infected with *P. testudinis*, the difference (8/20 versus 2/23) is significant (P < 0.05 by Fisher’s test). *Pasteurella testudinis* may play a more specific role in abscesses of turtles than in the other conditions in which it was encountered, as suggested by its occurrence in pure culture and appreciable numbers (3+ or 4+) that was limited to 6 such cases, apart from 1 instance of apparent bacteremia in a tortoise in which liver and lung yielded similar results. Further, in the 15 cases found in a retrospective search of our laboratory records for microorganisms recovered from abscesses in tortoises and turtles (1984-1989), *P. testudinis* was present on 8 occasions and a variety of organisms were found in the other 7 cases, including *Proteus*, *Clostridium*, *Staphylococcus*, *Streptococcus*, and *Pasteurella* species. No 2 cases lacking *P. testudinis* had any bacterial flora in common. The sparse pertinent literature, although recognizing abscesses of chelonia, particularly those related to the ear, as common problems,2,3,10 points to no single or even predominant etiologic agents. The broad assortment of agents reported includes various members of the family Enterobacteriaceae,1,2,3 but also *Aeromonas* sp., *Pseudomonas* sp.,2,10 *Corynebacterium muriurn*,2,10,11 *Brachamella* (Neisseria) catarrhalsis,2,10 *Streptomyces* sp., and *Pasteurella* sp.2 Most reports antedate the description of *P. testudinis*.

The apparent prominence of *P. testudinis* in turtle abscesses does not necessarily point to a specific pathogenic role of the agent in this condition. As a common commensal, it may simply be in a preferential position to exploit the established chelonian propensity toward middle ear problems, particularly manifest in turtles.2,10

**Sources and manufacturers**

a. Difco Laboratories, Detroit, MI.  
b. Anaerobe Systems, San Jose, CA.

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**References**


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**Spinal cord ischemic necrosis due to fibrocartilaginous embolism in a horse**

I. Carmen Fuentealba, Brad R. Weeks, Michael T. Martin, Joseph R. Joyce, Grant S. Wease.

Fibrocartilaginous embolism is a syndrome of acute spinal cord infarction that has been described in humans,1,2,4,10 dogs,2,4,5,10 and swine.2,4,5,10 Cases have also been described in a cat10 and in 2 horses.2,10 In all species, the clinical signs of fibrocartilaginous embolism are typically very acute, developing over minutes to hours, and they range from ataxia or mild paresis to total upper or lower motor neuron paralysis with anesthesia. Although the precise pathogenesis of this condition is not clear, the hallmark lesion is ischemic necrosis of spinal cord segments, associated with fibrocartilaginous emboli.

An 8-year-old Quarterhorse gelding was presented with an acute onset of grade 5 lameness on the left foreleg and trembling of the other limbs. The horse had been found trapped in a narrow cattle loading chute the previous evening, but there had been no evidence of trauma, and the horse appeared normal after being freed. The following morning the horse would not bear weight on the left forelimb but was bright...
and alert and had a normal appetite. The horse then became ataxic and recumbent within 2 hours.

When examined, the gelding was alert and had normal vital signs. Neurological evaluation showed a loss of superficial and deep pain perception in both forelimbs and in the left lower cervical area. Pelvic limb reflexes appeared normal. The horse could raise his head and upper neck but could not raise himself to sternal position. When physically raised to a sternal position, he rolled back to lateral recumbency.

Results of the neurological examination supported a focal asymmetrical lesion of the brachial plexus or thoracic spinal cord. The differential diagnosis included trauma, protozoal myelitis, and toxic plants. Rabies was also included because it is endemic in the area and because of the rapid progression of signs.

Therapy with intravenous fluids, phenylbutazone, flunixin, DMSO, and calcium was initiated without clinical improvement. Results of the complete blood count, serum chemistry panel, and ionized calcium test were within normal limits. The animal was euthanized 24 hours after the onset of clinical signs and submitted for necropsy.

Postmortem gross lesions were only identified in the spinal cord. Irregular small dark-red hemorrhagic areas, involving both the grey and white matter, were evident in transverse sections of formalin-fixed spinal cord from C6 to T1 (Fig. 1). Histologic examination revealed necrosis of the grey (dorsal and ventral horns) and white matter (dorsal and lateral funiculi) of the spinal cord from C6 to T1 (Fig. 2). The areas of necrosis were associated with focal hemorrhage, spongiosis, and axonal swelling. Pale basophilic, amorphous material (emboli) occluded the lumen of numerous blood vessels in spinal cord segments from the level of the sixth cervical to first thoracic vertebrae. Affected vessels were also seen at the periphery of the infarcted areas and in the meninges. The emboli stained positive for mucopolysaccharides: bright red with periodic acid-Schiff stain, red with mucicarmine stain (Fig. 3), and blue with azocarmine, Alcian blue, and Masson’s trichrome stains and were interpreted as fibrocartilaginous emboli.

Fibrocartilaginous emboli presumably represent extruded intervertebral disc material from the nucleus pulposus. The path of entry of this material into the vascular system is not clearly understood. Arterial, venous, or a combination of both arterial and venous emboli may be present. Direct entrance of fibrocartilage into the spinal artery and its branches during acute disc hemiation has been proposed as a mechanism. Other possible mechanisms are hemiation of disc material through the vertebral end-plate into the marrow cavity of the vertebral body with subsequent retrograde entrance into the vasculature supplying the spinal cord.
extrusion of disk material secondary to chronic fibrous disc degeneration, with entry into the spinal vasculature. Two cases of fibrocartilaginous embolism have been previously described in horses, but unlike the present case, both were associated with degenerative disk disease. One case report is similar in several respects to the case presented here: an 8-year-old Quarterhorse gelding, which suddenly became recumbent, had a focal ischemic infarct of the spinal cord between C6 and C7, with changes compatible with intervertebral disc degeneration and displacement of the nucleus pulposus. Small pieces of fibrocartilage attached to the dura mater over the infarcted segment were observed microscopically and were interpreted as evidence of a direct relationship between embolism and disc degeneration. A similar association was stated in the second case report, which involved an 11-year-old pony presented with a sudden episode of ataxia. Focal areas of malacia were observed at C6-7 with marked degenerative changes in the C6-7 intervertebral disc.

Trauma and/or vigorous exercise may play an important role in the production of fibrocartilaginous embolism. The stress placed on the spine appears to suddenly increase intraabdominal pressure, which contributes to retrograde blood flow in the spinal vasculature. The associated history in this particular case of entrapment in a cattle chute with presumed struggling and the lack of evidence of intervertebral disk degeneration suggests a relationship between physical exertion, fibrocartilaginous embolism, and the development of ischemic necrotizing myelopathy. Therefore, clinicians should include fibrocartilaginous embolism in their list of differential diagnoses in horses with acute severe spinal disease following episodes of casting, capture, or similar violent manipulations.

References

Figure 2. (Top). Photomicrograph of a section of caudal cervical spinal cord (C6) of a horse demonstrating the multifocal pattern of spongiosis and/or hemorrhage involving the dorsal and lateral funiculi and extending into the grey matter. H&E stain. 20 x.

Figure 3. (Bottom). Photomicrograph of a section of caudal cervical spinal cord grey matter (C6) of a horse demonstrating the fibrocartilaginous emboli. 250 x.