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Case Report

Basilar artery incarceration secondary to a longitudinal clivus fracture: A rare and favorable outcome of an often devastating injury

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ABSTRACT

Background: Clival fractures are a rare traumatic finding and are often the result of high-impact craniofacial trauma. Rarely, longitudinal clival fractures can be associated with incarceration of the basilar artery within the fracture and/or the sphenoid sinus. Of the 12 reported cases of basilar incarceration, 11 of these injuries have proved to be fatal due to pontine infarction. We present a patient with basilar artery incarceration without any neurologic deficits.

Case Description: The case reported is a 17-year-old male who presented after a motor vehicle collision with a linear and longitudinal clival fracture with entrapment of the basilar artery within the sphenoid sinus. Diagnostic subtraction angiography showed a small intimal tear with possible intraluminal thrombus. The patient was started on aspirin and at 3-month post injury had no neurologic deficits.

Conclusion: Basilar artery incarceration is an injury often associated with pontine infarction secondary to basilar artery dissection and/or thrombus developing at the site of entrapment. Our case illustrates a favorable outcome after this injury. Based on these results, antiplatelet therapy may be a viable option for prevention of brainstem infarcts in patients with this injury; however, further prospective studies must be done to assess the overall efficacy and validity of this treatment. There are no established treatment guidelines for this condition. Further research on this topic should also be tailored toward early identification of this pathology and prevention of thromboembolic sequelae of this injury.

Keywords: Basilar artery entrapment, Basilar artery dissection, Clival fracture, Trauma

INTRODUCTION

Clival fractures are a rare traumatic finding and are often the result of high-impact craniofacial injuries. The incidence of these injuries in the literature ranges from 0.6% to 1.2%.[11] Clival fractures can be classified into one of three types — longitudinal, transverse, and oblique.[12] Of the subtypes, longitudinal fractures are the only reported fractures to be associated with basilar artery dissections and, rarely, basilar artery incarceration. To the best of our knowledge, there are 12 reported cases in the literature of basilar artery incarceration within a clival fracture. The majority of these injuries were fatal and/or associated with pontine infarction.[1,3,4,5,7,8] We report a case of a 17-year-old male who presented after motor vehicle collision with a longitudinal clival fracture and basilar artery incarceration without brainstem infarction or neurologic deficits.

CASE DESCRIPTION

Patient is a 17-year-old male who was the driver in a motor vehicle collision. On presentation, he had a Glasgow Coma Score (GCS) of 6T – intubated, no eye opening, localizing bilateral upper extremities to the central painful stimulus without any weakness, and pupils equally round and reactive to light. Trauma computed tomography (CT) scans revealed a linear, non-displaced longitudinal clival fracture, right frontal and left parietal subdural hematomas, suprasellar and interpeduncular subarachnoid hemorrhage, bilateral maxillary bone fractures, right orbital wall fracture, and C2 hangman's fracture [Figure 1].

Given his low GCS < 8, an external ventricular drain and Licox monitor were placed the day of admission. Given the cervical spine and clival fractures, CT angiogram of the head-and-neck was obtained. CT angiogram showed herniation of the basilar artery into the sphenoid sinus through the longitudinal basilar skull fracture. The basilar artery had some mild stenosis in the mid-basilar section next to a presumed intimal tear and possible intraluminal thrombus [Figure 2]. Diagnostic catheter angiogram was performed to evaluate the stenosis and thrombus. Imaging showed that there were no filling defects, dissection flaps, nor any thrombus throughout the vertebrobasilar system [Figure 3]. There was high-grade stenosis at the entry point of the basilar artery into the clivus for which he was placed on Aspirin 81 mg. Low-dose aspirin was chosen over 325 mg due to the patient's extensive intracranial hemorrhages. One-week post injury day, the patient underwent a C2-C4 fusion for his unstable C2 fracture. He was discharged to an outside facility on day 18 of hospitalization. At the time of discharge, he was extubated, without cranial nerve deficits, and had full strength in all his extremities. Six weeks after his injury, he was seen in outpatient follow-up where he reported intermittent blurry vision but had no other significant complaints. For these concerns, he was referred to ophthalmology; however, he did not follow-up with them due to resolution of his symptoms. Repeat CT angiogram of the head at 1-month post injury demonstrated unchanged findings. The patient is currently doing well on aspirin and is awaiting his 6-month repeat CTA. At his follow-up visit, the necessity of continued aspirin therapy will be determined based on his CT angiogram findings.

