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Postanesthetic Poliomyelopathy in a 7-Day-Old Calf

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A 7-day-old Holstein-Friesian calf weighing approximately 54 kg was presented to Cornell University Hospital for Animals (CUHA), Large Animal Medicine Service, for acute diarrhea and abdominal distention. She had been healthy before the acute onset of illness. No other calves in the barn were ill, but 2 other calves on the farm had died as a result of abomasitis (diagnosed by postmortem examination) in the past year. On the morning of presentation, the calf was bloated and developed diarrhea. The referring veterinarian treated the calf with flunixin meglumine (1.1 mg/kg, IV), dextrose (100 mL/kg, IV), and dexamethasone (0.2 mg/kg, IV), and the calf was referred to the CUHA.

Upon arrival, the calf was obtunded, recumbent, and extremely dehydrated, with sunken eyes and prolonged skin tenting. The calf was hypothermic, 37° C, and tachycardic (160 beats/min), with no arrhythmias or murmurs ausculted. Her mucous membranes were pale and capillary refill time could not to be assessed. The calf was tachypneic (50 breaths/min) and auscultation of the lungs identified slightly harsh bronchovesicular sounds bilaterally. The calf's abdomen was severely distended bilaterally with no pings heard on either side. She had profuse light-brown, foul-smelling diarrhea with no blood noted. A nasogastric tube was passed immediately, but only a small amount of fetid fluid was retrieved. A jugular catheter was introduced and hetastarch (500 mL IV), lactated ringers solution (240 mL/kg/h), and oxyglobin (10–30 mg/kg) were given. The calf also received intranasal oxygen insufflation at 5 L/h. Blood gas analysis, serum electrolytes, and glucose concentrations were determined immediately with stall-side point-of-care equipment.[a]

The calf had severe mixed metabolic and respiratory acidosis with a pH of 7.07 (reference range, 7.32–7.44). Bicarbonate value was 18 mEq/L (reference range, 25–30 mEq/L) and base excess −12 mEq/L. The venous P_{CO_2} was 57.3 mm Hg (reference range, 36–46 mm Hg). Blood lactate concentration was 13.3 mmol/L (reference range, 0.3–1.5 mmol/L) and glucose concentration was 359 mg/dL (reference range, 31–77 mg/dL). Serum sodium concentration was 137 mEq/L (reference range, 134–145 mEq/L), potassium was 3.9 mEq/L (reference range, 3.9–5.3 mEq/L), and chloride was 96 mEq/L (reference range, 98–105 mEq/L). PCV was 36% (reference range, 23–35%) and total solids were 4.8 g/dL (reference range, 5.9–8.1 g/dL). The white blood cell count was 16,200/μL (reference range, 5,600–13,700/μL) with a neutrophilia (11,700/L) and a left shift (2,400 bands/μL). A mild lymphopenia of 1,600/μL (reference range 1,900–7,400/μL) was also present. Mild toxic changes were noted in the white blood cells. Activated charcoal (35 cm^2) and penicillin (180,0000 U) were given via the nasogastric tube to decrease toxin absorption and anaerobic gram-positive or gram-negative bacterial growth. Potassium penicillin (20,000 IU/kg, IV q8h) was given after placement of the jugular catheter. Within 1 hour of hospital admission, a gastrotomy was performed to remove abomasal contents. Surgery was performed in this calf to be able to identify severe abomasal ulcerations and to surgically close any that were found, as well as to remove abomasal contents, gas, and potentially toxic bacteria. Mean blood pressure measured with a DINAMAP[b] before anesthesia was 80 mm Hg. The calf was anesthetized and placed in left lateral recumbency. During surgery, mean arterial pressure decreased as low as 30 mm Hg and averaged 45 mm Hg. During recovery, the calf was treated with Torbugesic[c] (0.4 mg/kg, IV as needed for postoperative pain management), hypertonic saline (30 mL IV once), and lactated ringer's solution with 5% dextrose (120 mL/kg/h, IV). Intranasal oxygen insufflation was continued at 5 L/h and a heat lamp and Bair Hugger[d] was used to correct hypothermia. Arterial blood gas values postsurgically revealed Pa_{O_2} 177 mm Hg, Pa_{CO_2} 56.8 mm Hg, pH 7.13, bicarbonate 19 mEq/L, and oxygen saturation 99%, and lactate concentration had decreased to 7.7 mmol/L. Serum sodium concentration was 137 mEq/L, potassium was 3.9 mEq/L, and chloride was 98 mEq/L. The PCV was 23% and total protein had decreased to 4.0 g/dL. During recovery, the calf was given furosemide (1 mg/kg, IV) because she had not urinated since she had been hospitalized. The calf urinated normally soon after the furosemide. Potassium penicillin (20,000 IU/kg, IV q6h) and amikacin (20 mg/kg, IV q24h) were given after the calf had urinated. Whole blood (500 mL) also was given. Heparin (40 U/kg) was added to the blood in an attempt to activate antithrombin III activity. Blood pressure (measured with a DINAMAP[b]) and blood glucose (measured with an Accucheck[e]) were monitored q4h postoperatively. Blood pressure (85–90 mm Hg) and glucose concentrations (55–75 g/dL) remained normal overnight. Her urine production appeared adequate and she passed loose feces with blood.

