

Meningeal Worm in Central Iowa Goat Herds

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Summary and Implications

Aberrant migration of the deer meningeal worm, *Parelaphostrongylus tenuis*, is a commonly recognized cause of neurologic disease in South American camelids but is less frequently considered in other small ruminant species. After an unusually cool and wet summer season, central Iowa goat herds serviced by Iowa State University's Veterinary Field Services have been increasingly affected by *P. tenuis*, with clinical cases presenting as early as August. Most caprine cases present with progressive neurologic deficits, starting with hind-end weakness and ataxia which may lead to complete paresis and death. Both sporadic cases and herd outbreaks involving as much as 15% of the herd have been observed. These cases involve a range of ages from older pre-weaned kids (4-6 months) to mature animals. Herds browsing wooded areas with a high deer density appear to be especially at risk. Field diagnosis is based on exposure history, clinical presentation, cerebrospinal fluid analysis, and response to treatment. Treatment protocols include extra-label high-dose, extended course fenbendazole, anti-inflammatories, and supportive care along with single dose ivermectin for control of immature larvae. Ambulatory animals generally respond to treatment but may retain residual neurologic deficits; prognosis for downer animals is poor. Control effort may include restricting access to high-risk browse or grazing sites, exclusion of deer and mollusks, and chemoprophylaxis.

Introduction

While retrospective studies suggest that aberrant migration of the white-tailed deer meningeal worm, *Parelaphostrongylus tenuis*, is a relatively infrequent cause of spinal cord lesions in goats, it has become increasingly common in our clinical practice. *P. tenuis* is enzootic in the Iowa white-tailed deer (*Odocoileus virginianus*) population and the author has observed an uptick in presumptive and definitively diagnosed cases of meningeal worm among client herds. Anecdotal reports from producers suggest that other areas of the Upper Midwest are similarly affected. It appears that this year's unseasonably cool and wet summer has contributed to both greater and earlier exposure to the parasite, especially for goats grazing woodland habitat. Under normal conditions, *P. tenuis* completes an indirect neurotropic life-cycle that passes between deer (definitive host) and a variety of terrestrial gastropods (intermediate host). This life-cycle typically takes four months to

complete and little to no clinical disease is observed in infected deer.

Goats and other accidental hosts become infected by ingesting infective L3 larvae present either in the snail or slug, or in the slime trail. Once ingested, normal larval maturation is disrupted, resulting in aberrant migration through the spinal cord and CNS. No eggs are produced, but the neural inflammation and parenchymal damage from aberrant migration causes clinical central nervous system disease with variable signs depending on the anatomic location of the lesions. Although intra-cranial deficits, upper motor-neuron, lower-motor neuron, and sensory deficits have all been described in a variety of species, in goats the most common signs include upper- and lower-motor neuron and peripheral limb sensory deficits.

Clinical Presentation and Diagnostic Approach

Given that the meningeal worm life cycle lasts four months in deer, that there is a forty-five to sixty day delay from infection to clinical disease in accidental hosts, and that mollusk activity declines during winter hibernation and mid-summer aestivation, most cases of meningeal worm present over the fall and winter season. This year, affected goats started showing signs of meningeal worm as early as the beginning of August, suggesting that the mollusks' aestivation period was reduced by the moderate summer weather. On presentation, exposure to white-tailed deer was a common, though non-discriminating finding in the history and most cases had been exposed to substantial browse in wooded areas. Affected goats generally remained bright and alert with a good appetite and no fever; however, clients complained that the animal was lame, stiff, weak, ataxic, slow or reluctant to move, or unable to rise. The most consistent physical exam findings include asymmetric multi-focal neurologic disease with emphasis on pelvic limb deficits. Specific signs varied depending on the anatomic location of the lesions, but evidence of ataxia, hemi- or tetra-paresis, impaired proprioception, decreased nociception, and altered reflexes were common. Although asymmetric cranial nerve deficits (including facial paralysis and loss of menace reflex) have been reported in goats, they are less common than spinal lesion signs and were not observed in these cases. Clinical signs progressed over the course of several weeks, terminating in death or euthanasia for a minority of cases. Peritonitis and colic symptoms have been reported in goats after high-dose experimental infections but were not observed in these cases. *P. tenuis* associated linear pruritic dermatitis lesions have also been reported; several of these herds did have issues with focal hair loss and itching over the body or trunk but the pruritus and alopecia could not be definitively associated with meningeal worm infection.

