In cooperation with

What Is Your Diagnosis?

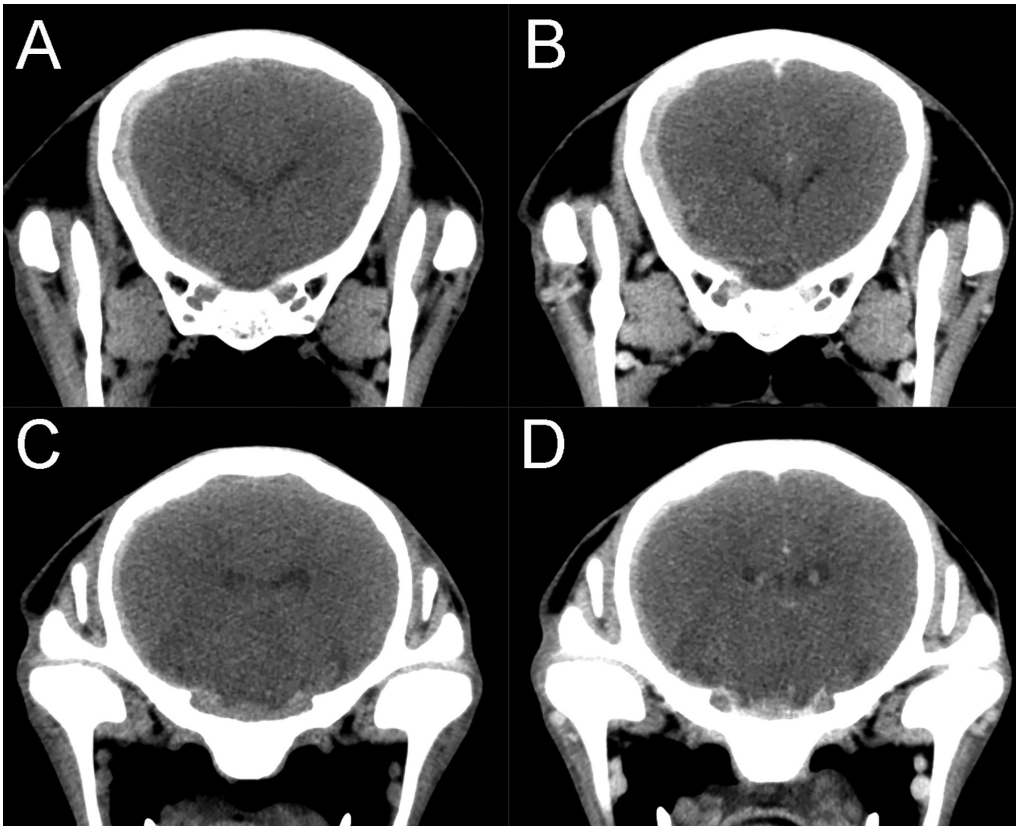

Figure 1—Reconstructed transverse plane CT images of the cranium before (A and C) and after (B and D) IV administration of contrast medium to a 14-day-old male Tennessee Walking Horse that the owner had found wedged under a gate earlier in the day. The images are at the levels of the rostral (A and B) and caudal (C and D) portions of the presphenoid bone, with the colt's right side toward the left in each image. The images are displayed in a narrow brain window (window level, 35 HU; window width, 150 HU) with a 3-mm slice thickness.

History

A 14-day-old 80-kg (176-lb) male Tennessee Walking Horse was examined after the owner found it wedged under a gate earlier in the day. On physical examination, the colt was recumbent, minimally responsive to comatose, hypothermic (34.6°C [94.3°F]; reference range, 37.5°C to 38.9°C [99.5°F to 102°F]), and tachypneic (42 breaths/min; reference range, 20 to 40 breaths/min) and had multiple abrasions on its head, limbs, and hip joints. On neurologic evaluation, the colt had miotic pupils and horizontal nystagmus, low tone in its tongue, and delayed withdrawal reflexes. Signs of deep pain could be elicited in all 4 limbs, and anal and tail tone were clinically normal. Given these findings, we localized the neurologic lesion to the brain and suspected traumatic brain injury. Orthogonal radiographic projections of the colt's head were obtained (not shown), and no abnormalities were identified. Treatment was initiated with dimethyl sulfoxide (DMSO; 1 g/kg [0.45 g/lb], IV) administered as a 10% DMSO solution in 0.45% saline (NaCl) solution with 2.5% dextrose at a rate of 250 mL/h once. Following DMSO administration, fluid therapy with 2.5% dextrose and 0.45% saline solution supplemented with 20 mEq/L of KCl and 21.38 mEq/L of calcium (20 mL/L of 23% calcium gluconate solution) was continued at a rate of 250 mL/h, IV, and the colt was hospitalized overnight. The next day, the colt was slightly improved in that it was normothermic and could hold its head up for very brief periods. The owners elected to pursue CT. Therefore, the colt received midazolam (0.25 mg/kg [0.11 mg/lb], IV) and butorphanol (0.01 mg/kg [0.0045 mg/lb], IV) and underwent CT, with images acquired before and after administration of iodinated contrast medium (ioversol; 350 mg I/mL; 2.2 mL/kg [1 mL/lb], IV; **Figure 1**).