The following day, the calf was alert and responsive. She drank 300 mL colostrum with a lactaid[f] tablet added to help with digestion. Parenteral nutrition (112 mL/h constant rate infusion of 50% dextrose, 8.5% amino acids, 20% lipids, and vitamin B complex) was begun on postoperative day 1 and lactated ringer's solution with 20 mEq KCL (180 mL/kg/d) was continued, as were antimicrobials. The calf also was given a liter of bovine plasma IV postoperatively on day 1. The calf was unable to rise on her own the morning after surgery. She could stand with assistance and bear

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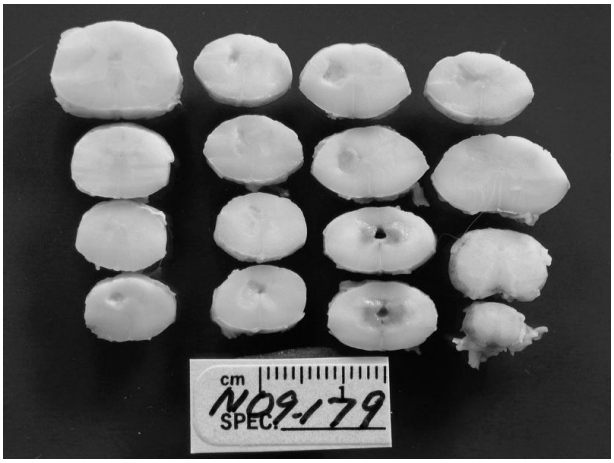

Fig 1. Gross transverse sections of spinal cord from the Holstein calf from T7 to L3. The left grey matter was soft and depressed from the surface. These lesions were bilateral from L3 through L6 and slightly discolored.

weight on all 4 limbs but was unable to propel herself with her hind limbs. Head, neck, and thoracic limb function were normal. The calf's ability to stand did not improve by postoperative day 3. Neurologic evaluation determined that the calf had little voluntary use of the right and left hind limbs, with the left hind limb being the most affected. Muscle tone was present but decreased in both hind limbs. Nociception was intact in both hind limbs but the patellar reflex was severely decreased in the left hind limb. The right hind limb had normal reflexes. Neuroanatomically, the lesion was localized between T3 and L3, with some grey matter involvement on the left between L4 and S1. Differential diagnoses for pelvic limb paresis were spinal cord trauma, ischemic poliomyelopathy, thromboembolism, and fibrocartilagenous embolism.

The calf's attitude, appetite, and manure returned to normal over the next 4 days. Intravenous fluids and parenteral nutrition were discontinued. Postoperative intravenous antimicrobials were discontinued on day 6 and procaine penicillin was used instead (20,000 U/kg, SQ q12h). The calf continued to be hypotonic in her left hind limb and had little or no improvement in ability to use her hind limbs. She had marked scoliosis in her thoracolumbar spine, which curved to the right. Cutaneous hypalgesia was noted on the left thoracolumbar trunk. Physical therapy of the hind limbs was initiated.

