Pathology in Practice

History

A 1-year-old sexually intact male Holstein-Friesian calf was evaluated at the Scottish Centre for Production Animal Health and Food Safety at the University of Glasgow School of Veterinary Medicine. The calf was in good body condition and weighed 390 kg (858 lb) but had had an abnormal hypermetric hopping gait and bilateral hyperextension of the pelvic limbs since birth.

Clinical and Gross Findings

Clinical examination revealed slightly harsh chest sounds. Tens of small (0.5 to 1 cm in diameter), ovoid, alopecic, crust ing areas consistent with dermatophilosis were scattered over the right lateral aspect of the neck extending caudally over the right shoulder, with similar, solitary lesions over the right lateral thorax. Wide placement of the hind feet and mild muscular atrophy over the pelvic limbs were apparent (Figure 1). No other abnormalities were detected. A single analgesic treatment with meloxicam (0.6 mg/kg [0.27 mg/lb], IV), an NSAID, did not visibly improve the calf’s abnormal gait.

Blood and fecal samples were submitted to Veterinary Diagnostic Services at the University of Glasgow for biochemical analysis, hematologic evaluation, and fecal analysis. Blood was also submitted to the Scottish Agricultural College, Ayr, Scotland, for viral serologic evaluation. The serum biochemical analysis and hematologic evaluation results were within reference limits, and no abnormalities or parasites were detected during microscopic examination of a blood smear. Fecal analysis (microscopic examination of a fecal smear as well as modified McMaster, Baermann, and Boray sedimentation testing) revealed no parasites. Results of bacteriologic culture of feces for Salmonella spp were negative. The serum was negative for antibodies against louping ill virus (determined via hemagglutination inhibition) and bovine herpesvirus 1 (determined via glycoprotein B assessment [26% positivity]) but positive for antibodies against bovine viral diarrhea virus (determined via ELISA [142% positivity]).

Given the poor prognosis, the calf was euthanized and transported to the Anatomic Pathology Service at the University of Glasgow for postmortem examination. Grossly, the spinal cord at the level of the thoracolumbar intumescence (T8 to L4 vertebrae) was markedly enlarged by an intramedullary, soft linear swelling (Figure 1). The circumference of the spinal cord at the widest point was 7 cm, narrowing to 4.5 cm cranial and caudal to the lesion. Measurements of the brain and visceral organs were considered normal. On cut section, the central canal and gray matter were segmentally replaced by a large, well-demarcated cystic cavity filled with approximately 15 mL of clear, mildly viscous fluid. An ear notch sample was negative for bovine viral diarrhea virus antigen.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page →

Figure 1—Photographs of the spinal cord of a 1-year-old sexually intact male Holstein-Friesian calf with an abnormal hypermetric hopping gate and bilateral hyperextension of the pelvic limbs since birth that was euthanized because of poor prognosis. A—Grossly, the spinal cord at the level of the thoracolumbar intumescence (vertebrae T8 to L4) was markedly enlarged by an intramedullary, soft linear swelling. B—On cut section, the central canal and gray matter of the spinal cord were segmentally replaced by a large, well-demarcated cystic cavity filled with approximately 15 mL of clear, mildly viscous fluid.
Histopathologic Findings

Samples of the spinal cord, brain, skeletal muscles, and other visceral organs were processed for histologic examination. At the level of the cranial lumbosacral intumescence (cranial extent of the lesion), the white matter of the spinal cord directly ventral to the central canal was markedly expanded and rarified by clear spaces (interpreted as edema) in a wedge shape extending to the glia limitans and ventral aspect of the leptomeninges. Within the edematous area, there were scattered eosinophilic, degenerate astrocytes and a low number of microglia. The median septum of the gray matter adjacent to this area was also markedly loosened by vacuolar clear spaces, in which there were similar numbers of brightly eosinophilic, degenerate astrocytes.

At the level of the midlumbosacral intumescence, the spinal cord was markedly dilated (and the neuropil was markedly and peripherally compressed) by a large, central cystic space (syrinx) that merged with and replaced the central canal (Figure 2). The syrinx was intermittently lined with remnants of the ependyma of the central canal. The gray matter was markedly compressed and distorted on either side of the syrinx; the white matter tracts were multifocally pale and loosened by clear spaces of myelin degeneration and edema but did not communicate directly with the central syrinx. The caudal lumbosacral intumescence of the spinal cord was similar to the cranial portion of the lesion.

Cranial to the lesion, there was mild, diffuse, chronic Wallerian degeneration within the dorsal funiculus of the white matter, with occasional spheroid formation. Moderate to marked, diffuse Wallerian degeneration was also observed within the sciatic nerve. Findings for tissue samples from the brain, skeletal muscles, and other visceral organs were considered normal.

