CASE REPORT

Traumatic basilar artery entrapment with patency of pontine perforators and absence of significant brainstem infarction: report of an unusual case

Stefan Kliesch, Christian Bauknecht, Georg Bohner, Thomas Liebig, Eberhard Siebert

Department of Neuroradiology,
Charité Universitätsmedizin
Berlin, Berlin, Germany

Correspondence to

Dr Stefan Kliesch, Department
of Neuroradiology, Charité
Universitätsmedizin Berlin,
Charitéplatz 1, Berlin 10117,
Germany;
stefan.kliesch@charite.de

Accepted 4 August 2016

SUMMARY

We report a rare case of entrapment of the basilar artery into the sphenoid bone caused by a longitudinal fracture of the clivus. Using high resolution three-dimensional flat panel angiography, we show preservation of the basilar artery perforators in spite of severe stenosis of the entrapped segment of the basilar artery. There were no obvious signs and symptoms of posterior fossa stroke clinically or radiographically as far as could be assessed under given clinical circumstances.

BACKGROUND

Basilar artery (BA) entrapment into a clival fracture is a very rare condition and only a few cases have been reported in the literature. As indicated by previous reports, the condition portends a poor prognosis with a high morbidity and mortality, especially because of posterior fossa stroke. Using three-dimensional flat panel angiography (3D-FPA), we were able to show preservation of brainstem perforators in the entrapped segment of the BA and lack of major posterior stroke in follow-up CT examinations. This finding may have prognostic value, although our case resulted in death because of severe supratentorial traumatic brain damage and its resulting complications.

CASE PRESENTATION

A previously healthy young adult was involved in a high velocity motor vehicle accident as a pedestrian. On arrival of the emergency team, the patient scored 11 on the Glasgow Coma Scale. Frontal head and face trauma was obvious with exophthalmos, scalp laceration and cephalic

haematoma. The patient was brought into our emergency department where he was intubated for safety and diagnostic reasons. A whole-body CT performed on admission as part of the trauma protocol showed a dislocated and dura-perforating fracture of the frontal bone including the frontal sinuses and left orbit. The fracture continued into the skull base and extended in a longitudinal pattern into the clivus ([figure 1](#)). CT angiography (CTA) showed adherence of the artery to the bone with a concomitant high-grade irregular stenosis and subtle signs of focal herniation into the sphenoid sinus ([figure 1](#)). Furthermore, there were extensive traumatic subarachnoid haemorrhages and large bifrontal contusions, but no brainstem or cerebellar infarction was evident on CT ([figure 1](#)). The patient was brought to the operating room for treatment of the dislocated skull and orbital fractures. In addition, an intracranial pressure monitoring tube was installed. To evaluate the impact of the BA pathology and the blood supply of the brainstem, we decided to perform conventional angiography including high-resolution 3D-FPA. BA entrapment was confirmed and we could clearly show the prolapse of the opacified middle portion of the BA into the sphenoid sinus over a distance of about 8 mm with its proximal start just distal to the ostium of the anterior inferior cerebellar artery. There was associated high-grade stenosis distal and proximal to the prolapse. Nevertheless, numerous perforator branches were passing the fracture line within the clivus and were seen to extend to the middle and upper portion of the pons ([figures 2 and 3](#)). Furthermore, the right superior cerebellar artery was seen to fill antegradely from the BA.

Figure 1 (A) CT volume reconstruction showing a displaced and multifragmented frontal fracture. (B) CT scan showing extensive frontal contusions and subarachnoid haemorrhage. (C) CT angiography showing subtle herniation of the basilar artery into the sphenoid sinus.

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To cite: Kliesch S, Bauknecht C, Bohner G, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-012558

Figure 2 (A) Left internal carotid artery injection showing collateral flow via the posterior communicating artery into the upper basilar trunk, both posterior cerebral and the left superior cerebellar artery. (B) Vertebral artery run showing pseudoocclusion of the basilar artery within the fracture cleft as well as distal filling of the basilar artery and several brainstem perforators within the sphenoid sinus re-entering the posterior fossa via the fracture cleft.

No unequivocal major perfusion deficit could be outlined with the head of the BA and the left superior cerebellar artery being perfused by a strong posterior communicating artery on the left. Furthermore, we did not see any signs of a traumatic arteriovenous fistula. Follow-up CT on day 2 showed enlarging bifrontal contusions and subarachnoid haemorrhage accompanied by generalised brain swelling. Consequently, anti-oedematous therapy was applied. Further follow-up imaging including unenhanced CT, CTA and CT perfusion in the setting of persistent high intracranial pressure values was performed on day 3, which showed even more progressive brain contusions as well as signs of diffuse axonal injury. There was no evidence of a significant brainstem infarction clinically or radiographically as far as could

