Pathology in Practice

In collaboration with the American College of Veterinary Pathologists

History

A 1-year-old 100-kg female pony was presented to the Veterinary Teaching Hospital- Equine Internal Medicine Service with a sudden-onset of hind limb paralysis and recumbence. The signs were first noted 24 hours prior to admission, and there was no history of trauma. There were 4 other horses at the farm, and all were clinically normal. The horses were not vaccinated or dewormed.

Clinical and Gross Findings

At the time of admission, the pony was lethargic and in left lateral recumbency. Physical examination revealed tachycardia (80 beats/min), tachypnea (38 breaths/min) with superficial breathing and dyspnea, and hyperemic and dry mucous membranes. The pony's rectal temperature was within reference limits.

Neurologic examination revealed spastic paralysis of both hind limbs and intermittent episodes of front limb pedaling. The pony was unable to stand even with assistance. No abnormalities on mental status and cranial nerves were detected. Urine and fecal production were normal. A CBC and serum biochemical analyses revealed leukocytosis (22.0×10^3 cells/ μL ; reference range, 5.4×10^3 to 14.3×10^3 cells/ μL) due to neutrophilia (18.4×10^3 cells/ μL ; reference range, 2.2×10^3 to 8.5×10^3 cells/ μL), high activities of aspartate aminotransferase (894 U/L; reference range, 0 to 366 U/L) and alanine aminotransferase (61.9 U/L; reference range, 0 to 23 U/L), and hyperlipidemia (432 mg/dL, reference limit, ≤ 200 mg/dL).

Fluid therapy with lactated Ringer solution was instituted as well as treatment with dimethyl sulfoxide (1 g/kg), dexamethasone (0.1 mg/kg), gentamicin (6.6 mg/kg), and penicillin (22,000 U/kg) for presumptive trauma. Within 24 hours after onset of treatment, the pony appeared alert but remained recumbent, with spastic paralysis of the hind limbs.

Luis H. G. Saraiva, DVM; Camila I. Amaral, DVM; Matheus V. L. Moreira, DVM, MSc; Jéssica G. Oliveira, DVM; Maristela S. Palhares, DVM, PhD; Raffaella B. C. Teixeira, DVM, PhD; Renata P. A. Maranhão DVM, PhD; Roselene Ecco, DVM, PhD*

Clinic and Surgery Department, Veterinary School, Universidade Federal de Minas Gerais, Belo Horizonte, Minas Gerais, Brazil

*Corresponding author: Dr. Ecco (ecco@vet.ufmg.br)

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Figure 1—Postmortem images of a longitudinal section of the vertebral column and spinal cord spanning T9 to T12 (A) and gross (B) and subgross (C) transverse section of the spinal cord at the level of T10 from a 1-year-old 100-kg female pony euthanized after minimal improvement with treatment following a sudden-onset of hind limb paralysis and recumbence. A and B—The spinal cord is yellow and enlarged (edema) surrounding a focal dark-red area. C—H&E stain; bar = 2 mm.

Due to severe neurologic dysfunction and poor prognosis, the owners elected euthanasia for the pony.

On necropsy, a longitudinal section of the vertebral column revealed lesions in the spinal cord, between T7 and T12. The spinal cord was enlarged, and on the cut surface, there was a yellowish and softened extensive area with loss of differentiation between gray and white matter. A translucent and mildly viscous fluid was associated with the yellow areas. At the level of T10, ventrally and involving meningeal space and superjacent white matter, there was an amorphous red-dark area (1.0 X 1.0 X 0.8 cm; **Figure 1**).

Formulate differential diagnoses, then continue reading.

Histopathologic Findings

Microscopically, a myxoid nodular tissue interspersed with vacuoles, especially in the central area, was found in the meningeal space extending to the white matter in the spinal cord at the level of T10. The myxoid area of the embolus was stained by Alcian blue and Toluidine blue special stains (**Figure 2**). This embolus was surrounded by a ring of collagenous tissue, which was confirmed by Masson's trichrome stain. In the parenchyma adjacent to the embolus, there were multifocal vacuolation and axonal degeneration in the white matter, as well as

Figure 2—Photomicrographs of a section of spinal cord from the pony described in Figure 1. There is an intravascular embolus characterized by myxoid tissue with multiples vacuoles in the central area and degeneration of the adjacent parenchyma. H&E stain; bar = 500 μ m. Inset—Myxoid tissue of the embolus is stained by Toluidine blue special stain and interpreted as glycosaminoglycans (proteoglycans). Toluidine blue special stain; bar= 200 μ m.