The calf had considerable monetary value and, in spite of the grave prognosis, the owner wanted to give the calf more time to recover at home; consequently, the calf was discharged. The calf was discharged with specific instructions for physical therapy, feeding q6h and prevention of prolonged recumbency.

Two weeks after discharge, the calf was returned to CUHA. She was very bright and had grown but remained paralyzed in her hind limbs. She was humanely euthanized and a postmortem examination performed.

Gross postmortem examination findings revealed a scar on the abomasum presumably from the gastrotomy site, along with an abomasal perforation covered by omentum. The right middle lung lobe was diffusely atlectactic. On gross examination of the transverse sections of the preserved spinal cord, from T7 though L3, the left grey matter was soft and collapsed from the surface. This change was bilateral from L3 through L6 and the tissue was slightly discolored (Fig 1). On histopathology, extensive severe poliomyelomalacia was observed, extending from T7 through S1. The grey matter on the left side was affected in all of the segments. The grey matter on the right side was affected in the middle and caudal lumbar segments. Where the grey matter lesion was most pronounced, the entire grey matter was degenerate, with complete necrosis at the periphery, including the adjacent white matter (Fig 2). In this region, all of the parenchyma was replaced by lipid-filled macrophages. In the central portion of the grey matter, the parenchyma still was present but many neurons were absent and those present revealed an ischemic degeneration. The more severe peripheral lesion was in the end distribution of branches supplied by the ventral spinal artery and penetrating blood vessels from the vascular plexus on the surface of the spinal cord. The spinal cord lesions in this calf were thought to be due to hypoxia, ischemia, or both, as-

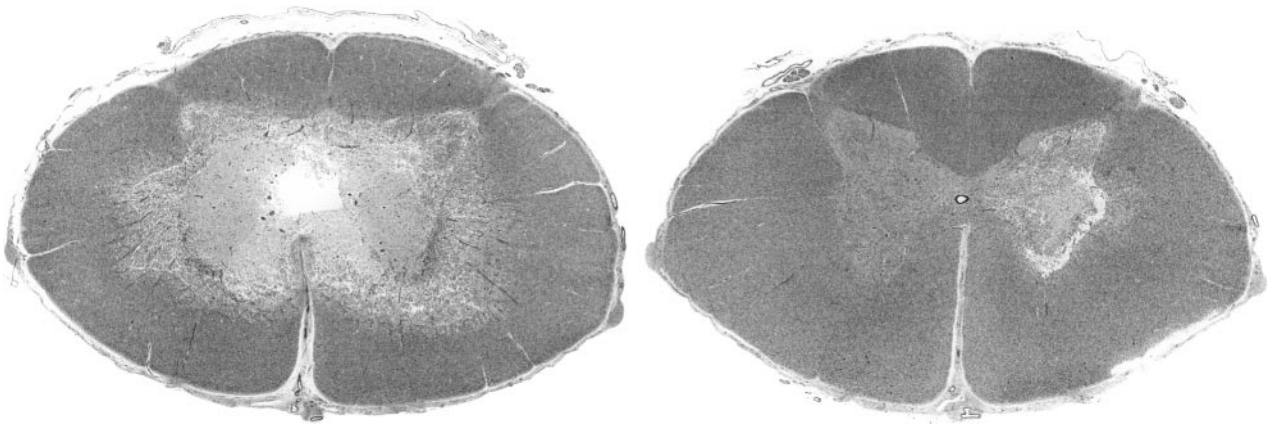

Fig 2. Two histological sections of spinal cord from the Holstein calf. H&E stain. Magnification: 13× (150 mm × 13 mm). Two cross-sections in which the grey matter lesion was most pronounced. The entire grey matter was degenerate with complete necrosis at the periphery, which included the adjacent white matter. All of the parenchyma was replaced with lipid-filled macrophages.

sociated with recumbency and low blood pressures during anesthesia and surgery. The ventral spinal artery supplies blood to the grey matter in regions in which the lesion was most extensive. No thrombi or fibrocartilaginous emboli were seen within the vessels supplying the spinal cord. The final diagnosis was postanesthetic poliomyelopathy.