Differentials for hemi-paretic cases included copper deficiency (enzootic ataxia, swayback), compressive lesions (vertebral malformation, trauma, abscess, or neoplasia), caprine arthritis-encephalomyelitis virus, and other putative viral causes of nonsuppurative meningoencephalomyelitis (such as rabies, pseudorabies, border disease, and theoretically West Nile virus). Presentations characterized by flaccidity or stiff, spastic movement and hyperreflexia could also be consistent with early stages of tetanus, laminitis, nutritional muscular dystrophy (vitamin E / selenium deficiency), polyradiculoneuritis, and myotonia congenita. Tetraparetic and terminal cases of caprine meningeal worm were differentiated from common causes of “downer” animals (polioencephalomalacia, listeriosis, pregnancy toxemia, and hypocalcemia) as well as less commonly etiologies (brainstem abscess, bacterial or viral meningoencephalitis, tick paralysis, organophosphate toxicity, and lidocaine toxicity). Rabies and scrapie are always considered on any open caprine neurologic case.

Diagnosis of *Parelaphostrongylus tenuis* is currently complicated both by the lack of antemortem tests as well as difficulty in locating the parasite on necropsy. Presumptive antemortem diagnosis was made based on history of exposure to white-tailed deer habitat, clinical signs and CSF abnormalities, exclusion of other differentials, and response to treatment. CSF was collected by lumbosacral puncture from representative cases and consistently demonstrated a marked eosinophilic pleocytosis. When present, high levels of eosinophils in the CSF is a near-pathognomonic finding; unfortunately, in early cases the CSF may be normal or only demonstrate elevated monocyte counts, which is more commonly associated with listeriosis or viral meningitis. Standard CBC and serum chemistry profiles obtained from representative cases were largely unremarkable, which is typical for meningeal worm. Definitive diagnosis of *Parelaphostrongylus tenuis* spinal larval migrans was made through postmortem observation of relatively characteristic necrotic and/or eosinophilic inflammatory lesions associated with migration tracts and nematode segments in the spinal tissue sections. A nested PCR technique available at the University of Tennessee was applied to formalin-fixed, paraffin-embedded tissues with success; this nPCR reportedly may work in the absence of visible parasite.

Treatment, Prognosis, and Prevention

Clinical therapy focused on three objectives: killing the parasite, reducing CNS tissue inflammation, and supporting vital functions. Treatment protocols included anthelmintic and anti-inflammatory medication along with ancillary supportive therapies and nursing care as clinically indicated; all drugs and doses used are extra-label in goats and must be administered under the advice of a veterinarian, with extended drug withdrawal intervals. Cases were treated with five days of high-dose oral fenbendazole (50 mg/kg), a single dose of ivermectin (0.4 mg/kg orally or 0.2 mg/kg SQ), and an anti-inflammatory (dexamethasone 0.1 mg/kg

IM or flunixin meglumine 1-2 mg/kg IV). Additional recommended ancillary therapies could include parenteral administration of vitamin B or B-complex, vitamin E as a non-specific antioxidant, and fluid therapy. Attention to primary husbandry considerations included ensuring access to shelter, food, and water. Downer animals can benefit from ample bedding and physiotherapy to limit secondary pressure myopathy, compartment syndrome, formation of decubital lesions, and contracted tendons.

Treatment prognosis is inconsistently dependent on severity of clinical signs at presentation; spontaneous recoveries have been reported. In general, recumbent animals and those exhibiting additional signs of depression or anorexia have a poorer prognosis for survival and require much more extensive supportive and nursing care. Ambulatory cases generally have a good to fair prognosis for survival and productivity, but progress is incremental. Neurologic deficits present six months or more after treatment are likely to be permanent. Of the cases that presented this year, most animals showed significant improvement and will be retained in the herds. Approximately 10% of the ambulatory cases died, were euthanized, or were culled for failure to improve enough to be productive in an extensive program.

Meningeal worm prevention strategies can be grouped into chemoprophylaxis and management-centric options. Chemoprophylaxis with ivermectin or doramectin has been used prophylactically in high-risk camelids to kill the early larval stages before they access the CNS. When used in this manner, they are often administered monthly, year-round, but can be targeted seasonally in some climates; for example, in locations that experience significant hard freezes and hot, dry summers, 85% of meningeal worm exposure occurs from September to December. Routine chemoprophylaxis is not routinely recommended for goats, as it promotes anthelmintic resistance in common gastrointestinal nematodes such as *Haemonchus contortus*. While vaccine development efforts are underway, they are focused on alpacas and have not yet progressed to a conditionally-available product. Although management strategies theoretically include excluding white-tailed deer and mollusks from the grazing areas through fencing, vegetative barriers, and/or mollusk control, as well as limiting livestock access to wet or marshy areas, on most operations it is effectively impossible to exclude deer or terrestrial gastropods from the goat pastures. Furthermore, applying copper sulfate molluscicides to pond perimeters will have little effect on dry-land or arboreal gastropods and will have ecological and animal health implications. In practice, some operations were able to restrict the goat's access to known high-risk woodlands. Offset (“3D”) electric strand fencing was discussed as a relatively cost-effective solution for deer exclusion and guinea hens could be considered as a more environmentally-sustainable approach to snail and slug control; however, the specific efficacy of those interventions is not known.

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