Acquired cervical scoliosis attributed to *Parelapthostrongylus tenuis* infection in an alpaca

Amy L. Johnson, DVM; Catherine G. Lamm, DVM; Thomas J. Divers, DVM, DACVIM

**Case Description**—A 2-year-old alpaca was evaluated because of acute onset of cervical scoliosis.

**Clinical Findings**—Physical examination revealed severe scoliosis of the caudal portion of the cervical vertebral column with a C-shaped curvature to the right side. No gait deficits were observed. Cervical radiography confirmed severe curvature of C4 to C6 but did not reveal any bony changes. Cerebrospinal fluid had high total protein concentration and extremely high nucleated cell count with a high proportion of eosinophils, suggesting parasitic infection.

**Treatment and Outcome**—The alpaca was treated for suspected parelapthostrongylasis with ivermectin, fenbendazole, flunixin, vitamin E, thiamine, physical therapy, and a custom-made neck brace. The alpaca's condition continued to deteriorate, and it developed tetraparesis and ataxia and was euthanized after approximately 1 month. Microscopic evaluation of the cervical spinal cord revealed marked vascular changes in the left medial portion of the ventral funiculus, mild lymphoplasmacytic infiltration, and multifocal granulomas. The lesions were continuous from C1 to C7 and were compatible with parasite migration.

**Clinical Relevance**—To the authors’ knowledge, this is the first report of acquired scoliosis in an alpaca, which appears to represent an unusual manifestation of parapleathostrongylasis that was reported in horses. (J Am Vet Med Assoc 2006;229:562–565)

A 2-year-old 56-kg (123.2-lb) female alpaca in the sixth month of gestation was evaluated because of acute onset of cervical scoliosis. The farm manager first noticed a slight curvature of the cervical vertebrae approximately 12 days prior to evaluation. No swelling or signs of pain were observed. The curvature worsened over several days despite treatment with warm compresses, flunixin meglumine, vitamin E, selenium, and methocarbamol (dosages unknown). The curvature then remained relatively static during the week prior to evaluation.

Physical examination revealed that the alpaca was alert and ambulatory with appropriate mentation. Severe caudal cervical scoliosis involving C4 to C6 was evident and formed a C-shaped curvature with the concavity to the right side. Manual pressure failed to substantially reduce the curvature. No muscle atrophy was detected; however, the cervical muscles on the left (convex) side were flaccid, and the cervical muscles on the right (concave) side were tense. Cutaneous sensation of the cervical region appeared normal but was difficult to assess because of the typical stoic nature of alpacas. The funicular portion of the nuchal ligament was taut and firm, forming a straight line from C2 to T2. The alpaca was able but reluctant to lift its head and neck above a horizontal plane and spent more time recumbent than a companion alpaca. No gait deficits were detected.

Cervical radiography confirmed severe curvature of C4 to C6. No bony changes were seen. Aspiration of lumbosacral CSF yielded pink, slightly cloudy fluid. Cytologic analysis revealed very high nucleated cell count (2,676 cells/μL) with 76% eosinophils, 12% lymphoid cells (including reactive and plasmacytoid forms), 9% macrophages (many were vacuolated or had phagocytized eosinophils), and 3% neutrophils. The RBC count (7,289 cells/μL) and total protein concentration (310 mg/dL) were also high. This evidence of eosinophilic inflammation was strongly suggestive of parasitic infection, presumptively attributable to *Parelapthostrongylus tenuis*.

Treatment for parasitic migration through the CNS was initiated with ivermectin (0.2 mg/kg [0.09 mg/lb], SC, q 24 h), fenbendazole (50 mg/kg [22.7 mg/lb], PO, q 24 h), flunixin meglumine (1 mg/kg [0.45 mg/lb], IV, q 12 h), vitamin E (35 U/kg [15.9 U/lb], PO, q 24 h), and thiamine (10 mg/kg [4.5 mg/lb], SC, q 24 h). Omeprazole (5 mg/kg [2.3 mg/lb], PO, q 24 h) and ceftiofur (2 mg/kg [0.91 mg/lb], IV, q 12 h) were administered prophylactically. Administration of flunixin and ceftiofur was discontinued on day 10; administration of ivermectin, fenbendazole, and thiamine was discontinued on day 14; and administration of vitamin E was discontinued on day 15. Physical therapy, consisting of 5 to 10 minutes of manual pressure to reduce the degree of curvature, was performed 4 to 8 times/d. After several days of medical and physical therapy, the curvature became more yielding to pressure and could be partially straightened, but the neck returned to its original position when pressure was removed. A specialist in prosthetic devices designed a custom-made, bivalved neck brace to provide gentle continuous pressure to straighten the neck. To create a neck mold, the alpaca was sedated with midazolam (0.1 mg/kg [0.05 mg/lb], IV) and butorphanol (0.05 mg/kg [0.02 mg/lb], IV). This sedation substantially increased the pliability of the alpaca’s neck, allowing it to be positioned in a nearly straight position with minimal pressure.

