Acquired Scoliosis in Equids: Case Series and Proposed Pathogenesis

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Acquired scoliosis in equids is uncommon. Potential causes such as trauma, neoplasia, abscessation, and protozoal myelitis can be excluded with historical information, physical examination, imaging, and serology or cerebrospinal fluid analysis. In the absence of an alternative explanation, equine veterinarians should be highly suspicious of *Parelaphostrongylus tenuis* infection. Treatment is unlikely to be successful, and a poor prognosis for improvement is warranted. Authors’ addresses: Department of Clinical Studies, New Bolton Center, School of Veterinary Medicine, University of Pennsylvania, 382 West Street Road, Kennett Square, PA 19348 (Johnson); 7 River Woods Drive, Apt. P-115, Exeter, NH 03833 (de Lahunta); and Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853 (Divers); e-mail: aljdvm03@gmail.com. © 2008 AAEP.

1. Introduction
With few exceptions, virtually all reported cases of scoliosis in horses have been attributed to congenital vertebral malformations, and many have been hypothesized to result from intrauterine malposition. Recently, six equine cases of acquired cervical scoliosis were described as a result of lesions consistent with parasite migration, and a parasite resembling *Parelaphostrongylus tenuis* was seen in one of the lesions. Since the publication of that report, three additional equids with acquired scoliosis have been evaluated at the Cornell University Hospital for Animals. These cases, in combination with those previously detailed, are unique in that the equids exhibited acquired scoliosis in the absence of trauma or congenital malformation. The following report summarizes the clinical findings for five of the equids and discusses the proposed pathogenesis of the scoliosis.

2. Materials and Methods
Over a 5-yr time period (2002–2007), five equine cases of acquired scoliosis were presented to the Cornell University Hospital for Animals. All equids were housed on farms in New York state. Two of these cases were previously described and underwent post-mortem examination, whereas the other three were discharged from the hospital after presumptive diagnosis.

All patients received careful physical and neurologic examinations, and particular attention was focused on palpation of the scoliotic region, assessment of cutaneous sensation, and evaluation of gait. Cervical and/or thoracic radiography was performed in all cases to look for evidence of trauma or malformation. Cerebrospinal fluid collection and analysis was performed in three of five horses, and four of five horses were tested for *Sarcocystis neurona* using CSF (3 of 4) or serum (1 of 4). Cervical ultrasonography was per-
formed in one case, and computed tomography was performed in one case.

3. Results

Table 1 summarizes the results from the five equids. Four cases were horses (one mixed breed, one Thoroughbred, one half-Arabian, and one Morgan) ranging in age from 6 mo to 9 yr, and one case was a 6-yr-old miniature donkey. All cases presented with a history of sudden development of scoliosis. Although the onset of scoliosis ranged from 5 days to 2 mo before presentation, the owners described the scoliosis as developing very quickly, often overnight. The four horses displayed cervical scoliosis (Fig. 1), whereas the donkey displayed mid-thoracic scoliosis (Fig. 2). History or evidence of trauma was not present in any case. After the scoliosis was apparent, it did not seem to progress, but progressive gait deficits were observed in the horse that survived for >2 yr post-discharge.

The scoliotic regions were easily noted on physical examination, and in all cases, the affected area of the vertebral column was C-shaped with flaccid muscles on the convex side and tense muscles on the concave side. Cutaneous sensation was decreased or absent over the convex side of the curve but present cranial and caudal to the affected area as well as over the entire concave side. All cases with cervical scoliosis displayed mild to moderate hemi- or tetraparesis and ataxia consistent with cervical myelopathy. If present bilaterally, gait deficits were significantly more pronounced ipsilateral to the convex side of the curve. The donkey with mid-thoracic scoliosis had no apparent gait deficits, but only a limited exam was performed because of the donkey’s resistance to being led.

Cerebrospinal fluid (CSF) was obtained by atlanto-occipital puncture in one case and by lumbosacral puncture in two cases. Nucleated cell counts and total protein were within normal reference ranges in all three cases, and no cytologic abnormalities were detected. All nucleated cells observed on the preparations were mononuclear; no eosinophils were noted. CSF samples were submitted for S. neurona Western blot tests; all three were negative. One horse from which CSF was not obtained had serum submitted for S. neurona Western blot testing with a weak positive result.

