What Is Your Diagnosis?

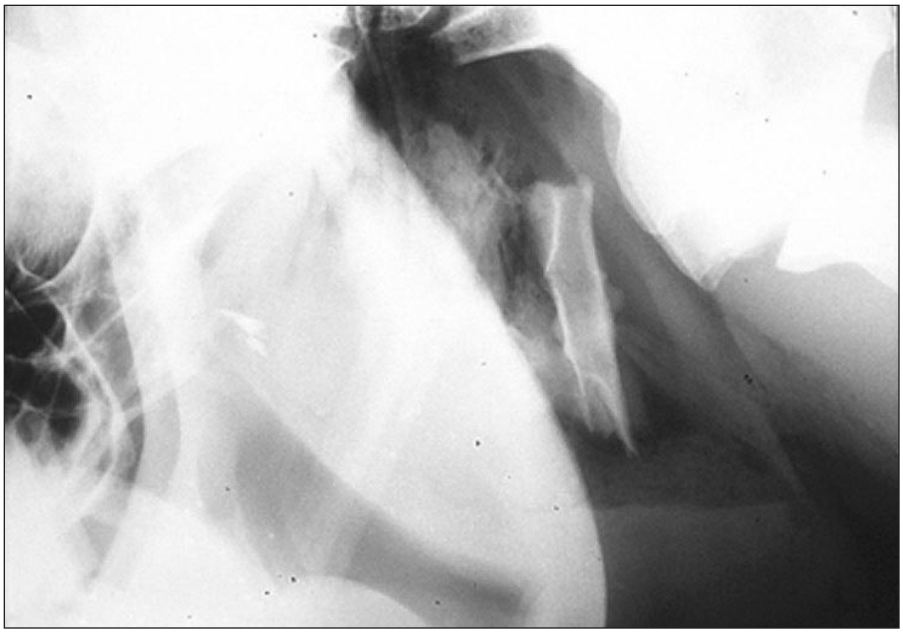

Figure 1—Lateral radiographic view of the skull of a horse with acute head trauma and severe bilateral epistaxis.

History

A 9-year-old Arabian stallion was evaluated because of acute head trauma. Two hours prior to evaluation, the horse became excited during a halter class, reared, and fell over, landing on the left side of its head. Immediately after the accident, the horse remained in lateral recumbency for several minutes and then moved to sternal recumbency without any apparent neurologic deficits or change in consciousness. Once the horse was standing, severe bilateral epistaxis began.

On admission, severe bilateral epistaxis was still evident. Heart rate, respiratory rate, and rectal temperature were within reference limits, and abnormalities were not detected during the initial neurologic examination. Radiographs of the skull were obtained (**Fig 1**).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page ▶

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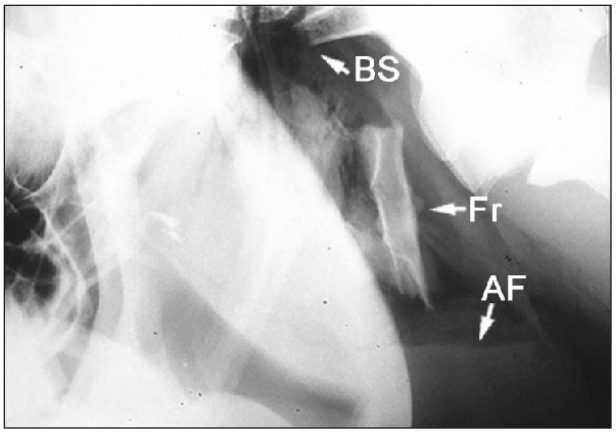

Figure 2—Same radiographic view as in Figure 1. Notice the basisphenoid-basioccipital fracture (BS) and displaced bony fragment (Fr). An air-fluid (AF) interface is also visible.

Diagnosis

Radiographic diagnosis—Displaced avulsion fracture of the basisphenoid-basioccipital bone **(Fig 2)**.

