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CASE REPORT

Horses and other equids

Basisphenoid bone fracture in two juvenile horses with different clinical presentation

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[Correction added on 27 July 2021, after first online publication: the order of author names was updated.]

Abstract

Basilar skull fractures in horses can occur at any age, although young horses are particularly prone to this type of injury. This manuscript describes two foals with a basisphenoid bone fracture, with clinical presentations ranging from mild neurological signs up to severe neurological compromise. The absence of neurological signs does not exclude a fracture of this bone, as lethargy and retropharyngeal swelling may be the only clinical signs. Radiography can help define skeletal trauma involving the basilar region of the head, but superimposition of complex skull anatomy limits its use. Endoscopy of the guttural pouches region can be also helpful, but it does not provide enough evidence for a diagnosis. Thus, computed tomography is the optimal imaging technique for a rapid and straightforward diagnosis, as it is superior to radiography defining the type of fracture and the location of the fragments as well as the soft tissue involvement.

BACKGROUND

Basilar skull fracture (BSF) occurs most commonly after a horse rears over backwards and strikes the ground with the poll of the head.[1,2] Hyperextension of the head leads to contraction of the longus capitis muscle, with the potential for a resultant fracture with or without displacement of fragments of the basisphenoid and basioccipital bones.[3,4] Although the traumatic event can occur at any age, young horses are particularly prone to this type of injury as they are more likely to resist head restraint.[5] Clinical signs are highly variable and the extent of structural damage can be difficult to determine clinically.[6] BSFs have a poor prognosis and the diagnosis by radiographic examination is difficult, especially if there are no displaced bone fragments.[7–9] Neurologic damage can be the result of either direct trauma by displaced bone fragments or intracranial haemorrhage, inflammation and secondary brain injury.[1] The enhanced resolution and sensitivity of CT make it the ideal imaging modality for the diagnosis of skull fractures in horses.[8,10] Compared to radiography, it also provides the possibility to precisely locate the fracture site within specific areas of the base of the skull. To date, there is a limited number of published studies or case reports of BSF in horses. This article describes the clinical presentation of two foals with a basisphenoid fracture, their diagnostic imaging findings and outcome.

CASE PRESENTATION

Case 1

A rescued 8-month-old crossbred colt, approximately 250 kg bodyweight, was referred to Unitat Equina-Fundació Hospital Clínic Veterinari, Universitat Autònoma de Barcelona. Previous to referral, the colt felt over backward during the oral administration of an anthelmintic drug. On admission, the foal showed lethargy and an inspiratory rasping noise. On complete neurological examination, no other deficits were observed.

Radiographs of the skull were taken under sedation, using a fixed system generator (SHF 835; SEDECAL, Algete, Madrid, Spain), and an X-ray tube (Varian G-292; Varex Imaging Corporation, Las Vegas, NV, USA) at a film focal distance of 100 cm. Left to right latero-lateral and dorsoventral views of the base of the skull were obtained using a Konica Minolta Regius model 170 computed radiology (CR) equipment (Konica Minolta). On the lateral view, two osseous fragments, 4.5 × 11.6 mm and 16.3 × 8.6 mm were seen just ventral to the basioccipital bone (Figure 1a). Soft tissue opacification of the guttural pouches and ventral deviation of the dorsal pharyngeal wall were also observed. On a dorsoventral view, a 2 × 9.5 mm fragment displaced to the right side and a lack of alignment between the basisphenoid and the

basioccipital bone was observed (Figure 1b). Endoscopy of the upper airways revealed severe oedema and hyperaemia of the nasopharynx and it was impossible to gain entrance to the guttural pouches. BSF was strongly suspected and CT of the skull was performed under general anaesthesia with the animal positioned in dorsal recumbency. The CT scan was performed using a 16 slices scanner (Brivo CT385; General Electric Healthcare, Chicago, IL, USA) and reviewed in bone and soft tissue windows. The scan parameters were 120 kV and 159 mA, and slice thickness of 1.25 mm and a gap of 0.6 mm. CT findings revealed a severely comminuted fracture of the basisphenoid-basioccipital junction, with a large osseous fragment (11.5 × 7.2 mm in the dorsal plane) displaced to the left side, and a second fragment (8.2 × 3.6 mm in the dorsal plane) displaced to the right side, with a displacement of the basioccipital bone to the right of the sagittal plane (Figure 2). A soft tissue attenuating structure was detected in the guttural pouches area, compatible with haemorrhage or hematoma. A diagnosis of BSF was reached.