Radiographs of the affected region were performed in all cases. Although lateral curvature of the vertebral canal was evident in each case, no fractures or other bony abnormalities consistent with either trauma or congenital malformation were observed. The 6-mo-old half-Arabian filly returned for recheck examination at 15 mo of age, and radiographs were repeated; on the second set of radiographs, mild-moderate degenerative joint disease was evident at C2-C3 and C3-C4 (Fig. 3).

Cervical ultrasonography was performed in one case (9-yr-old Morgan gelding) that developed scoliosis shortly after discharge from the hospital for medical management of an episode of colic. The convex side of the curve was on the same side of the neck in which a short-term IV catheter had been placed. Catheter placement was routine, and no extravasation of fluids or drugs was noted during the period of hospitalization. Ultrasonography revealed a normal, patent jugular vein with no abnor-

### Table 1. Characteristics of 5 Equine Cases of Acquired Scoliosis Presented to the Cornell University Hospital for Animals

<table>
<thead>
<tr>
<th>Signalment</th>
<th>Affected Region</th>
<th>Radiographs</th>
<th>CSF Analysis</th>
<th>EPM Testing</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-yr-old mixed breed gelding*</td>
<td>C3–C6; concavity on left</td>
<td>No bony abnormalities</td>
<td>NCC 1/µl; RBC 15/µl; TP 63 mg/dL</td>
<td>CSF WB negative</td>
<td>Ivermectin, fenbendazole, flunixin, vitamin E, vitamin C</td>
<td>Euthanized; necropsy</td>
</tr>
<tr>
<td>10-mo-old TB colt*</td>
<td>C5–C6; concavity on left</td>
<td>No bony abnormalities</td>
<td>NCC 0/µl; RBC 0/µl; TP 46 mg/dL</td>
<td>CSF WB negative</td>
<td>Ivermectin, fenbendazole, flunixin, DMSO, thiamine</td>
<td>Euthanized; necropsy</td>
</tr>
<tr>
<td>6-mo-old half-Arab filly</td>
<td>C2–C5; concavity on left</td>
<td>No bony abnormalities initially</td>
<td>NCC 2/µl; RBC 62/µl; TP 47 mg/dL</td>
<td>CSF WB negative</td>
<td>Ivermectin, fenbendazole, phenylbutazone</td>
<td>Discharged; still alive 2.5 yr later but no improvement</td>
</tr>
<tr>
<td>9-yr-old Morgan gelding</td>
<td>C3–C7; concavity on right</td>
<td>No bony abnormalities</td>
<td>Not done</td>
<td>Serum WB weak positive</td>
<td>Ivermectin, fenbendazole, flunixin, vitamin E</td>
<td>Discharged; euthanized 2 wk later; no necropsy</td>
</tr>
<tr>
<td>6-yr-old miniature donkey jenny</td>
<td>Mid-thoracic</td>
<td>No bony abnormalities</td>
<td>Not done</td>
<td>Not done</td>
<td>Ivermectin, fenbendazole</td>
<td>Discharged</td>
</tr>
</tbody>
</table>

malities within or adjacent to the jugular groove or vertebral column. Therefore, the history of catheterization was deemed incidental.

Computed tomography (CT) was performed in one case (10-mo-old Thoroughbred colt) to further investigate the cause of scoliosis. Although CT confirmed a combination of lateral deviation and mild rotation of C5 and C6, no cause for the scoliosis was evident. Despite IV contrast injection, the definition of the spinal cord was not sufficient to recognize any intramedullary lesions.

Post-mortem findings from two of the horses have been described previously. In brief, the 3-yr-old mixed-breed gelding with involvement of C3-C6 (concavity on left) had continuous discoloration of the right dorsal grey column from C3-C6 that correlated microscopically to severe inflammation with extensive necrosis involving the right dorsal grey column, intramedullary dorsal nerve roots, and adjacent white matter. Several sections of a nematode presumed to be a larvae of the Metastrongyloidea were present in the lesion at the C4 segment. The 10-mo-old Thoroughbred colt with involvement of C5-C6 (concavity on left) had a similar inflammatory, necrotizing lesion in the right dorsal grey column, intramedullary dorsal nerve roots, and adjacent white matter from caudal C5-T1. A focal accumulation of eosinophils

Fig. 1. Photographs of a 6-mo-old half-Arabian filly with acquired cervical scoliosis. (A) Dorsal cervical view showing a C-shaped scoliotic region. (B) Right lateral cervical view showing the convex side of curve. (C) Left lateral cervical view showing the concave side of curve.

