Fibrocartilaginous embolic myelopathy in a calf

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Abstract. A 5-month-old Angus heifer with a history of acute hindlimb paresis that quickly progressed to lateral recumbency was necropsied. Gross lesions included a 6-cm segment of gray to brown discoloration and softening of the right ventrolateral spinal cord between T2 and T3. Microscopically, there was liquefactive necrosis of ventrolateral white and gray matter, and multiple intravascular emboli partially or completely occluded many intralesional and adjacent spinal and meningeal arteries and veins. Emboli were alcian blue positive, consistent with fibrocartilage of the nucleus pulposus of the intervertebral disk. No gross abnormalities were detected in the vertebrae or intervertebral disks. Fibrocartilaginous embolic myelopathy appears to be very rare in cattle; however, it should be considered in cases of acute, nonprogressive spinal cord dysfunction.

A 5-month-old Angus heifer calf was presented to the ambulatory service of the Virginia–Maryland Regional College of Veterinary Medicine Veterinary Medical Teaching Hospital (VMTH), Blacksburg, Virginia, with a history of acute hindlimb paresis that quickly progressed to lateral recumbency. The calf was found in lateral recumbency and was unable to rise with assistance. Once positioned, the calf could remain in sternal recumbency. The calf was admitted to the VMTH the following day for complete neurologic examination. On presentation, the calf was tetraparetic to the point of recumbency but was bright, alert, and responsive. Cranial nerve responses were within normal limits. Patellar reflexes were intact and normal bilaterally. Withdrawal responses were normal in the right hindlimb and both forelimbs and decreased in the left hindlimb; however, the heifer had been in left lateral recumbency for at least several hours before neurological evaluation. Tail and anal tone were within normal limits. No localized pain was identified. The panniculus reflex was absent caudal to T3/T4. Cervical radiographs revealed no bony abnormalities.

Cranial nerve responses were normal. The panniculus reflex was absent caudal to T3/T4. Cervical radiographs revealed no bony abnormalities. Cervical spinal fluid analysis from the atlanto-occipital joint space was within normal limits. A thoracolumbar spinal lesion causing tetraparesis was suspected based on neurologic and physical exam findings. Differential diagnoses at the time of exam included spinal trauma and parasite migration (Parelaphostrongylus tenuis, Hypoderma bovis). Ultimately, the calf was euthanized and immediately submitted for routine postmortem examination because of a poor prognosis for quick recovery and return to function.

Necropsy findings included focal areas of subcutaneous edema caudal to the right scapula and over the right iliac wing. Gross examination of the formalin-fixed spinal cord revealed locally extensive softening and gray to brown discoloration of the right lateral and ventral portions of a 6-cm segment of spinal cord between T2 and T3 (Fig. 1). Cranially, the lesion was limited to the right lateral to ventral portions of the cord. The lesion progressed to bilateral involvement of the ventral cord more caudally and finally, involved the right lateral and ventral cord at the most caudal portion. Vertebral and intervertebral disks were grossly normal.

Microscopically, sections of affected cord contained locally extensive necrosis and degeneration of white and gray matter, including portions of the right lateral, right and left ventral funiculi, as well as both ventral horns (Fig. 2). Large
foci of liquefactive necrosis characterized by loss of cell outlines and increased clear space obscured normal white and gray matter tissue architecture. These foci contained multiple areas of hemorrhage and were surrounded by multiple dilated axon sheaths (up to 200 μm in diameter) containing hyperesinophilic, swollen (up to 160 μm in diameter) axons (spheroids); clear space (axon loss/demyelination); granular to fibrillar eosinophilic material; or occasional plump, foamy macrophages (gitter cells) (Fig. 2, inset). Ventral horn neurons were shrunken and hyperesinophilic with karyolysis. Plugs of homogenous lavender to coarsely granular basophilic material partially, and sometimes completely, occluded intralesional and adjacent spinal vessels (Fig. 3), including the ventral spinous artery and its branches, branches of the ventral radicular arteries, ventrolateral meningeal vessels, and ventral spinal veins. Intravascular emboli stained bright blue with alcian blue stain at pH 2.5, consistent with acidic glycosaminoglycans. Von Kossa staining revealed calcification of embolic material in a meningeal artery. Affected vessel walls were replaced by hyperesinophilic fibrillar material and neutrophils (fibrinoid necrosis). The morphologic diagnosis was focally extensive, acute to subacute, severe degeneration and necrosis of spinal cord white and gray matter with multiple arterial and venous fibrocartilaginous emboli and fibrinoid necrosis of vessels. Histologic findings were consistent with a diagnosis of fibrocartilaginous embolic myelopathy.

Although there is general agreement that fibrocartilage of the nucleus pulposus is the source of the embolic material, the pathogenesis of embolic showering of the spinal cord vasculature remains unclear.3-5,12 Several possible mechanisms have been proposed to explain both arterial and venous embolism in the various affected species. One suggested route of passage is dissection of the semi-fluid nucleus pulposus through a degenerating dorsal annulus fibrosis to exposed neovascularity. This is a particularly plausible route in many canine cases of FCE. Cases commonly involve large to giant breed, middle-aged dogs that are predisposed to type II intervertebral disk disease where neovascularization of a diseased annulus could be expected.3,5,12 Other hypotheses include direct penetration of spinal arteries or veins after rupture of the nucleus pulposus through the dorsal or dorsolateral annulus fibrosis and herniation of disk material through the vertebral end plate into the marrow cavity. In humans, such herniations are recognized as Schmorl’s nodules.13 Herniated disk material goes through the venous sinuses for retrograde entrance to the spinal cord. Schmorl’s nodules have not been documented in any veterinary cases of FCE.3,5,12 Because of the absence of gross lesions, however, neither intervertebral disks nor vertebrae were examined microscopically in this case. One final suggested mechanism is extrusion of disk material into anomalous or persistent embryonic vasculature of the annulus fibrosis.3,5,12 Because this case involved a young animal with no evidence of disk disease or vascular trauma, this may be the most plausible mechanism for this calf, although no anomalous or persistent embryonic vasculature was found at necropsy.

Another commonly cited factor in the development of fibrocartilaginous myelopathy is trauma or vigorous exercise (or both). It is believed that stress placed on the spine may increase the chance of acute disk herniation and provide sudden increases in intra-abdominal pressure to propel emboli into the circulation and contribute to retrograde blood flow in venous sinuses and spinal veins.3,12 Though no evidence of direct spinal trauma was found in this case, the 2 areas of subcutaneous edema on the nonrecumbent side of the calf may have been related to a recent traumatic incident. Diskspondylitis was suggested as a predisposing factor for the development of fibrocartilaginous emboli in pigs.

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References