The colt was hospitalized and treated with oral suxibuzone (3.3 mg/kg every 12 hours) and intravenous antimicrobial therapy with sodium penicillin (22000 UI/kg every 6 hours) and gentamicin (6.6 mg/kg every 24 hours). The clinical progression was good, the inspiratory stridor disappeared and the mental status improved over the first week of hospitalisation. The foal was discharged 2 weeks after admission with a recommendation to continue stall rest, avoidance of manipulation of the head and continuation of therapy with antibiotic and NSAID for another 2 weeks. Upon re-evaluation, 1 month later, neurological examination was normal, except for a mild right head tilt detectable while eating. Radiographic re-evaluation of the skull revealed callus bone formation at the fracture point. No obliteration of the guttural pouches or displacement of the dorsal pharyngeal wall were observed (Figure 3). Nine months after referral, the head tilt had resolved and the animal had atrophy of the left masseter muscle attributed to neurological atrophy likely caused by a lesion of the left mandibular nerve. No other deficits of the neurological examination were observed. Eighteen months after referral, the owner reported a good outcome with subtle remaining difficulties when eating.

Case 2

A 10-month-old Crossbred filly, approximately 400 kg bodyweight, was referred with a history of a fall over backwards a few hours prior to admission. Since the fall, the filly remained recumbent, unconscious and with continuous bilateral spontaneous horizontal nystagmus. The referring veterinarian administered intravenously a dose of 4 ml/kg 7.5% hypertonic saline solution, flunixin meglumine (0.5 mg/kg) and dexamethasone (0.1 mg/kg). The filly was referred under general anaesthesia with intravenous ketamine (1.1 mg/kg) and diazepam (0.5 mg/kg). On admission, the animal was unconscious and, when the effects of anaesthesia waned, the filly remained stuporous with persistent bilateral continuous horizontal, vertical and rotatory spontaneous nystagmus. Pupillary light reflexes were present but the pupils were anisocoric, with mydriasis of the right pupil. No clear attempts to stand or gain the sternal position were observed.

Lateral and dorsoventral radiographs of the skull were taken using the equipment and techniques described above. On the lateral view, a fracture at the basisphenoid-basioccipital junction was suspected (Figure 4a). On the dorsoventral view, an 11.5 × 8.9 mm osseous fragment was seen on the left side of the basisphenoid-basioccipital suture. Partial soft tissue opacification of the guttural pouches was also present (Figure 4b).

A CT of the skull was performed under general anaesthesia with the filly positioned in dorsal recumbency. The scan parameters were 120 kV and 35 mA, and slice thickness of 1.25 mm and a gap of 0.6 mm. Iobiditriol, an iodinated non-ionic contrast (Xenetix 300 mg iodine/ml; Laboratorios Farmacéuticos Guerbet, Madrid, Spain) was injected via intra-arterial 16G BD-Angiocath catheter (BD, Eysins, Nyon, CH) in the right common carotid artery using a pressure injector (Nemoto A-60; Nemoto Kyorindo Company, Tokyo, Japan). As previously described,[11] a continuous arterial infusion protocol during scanning was used with an injection rate of 2 ml/s (100 ml of contrast injected). The post-contrast scan was initiated with a 3 seconds delay from the start of the contrast medium administration.

CT findings revealed a severely comminuted fracture of the basisphenoid-basioccipital physeal junction (Figure 5). A large fragment of 23.3 × 7.6 mm was located in the right ventrocaudal aspect of the basisphenoid bone. Multiple fragments were located in the left dorsolateral aspect of the basioccipital bone. A soft tissue attenuating structure was detected in the left guttural pouch, compatible with haemorrhage or hematoma. The diagnosis of BSF was confirmed. Due to the poor prognosis and the absence of good clinical progression, the filly was euthanized. A complete post-mortem examination was not performed.

LEARNING POINTS/TAKE HOME MESSAGES

- BSF should be suspected in horses with a history of a fall over backwards.
- The absence of signs of intracranial trauma does not exclude a fracture at the basisphenoid-basioccipital suture. Lethargy and retropharyngeal swelling may be the only presenting clinical signs.
- CT is superior to radiography in defining the type of fracture and the location of the fragments as well as the soft tissue involvement.
- Delayed lesions of the cranial nerves may occur.

