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Nov 5, 2002, 10:28am

<http://www.vet.ohio-state.edu/docs/ClinSci/camelid/mening.html>

Parelaphostrongylus Tenuis (Meningeal Worm) Infection in Llamas and Alpacas

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The meningeal worm (*Parelaphostrongylus tenuis*), also known as the deer worm or meningeal deer worm, frequently infects llamas and alpacas. Aberrant migration of the meningeal worm in susceptible hosts such as llamas and alpacas causes damage to the central nervous system and may result in death.

Identification and Life Cycle

The meningeal worm is a nematode parasite belonging to the family Protostrongylidae. The definitive host is the white-tailed deer (*Odocoileus virginianus*) prevalent throughout much of eastern North America.¹ Adult meningeal worms reside in the meninges of white-tailed deer and rarely cause clinical signs of disease.^{1,2}

Adult worms lay eggs in the meninges of white-tailed deer. The eggs then pass into the venous circulation and travel to the lungs where they hatch into first-stage larvae (L1). The L1 are coughed up, swallowed, and passed in the feces of infected deer. Larvae then invade or are ingested by snails or slugs (terrestrial gastropods). Snails and slugs serve as intermediate hosts in which the first stage larvae develop into infective third stage larvae (L3) over a period of 3-4 weeks.¹⁻³

Infected snails or slugs are then ingested by susceptible aberrant hosts such as llamas, alpacas, goats, sheep, moose, wapiti, caribou, black-tailed deer, and red deer¹, and the L3 are released in the digestive tract. Infective third stage larvae migrate to the spinal cord and continue to migrate aimlessly within the central nervous system causing neurologic disease.¹⁻³

In the definitive host, the white-tailed deer, the infected snails or slugs are ingested and the L3 are released in the abomasum. The L3 then migrate to the spinal cord via the spinal nerves over the next 10 days. The larvae mature in the dorsal horns of the gray matter of the spinal cord for 20-30 days. Adult meningeal worms migrate to the subdural space, then to the brain through the dura mater and cranial venous sinuses.² The prepatent period in deer is 82-92 days.^{2,4}

Many snails and slugs prefer a moist or wet environment for survival. Consequently, low-lying and wet, poorly drained fields provide an increased risk of exposure to snails and slugs.³ However, exposure risk is not limited to wet climates since dry-land snails and slugs may serve as intermediate hosts. Snails and slugs feed on organic matter, leaf litter, and vegetation. Survival of L3 outside the intermediate host is believed to be short-lived unless water is available. Repeated freezing or desiccation has been shown to decrease survival of the infective L3.² Therefore, the risk of exposure to llamas and alpacas is lowest when there are prolonged periods of dry-heat or deep freezes.

Clinical Disease

Once the aberrant host is infected, clinical disease begins 45-53 days later as demonstrated by experimental inoculation.⁴ Clinical neurologic disease is the result of tissue destruction and inflammation caused by randomly migrating larvae. Thus, the clinical signs observed depend upon the location of the migrating larvae.³

Most commonly, clinical signs reflect asymmetrical, focal spinal cord lesions.⁴ These include hypermetria,^{2,5} ataxia,^{1,2,5,6} stiffness,^{1,2} muscular weakness,^{2,5,6} posterior paresis,^{2,6} paralysis,^{1,2} head tilt,² arching neck,² circling,^{1,2} blindness,^{1,2} gradual weight loss,² apparent depression, seizures, and death.² Clinical signs generally begin in the hind limbs and progress to the front limbs.^{2,4} The course of disease may be acute to chronic, ranging from death within days to ataxia which lasts months to years.² In our experience, clinical signs of meningeal worm infection are exacerbated during summer months because heat stress develops with prolonged periods of recumbency.

Differential Diagnoses

Clinical signs suggestive of meningeal worm infection are non-specific and may affect the spinal cord or brain. Clinical signs of spinal cord lesions include weakness, ataxia, gait abnormalities, lameness, proprioceptive deficits, paresis, and paralysis. Differential diagnoses (Table 1) for camelids with these clinical signs may include vertebral body subluxation or fracture, vertebral body abscessation, trauma, spinal

neoplasia, degenerative myelopathy, metabolic diseases such as copper deficiency in neonates, listeriosis, heat stress, and tick paralysis. Clinical signs of intracranial disease include ataxia, abnormal mentation (dementia, stupor, coma), visual abnormalities, circling, falling or rolling, weakness, delayed postural reactions, incoordination, head tilt, altered head and neck position, nystagmus, strabismus, and seizures. Differential diagnoses for camelids with these signs may include neoplasia, trauma, hydrocephalus or other congenital defects, cerebral abscessation, listeriosis, otitis interna, and polioencephalomalacia. Electrolyte imbalances such as hypocalcemia, hypomagnesemia, and hypoglycemia, ketosis, and dietary deficiencies such as copper, vitamin A, vitamin E, selenium, calcium, magnesium, potassium, and phosphorus may each present neurologic signs of disease. In addition, consider toxicoses such as lead poisoning, ingestion of poisonous plants, and salt poisoning. Rabies encephalitis may present with a variety of neurologic signs and should be considered in any neurologic case. These differential diagnoses must be ruled out prior to making a presumptive diagnosis of meningeal worm infection. Although consistent clinical signs and CSF eosinophilia are highly suggestive of meningeal worm infection, antemortem diagnosis of aberrant *Parelaphostrongylus tenuis* migration is often a diagnosis of exclusion.