Morphologic Diagnosis and Case Summary

Morphologic diagnosis: marked syringomyelia at the level of the lumbosacral intumescence.

Case summary: segmental syringomyelia in a 1-year-old calf.

Comments

Prior to necropsy, differential diagnoses considered for the 1-year-old calf with an abnormal hypermetric hopping gait and bilateral hyperextension of the pelvic

Figure 2—Photomicrographs of sections of the spinal cord of the calf in Figure 1. A—At the level of the midlumbosacral intumescence, the spinal cord was markedly dilated by a large, central cystic space that merged with and effaced the central canal. H&E stain; bar = 5 mm. B—The cystic space in the spinal cord was intermittently lined with remnants of simple cuboidal epithelium from the central canal. H&E stain; bar = 50 mm. C—Representative section from an unaffected region of the spinal cord cranial to the dilation illustrating the segmental nature of the lesion. H&E stain; bar = 5 mm.
limbs (upper motor neuron signs) included congenital defects of the lumbar vertebral canal or column or spinal cord, bovine viral diarrhea virus infection, focal abscess, degenerative disk disease or osteoarthritis, or Lupinus spp poisoning. Owing to the young age of the calf and the unremarkable clinicopathologic findings (ie, no indication of infection), a congenital spinal defect was most likely. Because this calf was negative for bovine viral diarrhea virus antigen but positive for antibodies against the virus, active infection was considered unlikely. *Lupinus* spp, ingestion of which causes neurologic signs in cattle, are not primarily found in the United Kingdom.

Syringomyelia is an uncommon tubular, cystic cavitation containing CSF and extracellular fluid that typically extends over several segments of the spinal cord and possibly into the medulla. Rarely acquired, this disorder is generally a congenital, spinal dysraphism in mammalian species. Most common in dogs (particularly young Weimaraners), syringomyelia is associated with bilateral hind limb deficits and a hopping gait in which the hind limbs move together. Sciolosis in the thoracolumbar region, depression of the sternum, and abnormal hair growth in the dorsal cervical region may also occur. There is no progression or regression of clinical signs with age, and clinical signs poorly correlate with the extent of the lesion. Often associated with spina bifida, syringomyelia also frequently develops in the dysraphic spinal cord of arthrogrypotic (primarily Charolais) calves. Hydromyelia, a dilation of the central canal of the spinal cord due to a blockage of CSF flow, is also common in these calves but has been attributed to persistent or transient internal obstructive hydrocephalus within the ventricles. Hydromyelia has been hypothesized as a prerequisite to syringomyelia but has been deemed an unlikely cause in most cases. Similarly, in humans, syringomyelia is attributed to a Chiari malformation (wherein the cerebellum blocks CSF outflow through the foramen magnum) in approximately 50% of cases; for the remainder of cases, syringomyelia is primarily attributable to other developmental malformations of the foramen magnum or within the spinal cord or vertebral canal, trauma, or inflammation.

Syringomyelia is not histologically apparent until at least 8 months of age, and the spinal cord may appear grossly normal. Often in the lumbar region of the vertebral column, cavitation generally develops in the central gray matter dorsolateral to the central canal, but the white matter may be affected in the commissures if the space extends from one side to another; the edematous tissue around the cavity stains poorly with H&E stain, and its communication with the central canal is difficult to demonstrate. These lesions generally occur throughout the spinal cord and possibly including the medulla; localized or segmental lesions surrounded by healthy spinal cord have not been reported, to our knowledge.

Although bovine viral diarrhea virus is panzootic and has a tropism for the CNS of calves persistently infected in utero, neuropathologic lesions associated with bovine viral diarrhea virus infection are rare. When present, the most common lesions are cerebellar hypoplasia or myelin deficiency leading to hypomyelination of the cerebellar white matter and ocular nerve. Because the calf of the present report was negative for bovine viral diarrhea virus antigen but positive for antibodies against the virus, persistent bovine viral diarrhea virus infection was considered unlikely; however, calves exposed to bovine viral diarrhea virus in utero from 150 to 175 days of gestation may develop congenital abnormalities without persistent infection.

The segmental nature of the syringomyelia in the case described in this report was highly unusual because typically the spinal cord is more uniformly affected. The clinical history of the calf suggested a congenital lesion: an abnormal hypermetric hopping gate with bilateral hyperextension of the pelvic limbs with the possibility of arthrogryposis since birth. The segmental nature of the syringomyelia was not indicative of continuous compression and persistent blockage of CSF drainage. There were no apparent external sources of spinal cord compression or vertebral instability detected adjacent to the lesion. Given the lack of notable spinal column abnormalities, morbidity in this calf likely resulted from segmental syringomyelia as a result of dysraphism (a congenital disease resulting in gray matter hypoplasia or alterations in the flow of CSF) or, less likely, a dynamic spinal cord compression at the thoracolumbar intumescence.

References