DISCUSSION

In the present case report, both horses were diagnosed with a basisphenoid fracture. Despite having similar fracture locations, clinical signs varied significantly, ranging from moderate to profound neurological signs. The two cases showed deficits of the cranial nerves at some point of the clinical progression. Case 2 had apparent involvement of either cranial nerve (CN) III or VIII, whereas case 1 developed atrophy of the left masseter muscle consistent with a delayed onset lesion of the mandibular branch of the trigeminal nerve (CN V). There is a paucity of reports on the neurological signs associated with BSF in horses. Furthermore, cranial nerve

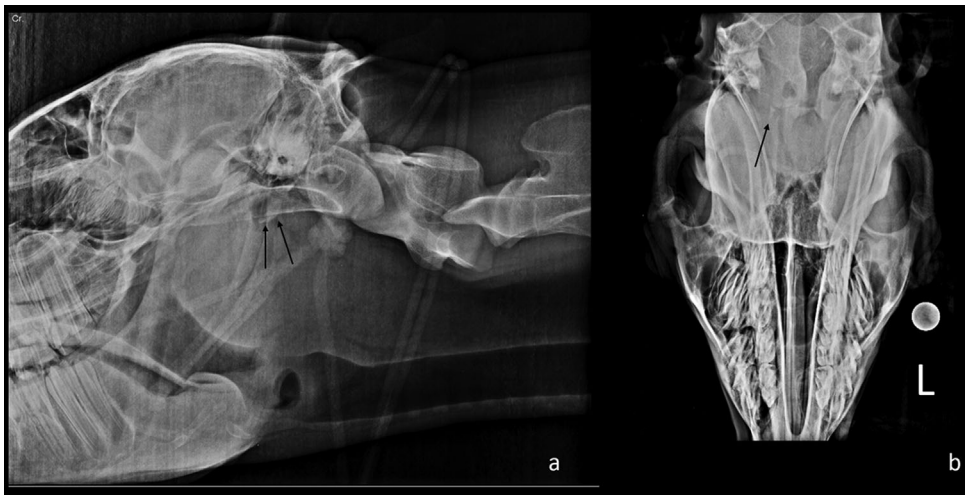

FIGURE 1 Radiographic projections of the skull of case 1: (a) latero-lateral view in which two osseous fragments ventral to the basioccipital bone are observed (arrows); (b) dorsoventral view in which a bone fragment and a lack of alignment between the basisphenoid and the basioccipital bone is observed (arrow). Almost complete soft tissue opacification of the guttural pouches is also present

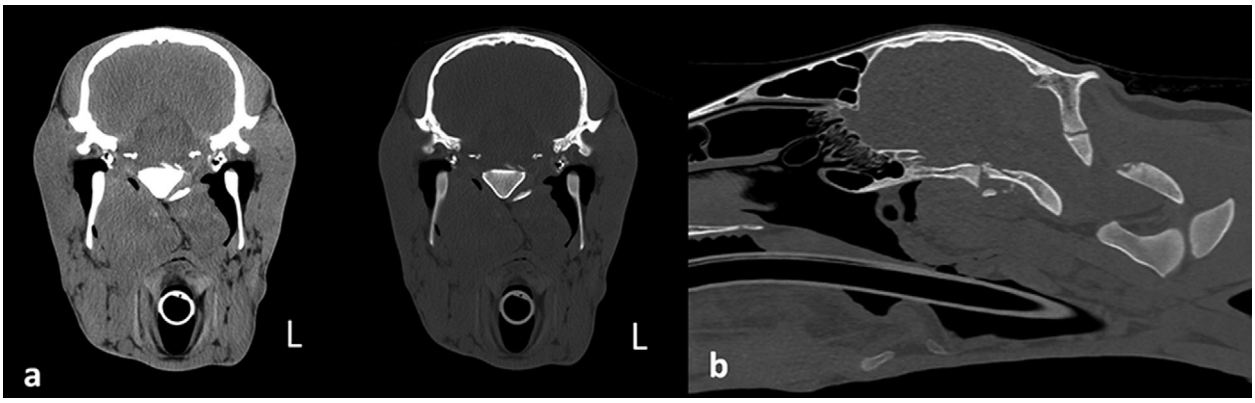

FIGURE 2 Slices of CT scan study of the head of case 1: (a) transverse views – on the left, the soft tissue algorithm andon the right, the osseous algorithm. The left side of the patient is on the right side of the images; (b) sagittal slice in the bone window. The two biggest fragments of the comminuted fracture of the basisphenoid-basioccipital physeal junction are evident in these images