Fig. 2. Photograph of a 6-yr-old miniature donkey with acquired mid-thoracic scoliosis.
was present where the dura mater surrounded the T1 spinal nerve.

4. Discussion

All of the equine cases of acquired scoliosis have displayed similar clinical signs beginning with sudden development of C-shaped scoliosis. Pertinent findings include flaccid muscles on the convex side, tense muscles on the concave side, hypalgesia over the convex side, and gait deficits ipsilateral to or more severe on the convex side. No history or evidence of trauma was present in any case. Using neuroanatomical principles, the clinical signs and development of scoliosis can be explained.

The lack of bony abnormalities, combined with the differing muscle tone on either side of the scoliotic region, suggests a neuromuscular cause for the scoliosis. Normal muscle tone requires the integrity of both general proprioceptive afferent neurons and general somatic efferent neurons. The general proprioceptive neurons originate from sensory receptors in muscles, ligaments, and joints, enter the spinal cord through dorsal nerve roots, and either ascend to the medulla or synapse in the dorsal grey column. The general somatic efferent neurons have cell bodies in the ventral grey column and axons that travel through the ventral nerve roots to synapse at motor end plates in the paraspinal epaxial muscles. The proposed pathogenesis of acute development of scoliosis involves unilateral weakness of the paraspinal epaxial muscles on the convex side of the curve. For significant unilateral weakness to occur, the proprioceptive afferent or somatic efferent innervation must be interrupted over several spinal-cord segments. A linear lesion that extends for several segments in the dorsal grey column, the ventral grey column, or the white matter near the nerve roots is most likely to cause the type of damage that would result in unilateral weakness. However, it is plausible that damage to multiple dorsal or ventral nerve roots in adjacent segments could cause similar unilateral muscle weakness. In either instance, the acute muscular imbalance results in sudden acquired scoliosis, similar to what is observed with facial paralysis in horses, where the nose is deviated toward the unaffected side.

All of the equine cases of acquired scoliosis have displayed clinical signs more compatible with a dorsal grey column and/or white matter lesion than with a ventral lesion. Lesions in the dorsal grey column and adjacent white matter would be expected to cause denervation atrophy of the cervical musculature but not hypalgesia. All of the equine cases displayed cutaneous hypalgesia over the convex side of the scoliotic region with no evidence of denervation atrophy. Although post-mortem examinations were not performed on the three new cases of acquired scoliosis described in this report, the six previously described cases all had a continuous unilateral dorsal grey column spinal cord lesion that extended over several segments and also into adjacent white matter and intramedullary dorsal nerve roots.

Although muscular imbalance is the first step in the proposed pathogenesis of acquired scoliosis, secondary changes within the vertebral column may contribute to the pathology. Lack of motion causes remodeling of the joint capsules of the articular processes, which may become “fixed” in the scoliotic position and prevent movement. Although not proven histologically, fibrosis in the joint capsules and associated muscles or osteoarthritic changes of the cervical vertebrae may also play a role. New bone formation or hypertrophy of soft tissue structure may impinge on the spinal cord, leading to progressive neurologic deficits. Osteoarthritic changes were seen in the only case for which long-term follow-up was available (Fig. 3). The neck can be easily straightened by the examiner early in the disease process, and the horse shows minimal pain on cervical manipulation. As osteoarthritic changes develop, straightening the neck becomes more difficult, and pain becomes more noticeable.

Post-mortem examination of the spinal cord of one of the horses revealed a nematode consistent with P. tenuis, and the other five cases that underwent post-mortem examination had nearly identical lesions that were located in the dorsal grey column and extended for at least three spinal-cord segments, consistent with parasite migration. Further evidence that P. tenuis can infect horses was provided in a recent case report in which nematodes of the family Protostrongylidae were found in the central nervous system of a 6-mo-old Arabian colt with neurologic deficits. Neurologic disease caused by aberrant migration of P. tenuis through the central nervous system of a 6-mo-old Arabian colt with neurologic deficits.
nervous system is well recognized in small ruminants and camelids. In these species, the most common clinical presentations are paraparesis and ataxia that may progress to tetraparesis and ataxia. However, acute development of cervical scoliosis has been described in an alpaca infected with *P. tenuis*. Species sensitivity to *P. tenuis* infection is variable, and llamas are considered to be the most susceptible. Horses have only recently been recognized as susceptible to infection, and based on these findings, they seem to have a relatively consistent yet unusual clinical presentation.