The alpaca remained in sternal or lateral recumbency when the brace was in place and would not eat or drink. The brace improved cervical flexibility in the
cranial to midcervical region but did not affect the caudal cervical region. The brace was left in place for approximately 2 hours at a time, 2 to 3 times/d. The alpaca was discharged on day 16 for continued physical therapy at home. Over the next 4 weeks, the scoliosis progressed and the alpaca became paretic and ataxic. Euthanasia was performed because of humane concerns.

A complete postmortem examination was performed. On external examination, there were 2 lateral curvatures in the cervical vertebral column. The less severe of the 2 curves extended from C2 to C3 with the concavity on the left. Extending from C3 to C7, the second curvature was more severe, with the concavity to the right. The spinal cord was removed by dissection of each cervical vertebra; no gross abnormalities were detected in the spinal cord. After the musculature was entirely removed by boiling, the cervical vertebrae were examined and were grossly within normal limits.

Representative specimens of the brain and spinal cord were fixed in neutral-buffered 10% formalin, routinely processed for paraffin embedding, and stained with H&E. In cross sections of the cervical spinal cord and in the left medial ventral funiculus, there was marked, radiating vacuolar change with collapse of the normal parenchyma, spheroids, and multifocal areas of fibrosis that replaced the normal architecture. Concentrated in the areas of fibrosis were mild multifocal infiltrates of lymphocytes and plasma cells. Changes in the ventral portion of the spinal cord were detected in sections from C1 to C7, although they were most pronounced in C5 to C7. On longitudinal section and within the ventral portion of the spinal cord, there was marked Wallerian degeneration characterized by numerous linearly arranged clear vacuoles containing small numbers of macrophages (myelin digestion chambers) that obscured the normal axonal architecture. Selected segments of the spinal cord were stained with Luxol fast blue, which highlighted areas of myelin loss. Within the dorsal lateral funiculus of C1 to C4, there was very mild, radiating vacuolar change. Randomly scattered within the left side of the cervical spinal cord were variably sized, discrete granulomas characterized by central aggregates of mineral surrounded by a rim of macrophages and plump reactive fibroblasts. Sections of the brain and spinal cord caudal to C7 were histologically normal.

Neurologic disease caused by aberrant migration of *P. tenuis* through the CNS has been reported in several domestic species, including sheep, goats, cattle, llamas, and horses. The life cycle of this parasite involves ingestion of third-stage larvae by white-tailed deer, larval migration from the gastrointestinal system to the CNS, reproduction in the subarachnoid space with egg deposition into the venous system, egg migration to the pulmonary vasculature, and embryonation into first-stage larvae in the lungs. The first-stage larvae undergo tracheal migration and are coughed up, swallowed, and passed in feces. The larvae infect gastropods and mature to third-stage larvae. Deer and aberrant hosts are infected by *P. tenuis* via ingestion of infected gastropods or the infected slime that the gastropods secrete; although deer have few clinical signs, aberrant hosts most commonly have severe neurologic deficits progressing from pelvic limb paresis and ataxia to tetraparesis and sometimes paralysis. Llamas appear particularly susceptible to infection and often have severe clinical signs with very few parasites.

Although spinal cord signs predominate, other clinical manifestations of *P. tenuis* infection have been reported. Recently, 6 cases of acquired cervical scoliosis in horses were linked to *P. tenuis* migration through the dorsal gray column of the cervical spinal cord. To the authors’ knowledge, no cases of scoliosis in South American camelids have been published in peer-reviewed literature. Discussion on popular Internet sites and among clinicians usually implicates unobserved cervical trauma and subluxation as the likely cause of acquired scoliosis. However, this case of acquired cervical scoliosis had no convincing historical or radiographic evidence of cervical trauma. Cerebrospinal fluid cytologic findings and postmortem spinal cord lesions strongly implicated parasitic migration as the cause.