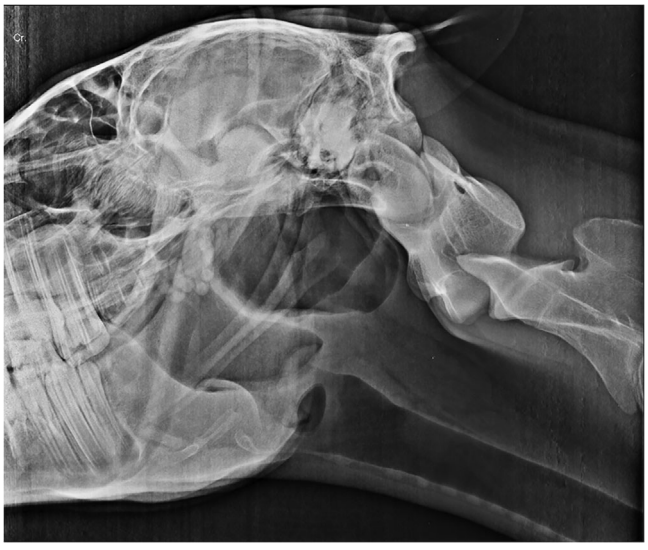

FIGURE 3 Latero-lateral radiographic projection of the skull of case 1 after 1-month progression. Note the callus bone formation at the fracture point. No obliteration of the guttural pouches or displacement of the dorsopharyngeal wall were observed

dysfunction is not described in most of the reports of BSF, due to the inherent difficulty in performing a full neurological examination in this subset of equine patients. There is one report of a horse with optic nerve damage and blindness secondary to an anterior skull fracture confirmed by CT scan.[6] Recently, a case of a horse with a posterior BSF with vagal nerve paralysis and suspected implication of the glossopharyngeal nerve has been described.[12] Horses included in the present case report showed variable clinical signs, all potentially related to the skull fracture and intracranial inflammation. Moreover, case 1 showed mild neurological signs and a good outcome, which differs from the common perception of poor prognosis related to skull trauma. In particular, this case showed a very exceptional clinical outcome. As mentioned, the colt showed left mandibular nerve deficits (i.e. neurogenic masseter atrophy) 9 months after the acute trauma. The delayed involvement of this particular nerve is uncommon. The mandibular nerve emerges from the foramen lacerum,[13,14] a triangular hole in the base of the skull, located between the sphenoid, the apex of the petrous temporal and the basilar part of the occipital bones. Therefore, signs of mandibular nerve lesion might have been caused by bone remodelling observed on the radiologic follow-up in this area.

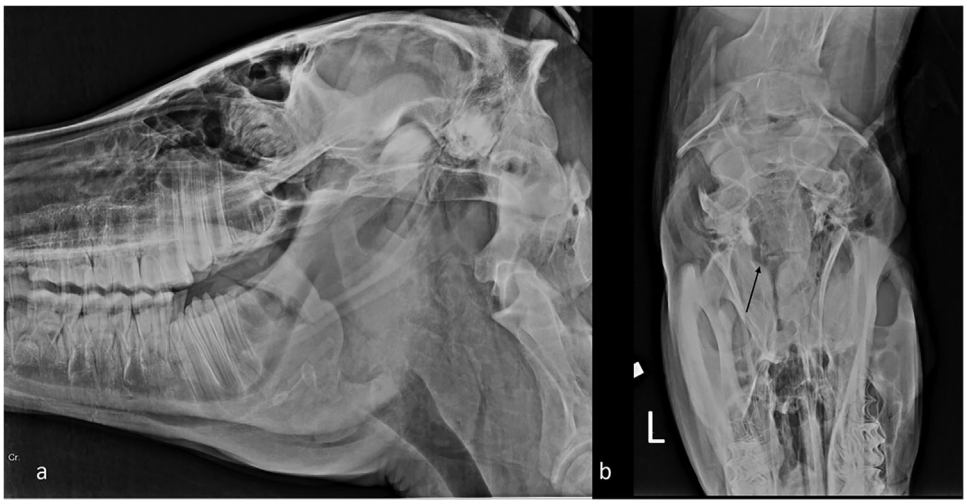

FIGURE 4 Radiographic projections of the skull of case 2: (a) latero-lateral view; (b) dorsoventral view in which an osseous fragment is seen on the left side of the basisphenoid-basioccipital suture (arrow). Moreover, partial soft tissue opacification of the guttural pouches is also present