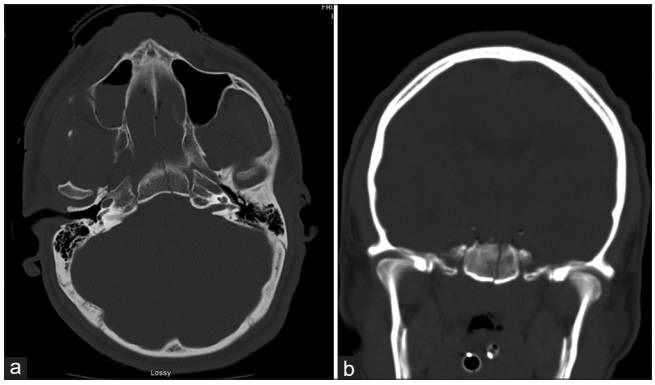

Figure 1: Axial (a) and coronal (b) noncontrasted CT imaging of the head illustrating a longitudinal fracture through the clivus.

DISCUSSION

This case illustrates a rare injury — entrapment of the basilar artery within a longitudinal clival fracture. To the best of our knowledge, there are 12 reported cases of basilar artery incarceration within the literature.[11] The most commonly reported complication of this injury is pontine infarction secondary to basilar artery dissection and/or thrombus development at the site of incarceration. Because of this, the majority of these cases have had poor neurologic and functional outcomes.

The earliest suggested mechanism for this injury was reported by Sights in 1967.[9] This case involved a 23-year-old male who was immediately unresponsive after a motor vehicle collision. The patient had multiple pontine infarctions secondary to an occluded basilar artery which had herniated through the fracture defect. In the mechanism that they described, there are several necessary forces required for this to occur. First, there must be a high impact frontal collision causing a longitudinal fracture of the clivus with associated laceration of the dura. Second, due to inertial forces, the brainstem and cerebellum herniate anteriorly with respect to

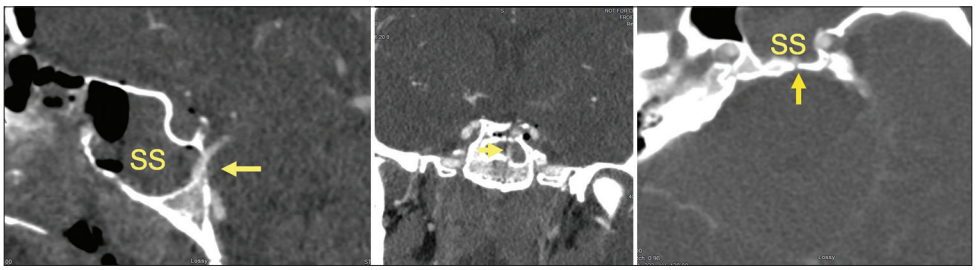

Figure 2: CT angiography of the head with sagittal, coronal, and axial views, respectively, from left to right. Sphenoid sinus (SS) and arrows identify the basilar artery encased within the fracture line within the sphenoid sinus.

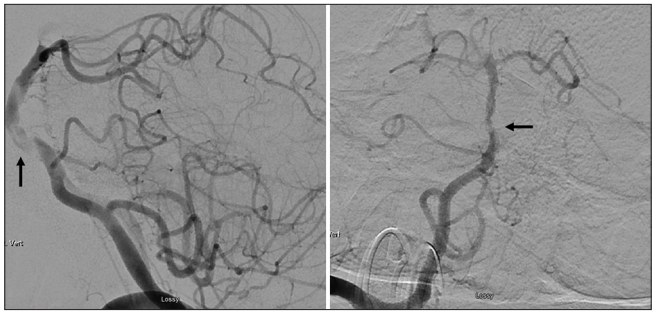

Figure 3: AP and lateral digital subtraction angiogram of the vertebrobasilar system through the right vertebral artery injection. Arrows identify the area of stenosis of the basilar artery where it enters the clival fracture.

the skull, resulting in herniation of the basilar artery into the fracture. After this herniation event, the fracture then closes on the artery after cessation of movement which can result in the development of a thrombus and subsequent pontine infarction.