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

This report was submitted by Silke Hecht, Dr med vet; Melissa T. Hines, DVM, PhD; Federica Morandi, DVM, MS; and Linden E. Craig, DVM, PhD; from the Departments of Small Animal Clinical Sciences (Hecht, Morandi), Large Animal Clinical Sciences (Hines), and Biomedical and Diagnostic Sciences (Craig), College of Veterinary Medicine, University of Tennessee, Knoxville, TN 37996.

Address correspondence to Dr. Hecht (shecht@utk.edu).

Diagnostic Imaging Findings and Interpretation

A peripheral, crescent-shaped, hyperattenuating lesion (68 HU; reference range, approx 25 to 30 HU for white matter and 35 to 50 HU for gray matter of clinically normal brains) was identified between the osseous boundaries of the skull and adjacent brain parenchyma on the right side **(Figure 2)**. The rostrocaudal extent of the lesion spanned along the right frontal, parietal, and temporal lobes to the level of the occipital lobes, and the dorsoventral extent spanned from the level of the falx cerebri to the presphenoid, basisphenoid, and basioccipital bones. There was a mass effect beyond this lesion indicated by compression and displacement of the right lateral ventricle and a midline shift toward the left. The white matter tracts of the right cerebral hemisphere were mildly and diffusely hypoattenuating (18 HU), compared with those of the left hemisphere (23 HU). No fractures were identified. On reconstructed sagittal plane CT images (not shown), there was no evidence of foraminal or transtentorial brain herniation. Following administration of contrast medium, no abnormal contrast enhancement was identified. These findings were consistent with an extensive subdural hematoma along the right hemisphere of the brain combined with an associated mass effect and vasogenic brain edema.

Treatment and Outcome

Supportive care for the colt continued with fluid therapy as previously mentioned, placement of a nasogastric tube for feedings, regular repositioning to avoid pressure sores, passive range-of-motion exercises, and administration of 20% mannitol (250 mL, IV over 1 hour, q 8 h), gentamicin (6.6 mg/kg [3 mg/lb], IV, q 24

