A 4-month-old Percheron filly that weighed approximately 300 kg (660 lb) was administered xylazine hydrochloride (300 mg, IV) and ketamine hydrochloride (1,100 mg, IV) in preparation for routine umbilical hernia repair surgery (day 1). The horse was intubated, and anesthesia was maintained via inhalation of an isoflurane-oxygen mixture. For the surgical procedure, the horse was placed in dorsal recumbency; the duration of anesthesia was approximately 55 minutes.

**Clinical and Gross Findings**

The horse recovered from anesthesia uneventfully except for some difficulty in standing. The horse was given phenylbutazone (1.5 g) and vegetable oil (500 mL) via a nasogastric tube approximately 5 minutes before standing. Although a moderate degree of hind limb weakness was evident, the horse stood unassisted for approximately 8 hours after surgery. At 10 hours after surgery, the horse was found recumbent and unable to rise. The horse was administered 17.5 mg of selenium and 350 mg of vitamin E IM and given a solution containing both 90% dimethyl sulfoxide (300 mL) and electrolytes in 6 L of water via a nasogastric tube. A CBC and serum biochemical analyses performed 24 hours after surgery revealed high creatine kinase activity (15,860 U/L; reference range, 10 to 350 U/L). The horse continued to ingest feed and water despite being recumbent. Additional treatment instituted at this time included lifting the horse in a sling 3 times/d and IV administration of a balanced electrolyte solution supplemented with calcium and a 20% lipid solution. Administration of phenylbutazone was repeated daily, and oral administration of dimethyl sulfoxide was repeated twice. The horse had some ability to stand with only moderate assistance from the sling on days 2 and 3 after surgery but became increasingly weaker and began to develop pressure sores on days 4 and 5. Because of the horse’s deteriorating condition, poor response to treatment, and poor prognosis, it was euthanatized on day 6 after surgery.

On gross postmortem examination, the ventral and dorsal gray matter horns of the lumbar and sacral spinal cord (region of L1 through S2) were friable and had well-delineated dark brown symmetric discoloration (Figure 1). The lesions were more pronounced in the lumbar segments of the spinal cord. Other lesions included multiple skin abrasions and contusions on the lateral and anterior aspect of the left thigh region. The healing sutured linear midline abdominal incision from the umbilical repair was also noted. No notable gross lesions were evident in other organs, including the brain. Results of fluorescent antibody and virus isolation procedures for detection of equine herpesviruses 1 and 4 performed on samples of the brain and spinal cord were negative. Liver copper, selenium, and vitamin E concentrations were within established reference ranges.

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

Sections of spinal cord were prepared for microscopic examination. Histologically, lumbar and sacral spinal cord segments L1 through S2 had severe neuropil rarefaction, cyst formation, edema, and accumulations of large numbers of macrophages and Gitter cells within the dorsal and ventral horns of the gray matter (Figures 2 and 3). Much of the vasculature within the gray matter horns was markedly congested, and endothelial cell hypertrophy was evident.

Severe neuronal depletion, swelling and hyalinization of remaining neurons, and minimal multifocal hemorrhage were also present within the affected spinal cord gray matter. Dorsal and ventral horns were equally affected. Occasional foci of axonal swelling and myelin sheath distention were also apparent within the adjacent white matter. Microscopic lesions were not evident within sections of the cervical or thoracic spinal cord. No intravascular thrombi or fibrocartilaginous emboli were detected in any of the spinal cord sections.

Morphologic Diagnosis

Severe bilaterally symmetric poliomyelomalacia (affecting lumbar and sacral spinal cord segments L1 through S2).

Comments

Postanesthetic poliomyelomalacia, a rare postsurgical complication in horses, occurs primarily in young (≤2 years old), fast-growing light- and heavy-breed horses that have been placed in dorsal recumbency under halothane anesthesia for purposes of elective surgery. The reported cases are characterized clinically by difficulty or failure of the patient to stand because of hind limb paresis or paralysis following episodes of anesthesia.1–10 Histologically, lesions may be present within the cervical,1–5 thoracic,1–5,9,10 lumbar,1–4,8,10 and sacral1–5,8 portions of the spinal cord. In some horses, the lesions extended into the cauda equina.1–6 The lesions associated with postanesthetic poliomyelomalacia are characterized by a variable extent of asymmetric to symmetric hemorrhage, neuronal degeneration, edema, and vascular congestion within dorsal and ventral horns of the gray matter.1–6,8,9 Foci of hemorrhage and edema also extend to the adjacent white matter in some horses. In those cases with a longer clinical course following surgery, mild axonal swelling in the adjacent white matter, gray matter endothelial cell hypertrophy, and accumulation of variable numbers of macrophages are also evident.2,10

Although the exact cause of postanesthetic poliomyelomalacia is unclear, the use of halothane and positioning of anesthetized equids in dorsal recumbency during surgical procedures have been considered possible risk factors. Inadequate perfusion of the spinal cord as a result of hypotension or venous congestion during surgical procedures have been considered possible risk factors. Inadequate perfusion of the spinal cord as a result of hypotension or venous congestion during surgical procedures have been considered possible risk factors.
have been hypothesized as possible pathogenic mechanisms.1–4,6–10 It is interesting to note that isoflurane was used in the horse of this report. Additionally, postanesthetic myelomalacia in a racehorse that was positioned in lateral recumbency and administered anesthetic agents other than halothane or isoflurane during a surgical procedure has also been reported.6 Although pathogenic mechanisms associated with inadequate spinal cord perfusion have been proposed, they do not completely explain why postanesthetic myelopathy in horses does not occur more frequently, given that anesthesia-induced hypotension and increased pressure on the caudal vena cava by the abdominal viscera during surgical procedures performed in dorsal recumbency are common occurrences and that postoperative complications do not develop in many instances.7 Immaturity of the spinal cord vasculature in young horses with the associated inability to accommodate the increased hemodynamic demands associated with anesthesia and dorsal recumbency may also be an important contributor.4,6,8,9 Hypovitaminosis E with resulting destabilization of biological membranes of the spinal cord has also been considered as a possible risk factor for this condition.3 Selenium toxicosis may induce severe malacia of the spinal cord gray matter horns in swine.11–13 In the horse of this report, both liver selenium and vitamin E concentrations were considered within reference ranges. Depth of anesthesia and the associated degree of hypotension may also be a factor in the development of postanesthetic poliomyelomalacia.9 Subtle anatomic differences in the spinal cord vasculature among horses may also be an important contributor to the pathogenesis of this condition. This rare postanesthetic complication in horses most likely represents a multifactorial disease process.

References