Of the 12 reported cases in the literature, all the patients developed pontine infarctions or cranial nerve palsies. The time to infarction has been variable with evidence of neurological decline anywhere from immediately post injury to up to 48 h afterward. The best reported outcome, to the best of our knowledge, was reported by Bala *et al*. in 2003.[2] In this case, the patient initially presented with a GCS of 15 and had a left abducens palsy. Within 12 h post injury, he subsequently developed left hemiparesis and was found to have multiple pontine and medullary strokes on magnetic resonance imaging (MRI). At 10-month post injury, his abducens palsy had resolved and he had a trace amount of the left distal leg weakness.

Specific guidelines for the management and treatment of this condition have not been established. With respect to nonoperative management in our case, we placed the patient on Aspirin immediately after the diagnosis was established. The decision to start either anti-platelet and/or anticoagulation in this situation is difficult. First, the efficacy of antiplatelet therapy in this case is unclear. There have been patients who have been managed similarly with antiplatelet therapies with variable success.[2,10] However, it is important to note that many of these patients who were started on Aspirin already had evidence of pontine infarction before establishment of the diagnosis. In addition, given the high impact nature of this injury, many of these patients often have concomitant intracranial hemorrhages, potentially making initiation of antiplatelet therapy high risk. From an endovascular standpoint, there has been one reported case where endovascular treatment of a basilar artery occlusion secondary to incarceration was attempted. However, this resulted in an arterial perforation due to the significant stenosis caused by the clival fracture.[6]

There are several factors which may have contributed to this patient's favorable outcome. First, the patient's injury was diagnosed early. He underwent CT angiogram within 3 h of presentation which demonstrated the injury. At that time, there was no appreciable thrombus within the basilar artery — only extrinsic compression. Diagnostic catheter angiography within 4 h from his presentation showed only basilar stenosis at the site of the artery herniating into the clivus. In the majority of cases, basilar artery incarcerations happen to be identified after the patient has already had a clinical neurological decline and/or has identifiable strokes on MRI. Finally, the early initiation of aspirin may have played a role in this favorable outcome by decreasing his thromboembolic complication rate associated with the stenosis.

The only other documented case of basilar artery incarceration with an objectively favorable long-term outcome was identified by Bala *et al*. in 2003 and had two similarities with our case. First, in both cases, the patient's injury was identified early and they initially presented with a favorable GCS. Second, antiplatelet therapy was started immediately after diagnosis of the basilar artery incarceration. The patient was started on Aspirin at a dose of 100 mg daily. In their case, the patient had a significant neurological decline within 12 h after presentation. However, despite this decline, the patient did make a substantial recovery. Starting antiplatelet therapy early may have prevented further thromboembolic sequelae and/or pontine infarction; however, we are unable to objectively prove this retrospectively. It is also possible that the patient's young age may have played a role in his overall favorable outcome as was the case with our patient. However, given the rarity of this injury and retrospective nature, we are unable to prove that either of these variables are protective factors.

CONCLUSION

Incarceration of the basilar artery is a severe traumatic finding with potentially devastating neurologic sequelae. This injury is the result of a high speed and high impact mechanism and can ultimately result in brainstem infarction relatively quickly. The majority of patients with this injury do poorly; however, there are a few cases with favorable outcomes. Our case presents a 17-year-old male who presented with basilar artery incarceration without any apparent brainstem infarction who was shortly treated with Aspirin after identification of the injury and at 3-month post injury has not had any neurologic sequelae from his accident. Based on this outcome, antiplatelet therapy may be a viable option for prevention of brainstem infarcts in patients with this injury; however, further studies must be done to assess the overall efficacy of this prophylactic treatment. Unfortunately, there are currently no established treatment guidelines for

this condition. Further literature on this topic should be tailored toward early identification of this potential injury and prophylactic treatment to prevent brainstem infarction in patients with basilar artery incarceration.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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