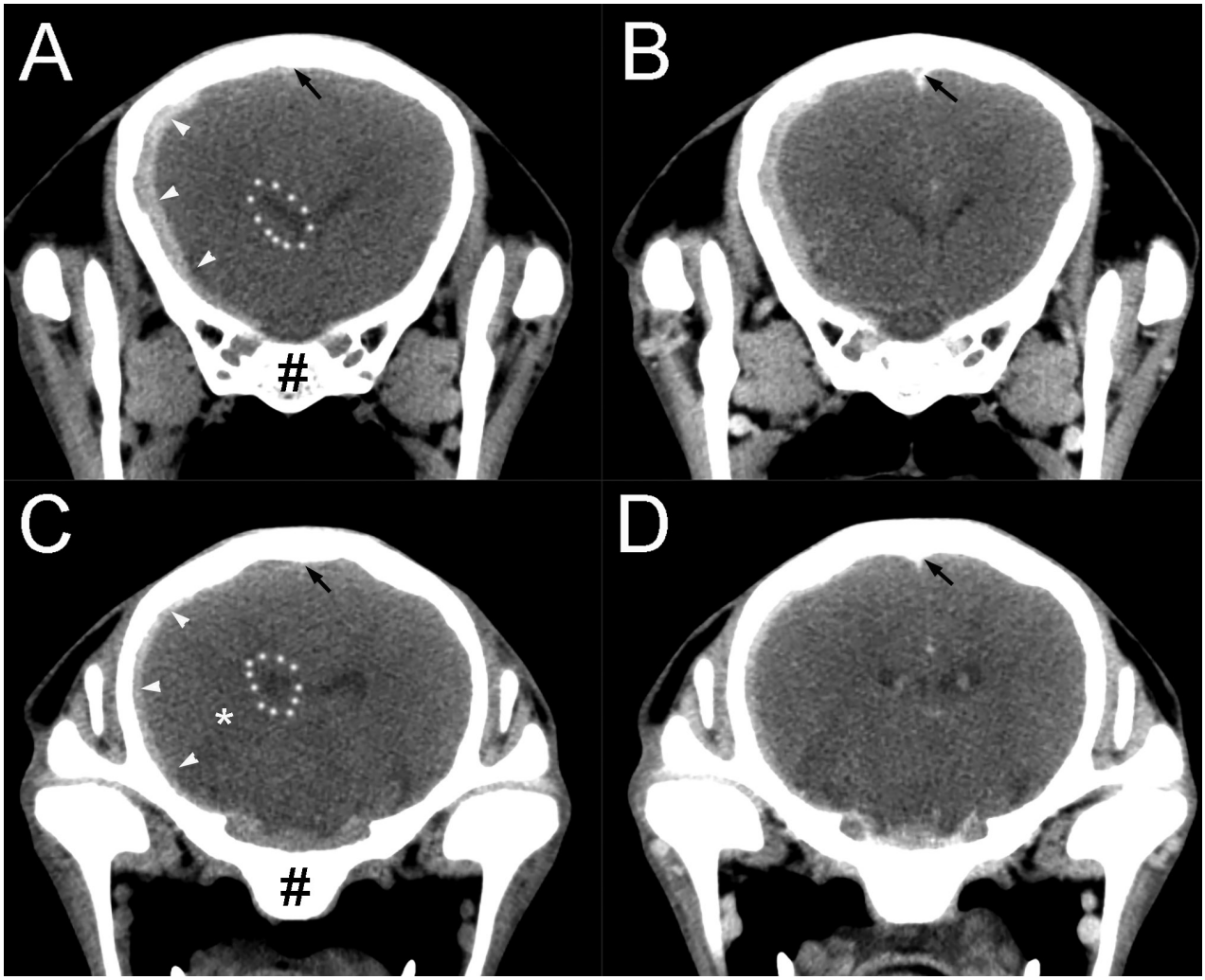

Figure 2—Same CT images as in Figure 1. There is a peripheral crescent-shaped lesion (arrowheads) between the osseous boundaries of the skull and adjacent brain parenchyma. The lesion is hyperattenuating (68 HU; reference range, approx 25 to 30 HU for white matter and 35 to 50 HU for gray matter of clinically normal brains) and extends dorsoventrally from the level of the falx cerebri (black arrows) to the right lateral aspect of the presphenoid bone (pound sign). There is compression and displacement of the right lateral ventricle (within the dotted ovals) and a midline shift toward the left, consistent with a mass effect in the right cerebral hemisphere. The white matter tracts of the right cerebral hemisphere (asterisk) are mildly and diffusely hypoattenuating (18 HU), compared with those of the left hemisphere (23 HU), consistent with vasogenic brain edema. No traumatic bone abnormalities are evident.

h), and cefazolin (11 mg/kg [5 mg/lb], IV, q 8 h). The colt improved, and on day 3 of hospitalization, administration of mannitol was discontinued, nasogastric-tube administration of sucralfate (18 mg/kg [8.2 mg/lb], q 12 h) and vitamin E powder (1,000 U, q 24 h) dissolved in 15 mL of water was initiated, and standing up the colt several times daily to further help avoid tendon contracture was initiated. On day 6, minocycline (4 mg/kg [1.8 mg/kg]) dissolved in 15 mL of water and administered through the nasogastric tube every 12 hours replaced IV administration of antimicrobials. The colt primarily received nasogastric-tube feedings until day 15, when it started nursing from the mare. By 3 weeks after admission, the colt could get up and lie down unassisted, drank milk from a pan, ate softened hay, and was bright, alert, responsive, and playful. However, during week 4 of hospitalization, the colt showed sudden severe signs of colic that were nonresponsive to pain management. Shortly thereafter, the colt had a seizure followed by cardiopulmonary arrest and died, despite CPR efforts.

Necropsy results indicated that the subdural hematoma detected with CT had largely resolved but that the colt had severe chronic multifocal encephalomalacia, consistent with prior trauma. Additionally, the colt had colonic hypoplasia and small intestinal distension, consistent with ileus and the signs of colic observed. Grossly, the abnormally small colon suggested colonic hypoplasia from colonic aganglionosis or lethal white foal syndrome; however, histologic examination revealed that the ganglia and neurons within the intestines were adequate.