be assessed in this clinical situation and the imaging methods applied. However, a new anterior cerebral artery infarct was seen on the left, most likely secondary to subfalcine herniation. The clinical course was complicated by persistently elevated intracranial pressure, for which bifrontal decompressive craniectomy was performed on day 4. In addition, transcranial duplex sonography showed increased blood flow velocities in the intracranial arteries. On follow-up angiography for significant vasospasms, no significant changes were seen regarding the prolapsed segment of the BA, although vasospasm also affected the BA tip including the proximal segments of the posterior cerebral arteries (figure 4) as well as significantly prolonged perfusion of the left middle cerebral artery territory. Anti-vasospasmic medication (Nimotop) was given via a nasal tube. Nevertheless, the patient developed nearly complete infarction of the left hemisphere and died due to subfalcine herniation and mesencephalic compression on the ninth day of admission. Even on the final CT, no brainstem or cerebellar infarction was visible.

DISCUSSION

BA entrapment in the presence of a clivus fracture is a very rare condition. Only a few cases have been reported in the literature.¹⁻⁷ As indicated previously, this condition is usually accompanied by massive traumatic brain injuries and indicates poor prognosis with high morbidity and mortality.¹⁻⁵ To the best of our knowledge, the present case of BA entrapment with prolapse of the entrapped segment into the sphenoid sinus is the first to demonstrate persistent patency of the BA perforator branches of the involved segment as demonstrated by high resolution 3D-FPA. In line with this finding, our patient did not develop frank clinical or imaging signs of brainstem or cerebellar infarction. Although, admittedly, CT is of limited value to

Figure 3 High-resolution flat-panel three-dimensional rotational angiography. (A) Fracture course in the central skull base resulting in a longitudinal median clival fracture. (B) Herniated basilar artery in the sphenoid sinus. (C, D) Patency of brainstem perforators arising from the trapped basilar artery segment and coursing through the fracture cleft.

Figure 4 (A) Follow-up CT shows enlarged contusions, signs of elevated intracranial pressure and herniation despite decompressive craniectomy. However, no ischaemic infarcts secondary to basilar artery pathology could be detected in the posterior fossa. (B) Follow-up angiography shows severe proximal vasospasm.

exclude posterior fossa stroke and is inferior to diffusion-weighted MRI in this type of stroke, it remains the method of choice to monitor critically ill patients for obvious reasons. Our case is in contrast to most other reports that describe significant posterior fossa infarctions.^{4–5} Differences in location and length of the entrapment, the variable spatial configuration, the variability of the circle of Willis as well as the direct effect on brainstem perforators seem to be discriminating factors in the development of infarctions in this situation. Although there is no reasonable neurosurgical or endovascular cure for this specific condition, detailed knowledge of the vascular and perfusion status has prognostic value, especially if MRI is not possible.

Bala and colleagues reported a case of BA entrapment resulting in a high-grade BA stenosis due to a longitudinal clival fracture following a motorcycle accident with good neurological outcome.⁶ In the 3D time-of-flight MR angiography images provided in their report, both posterior communicating arteries were patent and only a circumscribed mesencephalic infarct developed. It can therefore be assumed that most of the perforators were still in a functional condition. Fang and colleagues presented a case of BA herniation with a good outcome and complete clinical recovery at 20 months although, in the 6-month control, formation of a dissecting pseudoaneurysm was observed.⁷

In view of these cases and our own report, it has to be kept in mind that, even though BA injury usually seems to have a paramount prognostic impact, the injury pattern of a longitudinal clival fracture is almost invariably the result of a high impact

trauma to the head with further associated significant brain injury. In cases of frontal or facial impact like the one described here, the force transmitted in the anterior–posterior direction creates a transient coronal expansion of the skull resulting in a longitudinal clival fracture, while the inertance of the skull contents causes a relative anterior movement of the brainstem and thus the BA that may result in incarceration of the artery in a transiently clefting clival fracture.⁷ We hypothesise that, in our case, the patient died primarily from massive frontal brain contusions, herniation-associated infarctions, traumatic subarachnoid haemorrhage, vasospasm and ultimately critically elevated intracranial pressure despite decompressive craniectomy rather than from BA entrapment and its possible ischaemic sequelae. If, on the other hand, angiographically-proven occlusion of the BA and the originating perforators is evident, the overall prognosis may be driven by inevitable brainstem infarctions. However, standard imaging protocols including digital subtraction angiography may not be sufficient to reliably discriminate between these scenarios.

While high-resolution 3D-FPA including secondary reconstruction may certainly not be able to reliably demonstrate all perforators or exclude occlusions of single perforators, it may be helpful in differentiating between patients who may potentially recover with extensive treatment such as decompressive craniectomy and those who will suffer from major brainstem infarction independent of their additional injuries, especially if MRI is not possible.

Competing interests None declared.

Provenance and peer review Not commissioned; externally peer reviewed.

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