A case record search from 1975 to 2005 at our hospital revealed only 3 other cases of scoliosis in camelids. In one of those cases, *P. tenuis* was implicated as the cause of scoliosis; the affected 2-year-old llama was initially mildly tetraparetic and ataxic and was treated for aberrant parasite migration. No improvement was observed; the ataxia worsened, and severe cervical scoliosis developed acutely. The llama was referred to our hospital approximately 7 weeks after initial onset of signs. Cervical fracture and subluxation were suspected, and surgery was performed. A cerebellomedullary cistern CSF sample was obtained under general anesthesia; cytologic examination revealed no abnormalities of total protein concentration and cell counts but did reveal abnormal proportions of cells (few eosinophils and very few neutrophils and lymphocytes). The llama died from postoperative complications, and a complete postmortem examination was performed. On gross examination, there was lateral curvature of the cervical spinal column at the level of C3 to C5 with concavity to the left side. No gross abnormalities were detected in the cervical spinal cord or vertebrae. In cross sections of the cervical spinal cord segments and within the ventral medial funiculus, there was mild to moderate unilateral vacuolar change with scattered spheroids, which was most pronounced within the caudal segments. Rarely, there were small areas of collapse with fibrosis. Similar, though much milder, vacuolar changes were evident unilaterally within the dorsal lateral funiculus of C1 through C5. Within a single, unlabeled section of cervical spinal cord, there was very mild vacuolar change and collapse of the parenchyma within the medial aspect of 1 dorsal funiculus. Whether the right or left side of the spinal cord was affected could not be determined from the original report or from the histologic sections reviewed for this report. However, when changes were evident in the dorsal and ventral portions of the spinal cord in the same histologic section, they were located in the same side of the spinal cord.
Sections of the spinal cord caudal to C7 were histologically normal. Infection with *P tenuis* was the presumptive diagnosis made on the basis of eosinophils in the CSF and the histopathologic lesions. Although most cases of clinical paralapathostrongylosis are associated with high nucleated cell counts with eosinophilia in the CSF, the extended time period (7 weeks) between the onset of the llama’s clinical signs and collection of CSF, combined with previous treatment for aberrant parasite migration, likely accounted for the normal nucleated cell count and protein concentration in the llama’s CSF.

It is less clear whether *P tenuis* caused the other 2 cases of scoliosis because complete diagnostic testing and full necropsies were not performed. One affected alpaca was immediately euthanized, and mild vertebral malformations of C3 and C4 were evident at postmortem. A postmortem CSF sample had nonsuppurative inflammation with no eosinophils. The acute onset of scoliosis could not be explained by the mild, likely congenital vertebral malformations, but evidence of parasite migration was not seen microscopically in the spinal cord. Unfortunately, only a few sections of spinal cord were examined, and no explanation for the scoliosis was found. In the other case, the only diagnostic tests performed were cervical ultrasonography and radiology, which did not reveal any abnormalities. The alpaca had a milder case of scoliosis and was discharged from the hospital; a CSF sample was not obtained.

Acquired cervical scoliosis with or without paresis and ataxia is an unusual manifestation of *P tenuis* infection in horses and camelids. In the 6 horses with acquired cervical scoliosis, a continuous inflammatory lesion in the dorsal gray column of the spinal cord was detected. The proposed pathogenesis of scoliosis involved weakness of the paraspinal epaxial muscles on the convex side of the vertebrae thought to result from unilateral loss of proprioceptive innervation via interruption of the general proprioceptive afferent neurons originating from sensory receptors in muscles, ligaments, and joints. These afferents enter the spinal cord via dorsal roots and either ascend to the medulla or synapse in the dorsal gray column. On the basis of a previous case report, we expected to find lesions in the dorsal gray columns of the 2 camelids with scoliosis that underwent full necropsies. However, the changes within the ventral funiculi (white matter) were more prominent in both cases. The cell bodies of general somatic efferent neurons (lower motor neurons) are in the ventral gray column; their axons travel out through the white matter into the ventral nerve root to muscles before branching and terminating on a muscle cell at a motor end plate. We believe that disruption of the general somatic efferent neurons in the ventral white matter led to unilateral denervation of the paraspinal epaxial muscles. The subsequent muscular imbalance caused acute, acquired scoliosis of the cervical vertebrae, similar to what is observed with facial paralysis in horses, where the nose is deviated toward the unaffected side.