Diagnostic Testing

To thoroughly evaluate the patient, collect a database of information which includes the signalment, history including the onset and progression of clinical signs, complete blood count, and serum chemistry profile. Additional diagnostic testing include vertebral radiography, cerebrospinal fluid (CSF) analysis and culture, CSF creatine kinase activity, and plasma fibrinogen concentration. In select cases, advanced diagnostic testing such as myelography, X-ray computed tomography (CT), magnetic resonance imaging (MRI), or electromyography (EMG) may be indicated to rule-out other causes of spinal or intra-cranial disease.

Diagnosis of Meningeal Worm

A presumptive diagnosis is based upon clinical signs consistent with meningeal worm infection, history of exposure to areas inhabited by white-tailed deer, and response to treatment.^{2,6} No consistent abnormalities in CSF total protein or glucose concentrations, AST or CK activities were identified in llamas experimentally infected with *P. tenuis*⁴ (Table 2). The only consistent abnormality was a shift in nucleated cell count from predominantly lymphocytes and monocytes to eosinophils over the course of infection.^{4,9}

The presence of clinical signs and CSF eosinophilia may be used to make a tentative diagnosis of *P. tenuis* infection in llamas.^{2,4,6,7} Cerebrospinal fluid analysis may show eosinophilia, but absence of eosinophilia on CSF analysis does not rule out the diagnosis of meningeal worm infection.^{2,5,6} However, CSF eosinophilia in llamas has been most consistently reported in cases of clinical parelaphostrongylosis.^{2,6,9} Hematologic samples may show peripheral eosinophilia but frequently show no abnormalities.^{2,6}

One study showed a significant *P. tenuis* - specific serum antibody response in goats experimentally infected with 50 *P. tenuis* L3.⁸ Serum antibody titers were highest 8 weeks after infection. Results of this study suggest that a serum enzyme-linked immunosorbent assay (ELISA) using antigens of adult *P. tenuis* would be beneficial in the diagnosis of clinical parelaphostrongylosis in goats.² Modification of this test may show promise in detecting parelaphostrongylosis in llamas.^{2,9}

Definitive diagnosis of meningeal worm infection is made at necropsy. A confirmed diagnosis requires microscopic demonstration of the larvae within the brain or spinal cord.^{2,9} Microscopic examination of brain and spinal cord tissue may also show linear paths of tissue damage or inflammation suggestive of migrating tracts made by the larvae.⁶

Therapy

Treatment of meningeal worm infection is most successful when instituted early in the course of disease. A treatment regime (Table 3) which has proven successful at the Ohio State University involves fenbendazole (20 to 50 mg/kg body weight, PO, q24h for 5 days) and flunixin meglumine (1 mg/kg, IV, IM, or SC, q12h for 5 days) or dexamethasone in non-pregnant females and males (0.1 mg/kg, IV, IM, or SC, q24h for 3 days). DMSO (1g/kg given in 500 ml of 5% dextrose solution, IV, q24h) given to effect is useful in some cases but may cause severe appetite suppression. Discontinue DMSO if inappetence or anorexia occurs. Vitamin E, selenium, Vitamins B-complex, and Vitamin A are useful to assist healing of neural tissues.

Dexamethasone should not be administered to pregnant females because this drug may induce abortion. Alternatively, we have used prednisolone sodium succinate (0.5-1.0 mg/kg, IV, IM, or SC, q12h) for no more than three days in pregnant females without subsequent abortion. Prednisolone sodium succinate may have a reduced risk of abortion compared to dexamethasone because it lacks a carbon-16 substitution. Corticosteroids lacking a C-16 substitution may not cross the blood-placental barrier and large doses for prolonged periods of time may be required to terminate pregnancy.¹⁰

Ivermectin is most effective against larval stages prior to their entrance into the spinal cord, since it does not readily cross the blood-brain barrier.^{1,2,11} However, damage to nervous system tissues during larval migration may alter the blood-brain barrier. Although no clinical problem has been identified to date, we have been concerned for the possibility of ivermectin toxicity in these cases. The antiinflammatory drugs are critical to reduce the inflammation associated with the presence of the migrating larvae and the subsequent inflammatory response to the killed larvae. Use of antiinflammatory drugs is important to prevent the clinical signs from becoming more severe after instituting treatment.