Although treatment with anthelmintics and anti-inflammatory medication was attempted initially in all cases, none of the equids improved. One case is still alive 3 yr after the acute onset of signs, but despite medical and physical therapy, the scoliosis has not improved. Progression of neurologic deficits was seen in all cases for which short- or long-term follow-up information was available, likely because of secondary osteoarthritic changes of the vertebral column. Even if the treatment protocol successfully kills migrating *P. tenuis* larvae, the severe necrosis of the dorsal grey matter and the secondary changes are likely to impede correction of the scoliosis. Therefore, recovery is not expected, and a poor prognosis is warranted, although affected horses may survive for months to years.

Other disease processes that could cause a similar clinical presentation include trauma, ischemic myelopathy, equine protozoal myeloencephalitis, aberrant migration of other parasites, neoplasia, and an inflammatory process such as a drug reaction or abscess. Many of these problems (trauma, paraspinous neoplasia, and paraspinous inflammatory processes) can be excluded with historical information, palpation, ultrasonography, radiography, and/or CT. Negative serologic fluid or CSF results for *S. neurona* and *N. hughesii* are useful in excluding protozoal myeloencephalitis, but positive results are not definitive. Therefore, if serologic and/or cerebrospinal results return positive for *S. neurona* or *N. hughesii*, treatment with an anti-protozoal medication may be warranted. Although small ruminants and camelids with *P. tenuis* myelitis tend to have CSF eosinophilia, none of the equids for which CSF analysis was performed had eosinophils noted.

One of the limitations of this study was the lack of a definitive diagnosis for the three animals that were discharged alive. As is typical of many neurologic diseases of horses, definitive diagnosis requires postmortem examination. In these cases, presumptive diagnosis was based on thorough neurologic examination, lack of bony abnormalities on radiographs, and negative results for *S. neurona*. Vertebral radiography confirmed the presence of scoliosis noted clinically and was valuable in excluding vertebral malformations or traumatic injury as the cause.

Additional diagnostic tests frequently used for neurologic problems include electromyography, CT, and magnetic resonance imaging. Electromyography was not performed in any of the five cases in this series but was performed and showed no denervation potentials in one of the previously reported cases. Based on the lack of significant atrophy and the post-mortem lesions observed, none of the affected equids had evidence of lower motor neuron involvement, and electromyography probably would not aid in diagnosis. CT was performed in one case (10-mo-old Thoroughbred colt) and was useful in excluding vertebral malformations, fractures, and luxations. However, the definition of the spinal cord was not sufficient to recognize any soft-tissue lesions, despite the significant lesions observed on post-mortem examination. Magnetic resonance imaging yields superior resolution of soft-tissue structures compared with CT and has been used in the equid to support a diagnosis of equine protozoal myeloencephalitis. However, equipment limitations often preclude use of magnetic resonance imaging to evaluate the spinal cord in equids. A research tool that has not been validated in the horse but may aid in antemortem diagnosis is serology for antibodies against excretory/secretory proteins collected from *P. tenuis* larvae; the 3-yr-old mixed breed gelding was positive, whereas a control horse was negative.

Only seven equine cases of presumptive *P. tenuis* infection have been previously described, and three additional cases have been included in these proceedings. Ninety percent (9 of 10) of these cases displayed acquired scoliosis. Eighty-nine percent (8 of 9) of those cases had cervical scoliosis. Conversations with equine veterinarians confirm that there have been other cases of unexplained acquired scoliosis, possibly caused by *P. tenuis* infection. These cases may have been misdiagnosed as trauma. The authors contend that equine *P. tenuis* infection may be more common than previously thought and that the clinical syndrome is usually similar and easily recognizable as acute development of scoliosis. Although other disease processes may cause scoliosis, many of these diseases can be excluded with historical information and easily available diagnostic tests, such as physical examination, serology, ultrasonography, and radiography. Additional diagnostic tests such as CSF analysis, CT, and magnetic resonance imaging may exclude other potential causes of scoliosis and support the diagnosis of *P. tenuis* infection. Treatment can be attempted but is unlikely to be successful, and a poor prognosis for improvement is warranted.

References