologic disease of a moose calf six months p.i. with *E. alces*, after a disease period of 3.5 months. These observations indicate that a long duration of clinical signs may be expected in *Elaphostrongylus*-infected cervids; signs being present up to 0.5 year p.i. The main infection of hosts probably takes place in the autumn when the gastropod intermediate hosts are numerous and contain many infective larvae (Mitskevich, 1958; Pryadko *et al.*, 1963). This would suggest that naturally occurring disease is likely to occur during winter. Thus, *Elaphostrongylus* infections eliciting long lasting neurologic disease during winter may be an important determinant of survival for cervids, especially in northern areas with their long and severe winters.

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