Figure 3—High-magnification photomicrograph of the area adjacent to the embolus from the spinal cord shown in Figure 2. Vacuolation and axonal degeneration (outlined arrows) are evident in the white matter and neuronal chromatolysis (black arrow) is evident in the gray matter. H&E stain; bar = 100 μ m.

diffuse neuronal loss with marked multifocal chromatolysis in the gray matter (**Figure 3**). In this region, areas of moderate multifocal parenchymal necrosis with Gitter cells were also found.

Morphologic Diagnosis and Case Summary

Morphologic diagnosis and case summary: multifocal to coalescing and marked degenerative myelopathy associated with a locally extensive fibrocartilaginous embolus in a 1-year-old pony.

Comments

Fibrocartilaginous embolic myelopathy is an acute neurologic syndrome caused by the occlusion of blood vessels in the spinal cord parenchyma, surrounding leptomeninges, or both caused by fibrocartilaginous masses similar to the vertebral nucleus pulposus. This condition has been described in cattle,¹ dogs,^{2,3} cats,⁴ swine,⁵ tigers,⁶ turkeys,⁷ and humans.⁸ It is a rare disease in horses,⁹⁻¹³ and to our knowledge, this is the first report in a pony.

Neurologic signs can vary according to the anatomic location of the emboli and the extension of the lesion in the central nervous system.¹¹ The clinical signs of the pony of the present report were similar to a case described by Panziera et al,¹³ which to our knowledge is the only reported case with thoracic spinal cord lesion and paralysis of the pelvic limbs. The other reports are from cases of cervical spinal lesions with consequent paralysis of the thoracic and pelvic limbs.⁹⁻¹²

Microscopically, neuronal necrosis and axonal degeneration in the spinal cord are caused by ischemia secondary to vascular obstruction.¹⁴ Based on histopathology findings in the case from this report, the location of the embolus was in the spinal artery, causing ischemia in an extensive area of the spinal cord. The embolus stained by Alcian blue and Toluidine blue had similar components of the nucleus pulposus from the intervertebral disk.¹⁵ The morphology and special stains indicated the chronicity of the lesion. It is possible that early neurologic signs were mild and unnoticed by the owner or that exacerbation of an unstable lesion may have led to decompensation in the reported time frame. In addition, the vacuoles interspersed by myxoid tissue were interpreted as a result of degeneration and loss of cells from nucleus pulposus named notochordal cells.¹⁵ As changes were seen in the intervertebral disks, loss of notochordal cells might have occurred in a later stage, after embolus location in the spinal cord.

The intervertebral disk is the source of the fibrocartilaginous emboli, but the pathophysiology of this vascular invasion is not completely understood.¹⁶ The first proposed theory is the presence of minor trauma or physical activity resulting in increased intervertebral pressure, leading to infiltration of semifluid

material from the nucleus pulposus inside small arteries and retrograde propagation in the radicular artery.^{11,17} A second theory involves the presence of anomalous vasculature in the disk and consequent entrance of the nucleus pulposus material in the lumen of these vessels. These anomalous vessels could originate from reminiscent embryonic vessels or from neovascularization caused by chronic inflammatory processes.^{17,18}

Previous reports in horses did not show breed predisposition, but most were males and older than 8 years old.^{10–13} Those findings support the existence of predisposing factors such aging of the fibrocartilaginous ring or its fissure, leading to intravascular herniation of the material.¹⁸ Trauma and intensive exercise are also risk factors for the development of fibrocartilaginous myelopathy.¹⁹ For the pony of the present report, no macroscopic lesions suggestive of trauma were observed. There was also no reported history of strenuous exercise prior to this event, and because the horse was only a 1-year-old, this is very unlikely. The hind limb spastic paralysis was secondary to upper motor neuron lesions, caused by necrosis of the thoracic spinal cord. The rapid progression of signs could have been related to the extension of the ischemic lesions and their consequences such as spinal cord malacia.^{19,20}