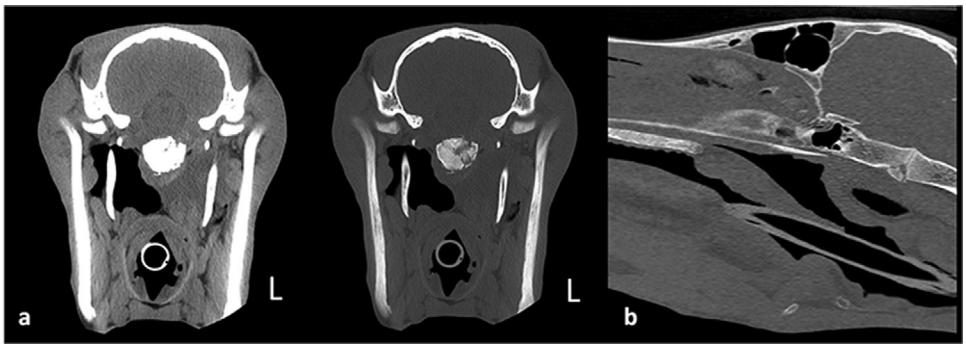

FIGURE 5 Slices of CT scan study of the head of case 2: (a) transverse views– on the left, the soft tissue algorithm andon the right, the osseous algorithm. The left side of the patient is on the right side of the images; (b) sagittal slice in the bone window. A severely comminuted fracture of the basisphenoid-basioccipital physeal junction is observed

Trauma to the head, with resultant damage to intracranial structures, is not uncommon in the horse.[3] The available literature is limited but indicates that the prognosis is guarded.[3,5,8,9] Subdural haemorrhage, epistaxis, severe neurological signs and compression of the laryngeal and pharyngeal area leading to asphyxiation are all potential sequelae of a BSF.[15] Bilateral mydriasis has also been reported as a sign of severe midbrain injury and as an indicator of poor prognosis.[4] BSF usually occurs at the weakest point, which in the case of foals, is the point of junction of the basisphenoid and the basioccipital bones.[3,7] This is the area in which fracture occurred in the two cases herein reported.

Haemorrhage at the level of the pharynx and guttural pouches can be secondary to either rupture of the rectus and longus capitis muscles and their associated vasculature,[15] or can also originate from the vasculature and venous sinuses associated with the roof of the guttural pouches.[8] In case 2, a soft tissue attenuating structure was detected on the CT scan within the left guttural pouch, compatible with haemorrhage or hematoma formation. Partial soft tissue opacification of the guttural pouches on radiographic examination was also present in case 1. An endoscopic examination can aid in the diagnosis of haemorrhage associated with BSF.[15] During the endoscopy examination of case 1, it was not possible to enter the guttural pouches due to soft tissue oedema and deformation of the nasopharynx. However, the degree of detail of the haemorrhage on CT in case 2 allowed for better characterisation of the soft tissue swelling and haemorrhage.

Radiographs of the basisphenoid and basioccipital bones in young horses are particularly hard to evaluate and easily misinterpreted because the suture line between these two bones remains open until 5 years of age.[1,5,8,9,16] Even when the suture line has closed, it may appear irregular in both shape and width. This makes it very hard to visualize a fracture, especially if it is not displaced.[8] Some authors have concluded that survey radiography can help define skeletal trauma involving the basilar region of the head, but superimposition of skull structures combined with the clinician's inherent inability to discriminate small differences in tissue density limit the use of radiography.[8,17] Thus, CT should be considered the optimal imaging technique for a rapid and straightforward diagnosis.[18,19] Disadvantages of the CT technique are related to the financial cost, need of adequate facilities and trained personnel and general anaesthesia of an already compromised patient unless standing CT is available. The advantages include the provision of detailed images without superimposition of anatomical structures and the ability to provide a high degree of soft-tissue contrast resolution.[13,14,18]

Here are described two cases with a basisphenoid fracture with differing clinical signs. Lack of neurological signs caused

by a BSF may be due to absent or minimal compression of the CNS. Case 1 showed only upper airway obstruction signs and the imaging findings confirmed extensive haemorrhage and soft tissue swelling of the guttural pouches and pharyngeal area. The location and topographical anatomy of the fracture as well as the severity of the injury and secondary brain damage, all contributed to the myriad of clinical signs manifested in the cases described here. If the injury is located most ventrally to the basisphenoid-basioccipital suture the prevalent signs will be epistaxis and asphyxia, while if the cerebral parenchyma is injured neurological signs will be prevalent.[8,9]

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