Comments

The horse was treated conservatively for an open skull fracture. Epistaxis resolved later that day but was observed intermittently for 2 weeks thereafter. Trimethoprim-sulfadoxine (24 mg/kg [11 mg/lb] of body weight, IV, q 12 h) and dexamethasone (0.15 mg/kg [0.07 mg/lb], IV, once; then 0.1 mg/kg [0.05 mg/lb], IV, q 12 h) were administered, and the horse was confined to a stall. A partial seizure resolved after treatment with diazepam (10 mg/kg [4.5 mg/lb], IV, once), phenobarbital (10 mg/kg [4.5 mg/lb], IV infusion over 30 minutes; followed by 5 mg/kg [2.3 mg/lb], PO, q 12 h), and DMSO (1 mg/kg [0.5 mg/lb], 10% solution in lactated Ringer's solution, IV, q 24 h for 2 days). A brief period of ataxia developed 6 days after admission but resolved without treatment. Skull radiography performed on days 8, 21, and 31 revealed ventral rotation of the fragment and gradual reabsorption of fluid within the auditory tube diverticula (guttural pouches). Three mild episodes of colic developed within the first 3 weeks following head trauma; all responded to medical treatment. The horse was discharged from the hospital on day 35.

The horse was readmitted 3 days after discharge (38 days after the initial injury) for surgical treatment of severe impaction colic. After surgery, the horse continued to have episodes of colic while hospitalized, and a nasogastric tube was placed. Following removal of the tube, the horse became dysphagic and acutely dyspneic. Endoscopy of the upper airways revealed severe pharyngeal swelling and bilateral arytenoid paresis. Endoscopy was repeated several times while the horse was hospitalized, and during the last endoscopy 68 days after the initial injury, the guttural pouches were also examined. The septum dividing the guttural pouches was not apparent, and a mucosa-covered mass protruded cranially from the caudal aspect of the pouches **(Fig 3)**. This mass corresponded to the displaced bony fragment of the basisphenoid bone. Skull radiography repeated at the same time revealed little change in the position of the fragment. The horse remained hospitalized for 52 days because of recurrent impaction colic. Approximately 2 months after the second discharge and 6 months after the initial injury, the owners elected to euthanatize the horse because of severe colic. Necropsy revealed a large-colon torsion, but the skull was not examined.

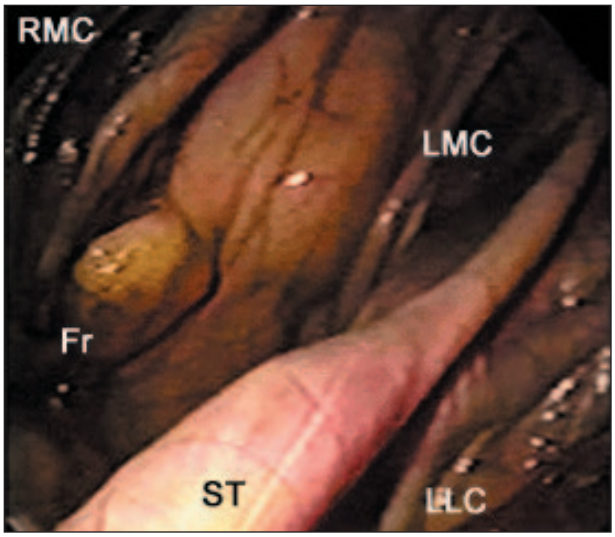

Figure 3—Endoscopic view of the upper airways of the horse depicted in Figure 1, obtained 2 months after the initial injury. A mass (Fr), presumed to be the displaced bony fragment identified on the original radiograph, is evident protruding from the caudal aspect of the guttural pouches. RMC = Right medial compartment. LMC = Left medial compartment. LLC = Left lateral compartment. ST = Left stylohyoid bone.

Initial radiographic abnormalities of basilar skull fractures may be subtle.[1] Typically, these fractures are minimally displaced and result in only an irregularity or step in the sphenoccipital suture line.[2] Radiographic confirmation of a basilar skull fracture may be obtained several weeks after the initial injury as new bone production becomes evident. In some horses, swelling of the retropharyngeal tissues attributed to hemorrhage may be apparent immediately after injury. Free gas may also be seen within the cranial vault.[2] In the horse described in this report, displacement of a large fragment of the basisphenoid bone was obvious on the initial radiograph. Although atypical, such severe displacement exemplifies the avulsive forces that contribute to this type of fracture.

In horses with a nondisplaced basisphenoid fracture, endoscopy will not reveal bony abnormalities. In this horse, though, the displaced fragment was evident. Although nerves within the guttural pouches did not appear damaged, it is possible that recurrent impaction colic was attributable to vagal nerve damage and altered gastrointestinal tract motility. Unfortunately, a thorough necropsy was not performed to confirm such a connection.

References

1. Ragle C. Head trauma. *Vet Clin North Am Equine Pract* 1993;9:171–183.

2. Ramirez O, Jorgensen JS, Thrall DE. Imaging basilar skull fractures in the horse: a review. *Vet Radiol Ultrasound* 1998;39:391–395.