

## CEREBROSPINAL PARELAPHOSTRONGYLOSIS IN LLAMAS

TALMAGE T. BROWN, JR.,<sup>□</sup> Department of Veterinary Pathology, College of Veterinary Medicine, Oklahoma State University, Stillwater, Oklahoma 74074, USA.

HELEN E. JORDAN, Department of Veterinary Parasitology, Microbiology, and Public Health, College of Veterinary Medicine, Oklahoma State University.

C. NED DEMOREST,<sup>▣</sup> Institute of Comparative Medicine, Baylor College of Medicine, Houston, Texas 77035, USA.

**Abstract:** Four of 11 llamas (*Lama guanicoe*) from a ranch near Houston, Texas developed clinical signs of a neurologic disease. Tissues from 2 of the 4 llamas were submitted for microscopic examination. Lesions in the central nervous system of both llamas appeared to have been caused by a migrating parasite. An adult nematode having morphologic features of the meningeal worm (*Parelaphostrongylus tenuis*) was found in the brain of one llama. The findings suggest that the neurologic disease in the llamas were caused by *P. tenuis*.

### INTRODUCTION

White-tailed deer (*Odocoileus virginianus*) are considered the natural definitive host for the meningeal worm *Parelaphostrongylus tenuis* but seldom show signs of infection. *P. tenuis* also infects other animals and there are numerous reports of infection in both domestic<sup>1,9,12,15</sup> and wild ungulates<sup>2-6,8,10,11,13,14</sup> living in close proximity to white-tailed deer. For some unknown reason, *P. tenuis* migrates much more extensively in the central nervous system of incidental definitive hosts, often causing severe, disabling neurologic disease.

This report will describe the clinical and pathologic findings seen in association with parelaphostrongylosis in two llamas (*Lama guanicoe*) from south-eastern Texas.

### CASE HISTORY

A herd of 11 llamas was being maintained on a private ranch 100 km west of Houston, Texas. These animals had been procured from a game ranch located in the Adirondack Mountains in upstate New York and moved to Texas a few months prior to the onset of clinical signs. Native white-tailed deer had free access to the grounds where the llamas were kept. In one year, 4 of the 11 llamas at the Texas ranch died after showing signs of a neurologic disorder. The signs included head tilting, arching of the neck, incoordination, difficulty in getting up and a gradual loss of weight over a period of several weeks. Three of the animals became so debilitated that they died or were humanely killed. The fourth llama was repeatedly treated for parasites with levamisole<sup>▣</sup> and diethyl-carbamazine<sup>▣</sup> and also administered

<sup>□</sup> Present Address: National Animal Disease Center, Science and Education Administration, U.S. Department of Agriculture, Ames, Iowa 50010, USA.

<sup>▣</sup> Present Address: Department of Clinical Sciences, School of Veterinary Medicine, University of California, Davis, California 95616, USA.

<sup>▣</sup> Tramisol, American Cyanamid, Princeton, New Jersey 08540, USA.

<sup>▣</sup> Caricide, American Cyanamid, Princeton, New Jersey 08540, USA.

large doses of dexamethasone<sup>□</sup> and vitamin B complex. Following treatment, the llama started gaining weight and the neurologic deficits diminished until they were minimal. The llama died unexpectedly without recrudescence of clinical signs.

#### MATERIALS AND METHODS

Necropsies were performed on 2 of the 4 llamas. Significant gross lesions, other than those of cachexia, were not seen. Tissues were fixed in 10% buffered neutral formalin. The brain from the llama that was debilitated before death was submitted to the Texas A&M Veterinary Medical Diagnostic Laboratory in College Station, Texas. The brain and sections of the spinal cord, heart, lungs, liver, kidney, ovary and uterus from the llama that died suddenly were sent to Oklahoma State University. Tissues were embedded in paraffin, sectioned at 6  $\mu$ m and stained with hematoxylin and eosin.

#### RESULTS

In multiple sections of brain examined at the Diagnostic Laboratory, scattered foci of hemorrhagic necrosis were seen.

In the tissues examined at Oklahoma State University, significant microscopic lesions were found only in the central nervous system. Randomly scattered focal areas of necrosis were present in the brain and spinal cord. The necrotic foci were small to medium in size, irregularly circumscribed and appeared to be of varying ages. The more acute lesions were characterized by focal areas of parenchymal loss with hemorrhage in and around the area of injury. In older lesions, hemorrhage was not evident but varying numbers of large, foamy macrophages were present. In some places, the macrophages contained a golden brown pigment interpreted as

hemosiderin. Swollen, eosinophilic axons were seen around some necrotic foci. An adult female nematode was found in an area of hemorrhagic necrosis just beneath the meningeal surface of the ventral thalamus (Fig. 1). The intestinal cells of the nematode were large and multinucleated, polymyarian coelomyarian muscle cells were identified and the uterus contained eggs typical of protostrongylids (Fig. 2).

#### DISCUSSION

The brain lesions seen by individuals in the Texas Diagnostic Laboratory were interpreted as being most compatible with lesions caused by a migrating parasite. Similar lesions were seen in the brain and spinal cord of the other llama plus an adult nematode having features of *P. tenuis*<sup>7</sup> in one area of brain necrosis. These findings and the clinical signs seen in the four sick llamas are similar to what has been described in other incidental hosts of *P. tenuis*.<sup>1-6,8-15</sup> Therefore, we concluded that the neurologic disorder in these llamas was caused by *P. tenuis*. The importance of these findings is not so much that this is the first report of parelaphostrongylosis (none could be found in the literature) in a llama as it is the occurrence of the disease in an exotic animal found in many zoological parks.

For many years, it has been recognized that where the ranges of white-tailed deer and other cervids overlap, cervids other than white-tailed deer often have a difficult time surviving because of infection with *P. tenuis*.<sup>2,4,11,13,14</sup> For the same reason, it is very difficult to transplant many cervids to ranges populated by white-tailed deer.<sup>3,4,6,10</sup>

In zoological parks having an exhibit of white-tailed deer or to which white-tailed deer have access, a potential disease problem may exist in exotic cervids and other hosts, such as the llama, which

<sup>□</sup> Azium, Schering Corp., Kenilworth, New Jersey 07033, USA.

are susceptible to *P. tenuis*. In such situations, precautions should be taken to prevent parelaphostrongylosis in these potential hosts.



FIGURE 1. An adult, female meningeal worm situated in a hemorrhagic necrotic focus in the ventral thalamus. A hemorrhagic tract of necrosis (arrow) leads to the area where the parasite is situated. M = leptomeninges. H&E.



FIGURE 2. Cross-section of meningeal worm in which large, multinucleated intestinal cells (i), polymyarian coelomyarian muscle cells (arrow) and a uterus containing eggs (u) can be identified. H&E.

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