The various treatments of the calf were performed for stabilization before surgery. Activated charcoal may not adsorb clostridial or sarcinia toxins but has been utilized in cattle with ruminal acidosis or bloat[1] and is considered safe for use in this species. The efficacy of penicillin against *Sarcina* is unknown, but it may be effective locally, and penicillin is safe.[2] The decision for surgery was considered to be the best option for this calf, although no studies have proven whether surgical or medical management is better for calves with abomasitis.

Anesthetic-related poliomyelopathy has been reported in horses, but this lesion has not been reported in calves. Its occurrence in the horse is rare and it occurs mostly in young animals undergoing elective surgery that are placed in dorsal recumbency.[3,4] After surgery, the only clinical signs noted involved the hind limbs and varied from progressive paraparesis to paraplegia without nociception.[3,5]

Postmortem examination of horses with anesthetic-related poliomyelopathy identified acute hemorrhagic poliomyelopathy, which appeared most severe and consistent in thoracic spinal cord segments. Gross pathology examination of the spinal cord in these horses revealed hemorrhage or congestion in the gray matter on transverse sections. Neuronal degeneration and hemorrhage were noted in dorsal and ventral columns of gray matter both asymmetrically and symmetrically. The ventral column was more extensively affected. The lesion predominates in areas of high vascularity.[6] In the cases reported, the horses all were placed in dorsal recumbency. This position may contribute to the disease process because the abdominal viscera could impede drainage of the caudal vena cava, azygous vein, or both, causing venous stasis and resistance to outward flow.[7,8] Another hypothesis is an inability to adjust to localized hemodynamic changes that occur with dorsal recumbency. Regardless, many horses undergo anesthesia for prolonged periods of time in dorsal recumbency and do develop hemorrhagic poliomyelopathy despite low systemic blood pressures.[7]

The majority of spinal cord grey matter is supplied by the ventral spinal artery and it has the highest area of vascularity, which makes it most susceptible to ischemia.[3–13] Distribution of the myelopathy lesions makes spinal cord ischemia the most commonly proposed mechanism.[8] Support for this hypothesis comes from experiments in rabbits and mice in which spinal cord ischemia is produced by occlusion of the abdominal aorta.[8,13,14] The lesions associated with the ischemia are poliomyelomalacia, and the severity of this lesion is related to the duration of ischemia.[6–8] Although the spinal cord is supplied segmentally by branches of spinal arteries at each intervertebral foramen, considerable variation exists in the presence and size of the these branches among species and individuals of one species. These differences may contribute to the risk of an individual animal for spinal cord ischemia associated with general anesthesia and recumbency.

In one case report, a 2-year-old thoroughbred developed pelvic limb paralysis 5 hours after general anesthesia and castration performed in right lateral recumbency, developed pelvic limb paralysis, and was euthanized 16 hours later.[6] Regional ischemia along with secondary congestion and edema probably were important causes of the horse's clinical signs.[6]

Muscle tone is dependent both on the somatic efferent innervation of striated skeletal muscle and the general proprioceptive afferents that innervate neuromuscular spindles, joints, tendons, and ligaments. Interruption of either the somatic efferents or the general proprioceptive afferents will lead to loss of muscle tone, and if this lesion is unilateral, the imbalance in tone can lead to vertebral curvatures.[15] This calf's lesions were most pronounced on the left side, affecting both the afferents entering the dorsal grey column as well as the efferents in the ventral grey column. The loss of muscle tone on the left side caused the scoliosis with concavity on the right side.

This calf's clinical signs were due to the poliomyelopathy caused by ischemia associated with general anesthesia and recumbency. The lesions were very similar to those reported in horses and humans with postanesthetic poliomyelopathy. Low blood pressure and recumbency for an extended period before and during surgery likely played key roles in the development of this lesion.

Footnotes

[a] i-STAT Corporation, East Windsor, NJ
[b] GE Medical Systems Information Technologies, Waukesha, WI
[c] Torbugesic, Fort Dodge Animal Health, Fort Dodge, IA
[d] Arizant Inc, Prairie, MN
[e] Hoffmann-La Roche Inc. (Roche), Nutley, NJ
[f] Lactaid tablets, McNeil Nutrionals LLC, Ft. Washington, PA

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