Comments

Head trauma is common in horses, especially those < 12 months of age, and often results from a horse rearing and flipping over onto its poll or from struggling after being trapped under a gate or fence.[1] When skull fractures are present, they may be diagnosed with radiography; however, when a fracture does not explain the clinical signs observed, there is no fracture, or radiographic diagnosis of fractures is difficult, especially owing to anatomic complexity, advanced imaging with CT or MRI is often needed.[1–3] Because CT of horses typically requires the use of general anesthesia, case selection should be with caution because of the inherent risks of anesthetic drugs and recovery in neurologically impaired animals. In severely obtunded patients, such as the colt of the present report, anesthesia may not always be necessary.

In our experience, CT is preferred for evaluating horses with acute head trauma because the procedure is quick and highly accurate in detecting fractures, intracranial hemorrhage, brain edema, and brain herniation. On CT, areas of acute intracranial hemorrhage are hyperattenuating, compared with the patient's unaffected brain parenchyma,[4,5] as was evident on CT images for the colt of the present report. Additionally, CT is useful in localizing intracranial hemorrhage, which may affect therapeutic options and prognosis. For instance, the peripheral, crescent-shaped, hyperattenuating lesion identified in the colt of the present report represented a subdural hematoma, which is usually caused by tearing of subdural veins and is an accumulation of blood in the potential space between the superficial dura mater and the deeper arachnoid membrane. This accumulation of blood may cross suture lines but is limited by the falx cerebri and the osseous tentorium. In contrast, an epidural (or extradural) hematoma is typically caused by laceration of a meningeal artery by a skull fracture, and blood accumulates in the potential space between the inner surface of the skull and the dura mater, forming a peripheral, focal, biconvex mass that may cross dural folds, such as the falx cerebri and tentorium cerebelli, but not suture lines.

In the colt of the present report, the white matter tracts of the right cerebral hemisphere were mildly and diffusely hypoattenuating (18 HU), compared with those of the left hemisphere (23 HU). This finding was consistent with vasogenic brain edema, an extracellular fluid accumulation that results in swelling and decreased CT attenuation of cerebral white matter, often with finger-like projections along white matter tracts.[4,6] The pathophysiologic processes of post-traumatic brain edema are complex and multifactorial,[7] and affected horses generally have a poor prognosis.

Treatment for horses with head injuries generally consists of supportive care. Risk factors associated with nonsurvival to hospital discharge in horses following traumatic brain injury include recumbency of more than 4 hours' duration after initial evaluation and fractures of the basilar bone.[1] In the present report, it was unexpected that, 4 weeks after the trauma and subsequent improvements, the colt would suddenly develop acute colic and die; these developments could have been related to prolonged hospitalization or colonic hypoplasia identified on necropsy. In people, gastrointestinal problems (eg, intestinal dysmotility) associated with traumatic brain injury have been reported.[8] Similarly, yet speculatively, the brain injury identified on CT and encephalomalacia diagnosed on necropsy of the foal of the present report could have contributed to its gastrointestinal signs and death.

References

1. Feary DJ, Magdesian KG, Aleman MA, et al. Traumatic brain injury in horses: 34 cases (1994–2004). *J Am Vet Med Assoc* 2007;2:259–266.
2. Ragle CA, Koblik PD, Pascoe JR, et al. Computed tomographic evaluation of head trauma in a foal. *Vet Radiol* 1988;29:206–208.
3. Anderson JM, Hecht S, Kalck KA. What Is Your Diagnosis? Skull fractures in a foal. *J Am Vet Med Assoc* 2012;241:181–183.
4. Hecht S. Brain. In: Schwarz T, Saunders J, eds. *Veterinary computed tomography*. Ames, Iowa: Wiley-Blackwell, 2011;185–196.
5. Parizel PM, Makkat S, Van Miert E, et al. Intracranial hemorrhage: principles of CT and MRI interpretation. *Eur Radiol* 2001;11:1770–1783.
6. Du Plessis V, Gaillard F. Cerebral edema. Radiopaedia. Available at: radiopaedia.org/articles/cerebral-oedema-1?lang=us. Accessed Nov 2, 2019.
7. Jha RM, Kochanek PM, Simard JM. Pathophysiology and treatment of cerebral edema in traumatic brain injury. *Neuropharmacology* 2019;145:230–246.
8. Patel JJ, Rosenthal MD, Miller KR, et al. The gut in trauma. *Curr Opin Crit Care* 2016;22:339–346.