Denervation atrophy of the affected cervical muscles would be an expected result within 1 week of the initial damage but was not detected via physical examination; the elongated, thin necks of camelid species do not have prominent musculature, which may explain why atrophy was not detected even if it were present. Histologic examination of muscle was, unfortunately, not performed. A secondary characteristic of the equine cases was cutaneous hypalgesia or analgesia on the convex side of the neck attributed to disruption of the nociceptive pathway in the dorsal gray column. Cutaneous hypalgesia was not detected in the camelid cases likely because the sensory afferent pathway does not pass through the ventral white matter.

Muscular imbalance is the first step in the proposed pathogenesis of acquired scoliosis. Lack of motion in the involved cervical vertebrae causes the joint capsules of the articular processes to remodel and adhere in the scoliotic position, preventing movement. Fibrosis of the joint capsules and the associated muscles may also play a role, but this has not been determined histologically. Early in the disease process, while the alpaca was heavily sedated, the vertebral column could be manipulated into a straight position. However, 1.5 months later when the alpaca was euthanized, the vertebral column could not be straightened, likely because of immobility of the synovial joints. Such secondary changes are likely to impede correction of the scoliosis even if the treatment protocol successfully kills migrating *P tenuis* larvae; therefore, a poor prognosis is warranted. This conclusion is supported by findings in the alpaca and llama because larvae were not detected in the spinal cord at necropsy despite continued clinical deterioration.

The definitive diagnosis of *P tenuis* infection requires identification and speciation of larvae detected microscopically in the spinal cord. Despite careful sectioning of the spinal cord in the alpaca and llama, no larvae were observed. Certainly it is possible that larvae were present but missed; however, the long duration of treatment in both cases supports successful killing of the larvae, either through anthelmintic treatment or spontaneous recovery. The prevalence of *P tenuis* infection in the geographic area where the alpaca was kept, CSF eosinophilia, and typical appearance of histopathologic changes in the spinal cord strongly support a diagnosis of *P tenuis* infection in the alpaca with scoliosis. Histologic examination of muscle was, unfortunately, not performed; therefore, a definitive diagnosis was not possible. In the llama the parasite migration was not seen microscopically in the spinal cord. Despite careful sectioning of the spinal cord, no evidence of parasite migration was not seen microscopically in the spinal cord. Therefore, the paralysis may not have been attributable to *P tenuis* infection.

### References


---

Selected abstract for JAVMA readers from the American Journal of Veterinary Research

Serum concentrations of calcium, phosphorus, magnesium, and calcitropic hormones in donkeys

Ignacio Lopez et al

**Objective**—To provide reference values for serum biochemical variables that are used for evaluation of mineral metabolism in donkeys and compare values with those in horses.

**Animals**—18 donkeys and 18 horses.

**Procedures**—Total calcium (tCa), total magnesium (tMg), and inorganic phosphorus (P) concentrations were measured in serum samples via spectrophotometry. Ionized calcium (iCa) and magnesium (iMg) concentrations were quantified with selective electrodes. By use of a micropartition system, tCa and tMg were fractionated to separate protein-bound (pCa, pMg) and ultrafiltrable fractions. Complexed calcium (cCa) and magnesium (cMg) concentrations were calculated by subtracting ionized fractions from ultrafiltrable fractions. Parathyroid hormone (PTH) and calcitriol (CTR) concentrations were measured via radioimmunoassay.

**Results**—Serum iCa concentration in donkeys (3.37 ± 0.21 mmol/L) was composed of pCa (1.50 ± 0.21 mmol/L [47.0 ± 4.2%]), iCa (1.69 ± 0.04 mmol/L [50.4 ± 3.0%]), and cCa (0.09 ± 0.08 mmol/L [2.6 ± 2.9%]). Serum tMg concentration (1.00 ± 0.08 mmol/L) was fractioned in pMg (0.23 ± 0.08 mmol/L [23.4 ± 8.1%]), iMg (0.59 ± 0.04 mmol/L [58.8 ± 5.1%]), and cMg (0.18 ± 0.08 mmol/L [17.8 ± 7.2%]). Serum concentrations of P (1.14 ± 0.30 mmol/L), PTH (20.4 ± 21.2 pg/mL), and CTR (13.4 ± 5.9 pg/mL) were determined.

**Conclusions and Clinical Relevance**—Serum variables of mineral metabolism in donkeys were within reference ranges for horses. However, compared with horses, donkeys had higher iCa, cMg, and CTR and lower pMg and PTH concentrations. *(Am J Vet Res 2006;67:1333–1336)*