In addition to drug therapy, supportive care and physical therapy are essential to aiding recovery. Using slings to support llamas that are unable to stand and performing physical therapy for muscles are beneficial. We also have used hydrofloatation therapy to facilitate recovery after prolonged recumbency (Figure 1). A great deal of perseverance is required to care for severely affected llamas; recovery may take several weeks to months to years.²

Prognosis

Prognosis for survival depends upon how severe the clinical signs become. In our experience, llamas that are unable to stand have a poor prognosis (10-20% recovery); llamas that are able to stand unaided have a fair to good prognosis (75-85% recovery). Llamas that survive clinical disease do not seem to develop patent infections and are unlikely to pose a health risk to other animals.^{2,3} Many animals suffer permanent neurologic deficits but may remain productive members of the herd for breeding and pets.

Prevention

Prevention of meningeal worm infection may be difficult. Ideally, llamas should not graze the same pasture as white-tailed deer.^{2,9} However, in many areas of the United States, it is not feasible to separate the two populations. Placing a deer-proof fence may offer some protection, but many fences do not present a sufficient barrier to prevent movement of deer.^{1,2} Additionally, thick ground cover can be removed to expose the environment to fluctuations in temperature, and vegetation-free buffer zones (i.e. gravel, limestone) can be placed around fencelines to reduce migration of snails and slugs into the pasture.^{2,9} Molluscicides may be considered to destroy snails and slugs which serve as intermediate hosts, thereby interrupting the life cycle of the meningeal worm and preventing infection in aberrant hosts.¹ Drainage should be established in low lying areas and access to swampy areas may be restricted by fencing off these areas. These compounds present a potential environmental risk from contamination of ground water and may be toxic if consumed by camelids or other animals.

Prophylactic treatment against migrating larvae may be achieved administration of ivermectin (0.2 mg/kg) every 30 to 45 days during the high risk periods or throughout the year regions which have mild summers and winters. Anthelmintic resistance is unlikely to become a problem in the meningeal worm because these infections do not become patent.² However, meningeal worm infection has occurred in some herds that maintain vigilant prophylaxis. These "breaks" in prevention of the larval migration may have been caused by insufficient dosing of anthelmintic, accidental failure to administer the anthelmintic, or some unknown mechanism.

Conclusion

Meningeal worm infection may be severely debilitating and potentially fatal, but infection can be effectively prevented. Routine dewormings every 4-6 weeks, minimized cohabitation with white-tailed deer, and a clean, dry environment unfavorable for the growth of snails and slugs will considerably reduce the herd's risk of infection with the meningeal worm.

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Table 1. Differential Diagnoses for camelids showing clinical signs of apparent neurologic disease.

| Spinal Cord Disease | Intracranial Disease |
|---------------------|----------------------|
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|-----------------------------------|---------------------------------------|
| Vertebral body subluxation | Neoplasia |
| Vertebral body fracture | Trauma |
| Vertebral body abscessation | Congenital defects (hydrocephalus) |
| Trauma | Cerebral abscessation |
| Neoplasia | Listeriosis |
| Degenerative myelopathy | Otitis interna |
| Copper deficiency (youngstock) | Polioencephalomalacia |
| Listeriosis | Electrolyte imbalances |
| Heat stress | Dietary deficiencies |
| Tick paralysis | Toxicoses |
| | Rabies encephalitis |

Table 2. Cerebrospinal fluid findings in normal and Parelaphostrongylus tenuis infected llamas.

| Variable | Normal⁷ | Meningeal Worm^{4,*} |
|---------------------------|---------------------------|---|
| Total Protein (mg/dl) | 43 ± 9 | 44 ± 11 |
| Creatine Kinase (IU/l) | 4.6 ± 4.7 | 48.8 ± 91.8 |
| RBC (cells/μl) | 256 ± 394 | 329 ± 384 |
| WBC (cells/μl) | 0.9 ± 1 | 110 ± 230 |
| PMN (%) | 27 ± 30 | 1.8 ± 4 |
| Lymphocytes (%) | 54 ± 29 | 9 ± 13 |
| Monocytes (%) | 15 ± 13 | 39 ± 30 |
| Eosinophils (%) | 1.6 ± 3 | 43 ± 33 |

* Samples obtained 60 days after experimental infection with P. tenuis

Table 3. Treatment options used for llamas with parasitic myelitis at Ohio State University.

| | |
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| <u>Anthelmintics</u> | <u>Antiinflammatories</u> |
|--|--|
| Ivermectin 0.3 mg/kg SC, q24h x 5d | Flunixin 1mg/kg IV, IM, or SC, q12h x 5d |
| Fenbendazole 20 to 50 mg/kg PO, q24h x 5d | Dexamethasone 0.1mg/kg * IV, IM, or SC, q24h x 3d |
| | Prednisolone sodium succinate 0.5-1mg/kg– IV, IM, or SC, q12h x 3d |
| | DMSO 1g/kg IV, q24h to effect |

* Not for use in pregnant animals

_ Use extreme caution in pregnant animals

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