Gross and histologic changes were consistent with ischemic myelopathy resulting from fibrocartilaginous embolism. The antemortem diagnosis of this disease is challenging using accessible diagnostic techniques.¹¹ Therefore, this condition should be considered in the list of differential diagnosis for neurologic signs consistent with spinal cord lesions in horses and ponies.^{10,11,13}

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References

1. Landolfi JA, Saunders GK, Swecker WS. Fibrocartilaginous embolic myelopathy in a calf. *J Vet Diagn Invest.* 2004;16(4):360–362.
2. Bartholomew KA, Stover KE, Olby NJ, et al. Clinical characteristics of canine fibrocartilaginous embolic myelopathy (FCE): a systematic review of 393 cases (1973–2013). *Vet Rec.* 2016;179(25):650.
3. Mari L, Behr S, Shea A, et al. Outcome comparison in dogs with a presumptive diagnosis of thoracolumbar fibrocartilaginous embolic myelopathy and acute non-compressive nucleus pulposus extrusion. *Vet Rec.* 2017;181(11):293.
4. Mikszewski JS, Van Winkle TJ, Troxel MT. Fibrocartilaginous embolic myelopathy in five cats. *J Am Anim Hosp Assoc.* 2006;42(3):226–233.
5. Haynes JS, Benson EJ. Ischemic myelomalacia due to cartilage emboli associated with chronic diskospondylitis in a group of swine. *J Vet Diagn Invest.* 1999;11(6):533–536.
6. Adaska JM, Lynch S. Fibrocartilaginous embolic myelopathy in a tiger (*Panthera tigris sumatrae*). *J Zoo Wildl Med.* 2004;35(2):242–244.
7. Stedman NL, Brown TP, Rowland GN. Intravascular cartilaginous emboli in the spinal cord of turkeys. *Avian Dis.* 1998;42(2):423.
8. AbdelRazek M, Elsadek R, Elsadek L. Case series of two patients with fibrocartilaginous embolism mimicking transverse myelitis of the spinal cord. *J Clin Neurosci.* 2017;40:66–68.
9. Taylor HW, Vandevelde M, Firth EC. Ischemic myelopathy caused by fibrocartilaginous emboli in a horse. *Vet Pathol.* 1977;14(5):479–481.
10. Fuentealba IC, Weeks BR, Martin MT, et al. Spinal cord ischemic necrosis due to fibrocartilaginous embolism in a horse. *J Vet Diagn Invest.* 1991;3(2):176–179.
11. Sebastian MM, Giles RC. Fibrocartilaginous embolic myelopathy in a horse. *J Vet Med A Physiol Pathol Clin Med.* 2004;51(7–8):341–343.
12. Dörner C, Uzal F, Carvalho F, et al. Compressive myelopathy caused by epidural haematoma associated with fibrocartilaginous embolism in a horse. *Eq Vet Educ.* 2015;27(8):405–409.
13. Panziera W, Bianchi RM, Pereira PR, et al. Ischemic myelopathy caused by fibrocartilaginous embolism in a horse. *Cienc Rural.* 2018;48(2):e20170436.
14. Walling BE, Stewart MC, Valli VE. Pathology in practice. Fibrocartilaginous embolism. *J Am Vet Med Assoc.* 2011;239(2):199–201.
15. Hansen T, Smolders LA, Tryfonidou MA, et al. The myth of fibroid degeneration in the canine intervertebral disc: a histopathological comparison of intervertebral disc degeneration in chondrodystrophic and nonchondrodystrophic dogs. *Vet Pathol.* 2017;54(6):945–952.
16. Cauzinille L. Fibrocartilaginous embolism in dogs. *Vet Clin N Anim Pract.* 2000;30(1):155–167.
17. De Risio L, Platt SR. Fibrocartilaginous embolic myelopathy in small animals. *Vet Clin North Am Small Anim Pract.* 2010;40(5):859–869.
18. Neer TM. Fibrocartilaginous emboli. *Vet Clin North Am Small Anim Pract.* 1992;22:1017–1026.
19. Tesser ES, Pavarini SP, Bezerra Júnior PS, et al. Fibrocartilaginous embolic myelopathy in a dog. *Acta Sci Vet.* 2009;37(3):291–294.
20. Benson JE, Schwartz KJ. Ischemic myelomalacia associated with fibrocartilaginous embolism in multiple finishing swine. *J Vet Diagn Invest.* 1998;10(3):274–277.