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Case Report

Delayed presentation of a traumatic scalp arteriovenous fistula

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ABSTRACT

Background: Arteriovenous (AV) fistulas of the scalp are extracranial vascular malformations commonly caused by trauma and typically present within 3 years. Although they follow a benign course, they can be esthetically displeasing.

Case Description: We present an atypical onset of scalp AV fistula in a patient with a 1-year history of the left-sided pulsatile tinnitus and scalp swelling 7 years after a traumatic epidural hematoma evacuation. Our patient was found to have an 8 mm AV fistula supplied by the deep temporal artery. Endovascular embolization was performed using eight coils. There was no complication from the procedure, and the patient's pulsatile tinnitus and swelling resolved immediately after embolization. Follow-up angiogram demonstrated complete obliteration of the AV fistula.

Conclusion: Delayed presentation of traumatic scalp AV fistula is very rare, and it is important to keep this in the differential in patients with scalp swelling after head trauma.

Keywords: Arteriovenous fistula, Head trauma, Iatrogenic fistula, Neuroendovascular

INTRODUCTION

Subcutaneous scalp arteriovenous (AV) fistulae are uncommon vascular lesions that occur spontaneously, iatrogenically, or following trauma. These lesions typically do not pose life-threatening risks to the patient. However, they can be cosmetically disfiguring and cause significant distress. The common presentations are headache, pulsatile mass, and pulsatile tinnitus.[1-3] A recent systematic review by Sofela *et al*. identified 242 cases in which spontaneous, traumatic, and iatrogenic etiologies comprised 60.2%, 32.3%, and 7.5%, respectively.[8] Traumatic AV fistulae typically present within the first 3 years after the inciting event. To the best of our knowledge, there has only been one report of a traumatic AV fistula occurring after 7 years.[2] We present a delayed presentation of a scalp AV fistula 7 years following a traumatic epidural hematoma evacuation.

CASE PRESENTATION

Our patient is a 16-year-old male who presented to the neurosurgery clinic with a 1-year history of progressive left scalp swelling and tinnitus. He had a history of trauma 7 years prior resulting in

a left 2.5 cm temporal epidural hematoma [Figure 1a] which required evacuation [Figure 1b] and a left posterior petrous temporal bone fracture. There was no scalp swelling in the immediate postoperative period. Seven days after the trauma, he presented to the emergency department with drowsiness, left cranial nerve VI palsy, and papilledema. He was found to have a left sigmoid, jugular bulb, and internal jugular venous thrombosis likely secondary to the overlying skull fracture. Thus, the patient was treated with 4 months of Lovenox® (enoxaparin) and 1 month of Diamox (acetazolamide).

On presentation, our patient had an intact neurological examination including cranial nerve and motor and sensory systems. There was no papilledema. Fullness was noted over the left forehead and temporal area. A tortuous vein was seen over the lateral aspect of the left eyebrow [Figure 2a and b]. A large dilated compressible area over the left temporalis incisional flap measuring 7–8 mm was noted. A loud bruit was present over this area on auscultation and a thrill on palpation. No chemosis or proptosis was noted.

A computed tomography angiography (CTA) of the head was obtained that demonstrated numerous dilated scalp veins overlaying the temporalis muscle and extending into the face [Figure 2c and d]. A four-vessel angiogram was performed and showed a scalp AV fistula supplied by the deep temporal arteries with collateral flow to the fistula from the superficial temporal artery and the ophthalmic artery. There was no evidence of intracranial venous drainage but there was significant hypertrophy of the deep temporal arteries supplying the fistula to a large venous pouch on the patient's scalp [Figure 3].

The AV fistula was embolized using a total of eight Penumbra smart coils® (Penumbra, Inc.; Alameda, CA). Four coils were deployed inside of venous pouch. One traveled distally into the venous pouch and three landed proximally. After deployment of these coils, there was good distal occlusion in the venous pouch. The microcatheter was then pulled back slightly until it landed at the fistula site. An additional four coils were deployed at this site. Follow-up angiogram showed no residual filling of the fistula [Figure 3].

The postoperative course was unremarkable without complications from the coil embolization procedure. The engorged facial veins were no longer visible. At 1-month follow-up, the patient denied any tinnitus, headaches, blurry, or double vision. No engorged facial veins were visible at the time. A 6-month postoperative angiogram was performed that demonstrated successful obliteration of the scalp AV fistula [Figure 4]. The deep temporal and superficial temporal arteries were no longer feeding the fistula.

DISCUSSION

There are two proposed mechanisms for traumatic AV fistulae. The laceration theory describes direct communication of a lacerated artery and venous pouch that developed from the incident.[6,7] The second mechanism is the disruption of the vasa vasorum, which results in endothelial budding and formation of several arterial vascular channels.[4] The mechanism in our case likely falls within the laceration theory, because there was only one main arterial feeder. Another possibility is due to the increased venous pressure

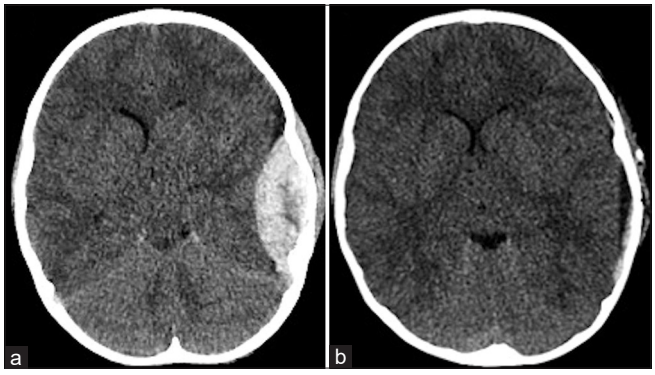

Figure 1: Initial head CT after trauma 7 years before AV fistula development. (a) L temporal epidural hematoma with 0.5 cm midline shift. (b) Postoperative CT scan demonstrating evacuation of EDH and improvement of midline shift.

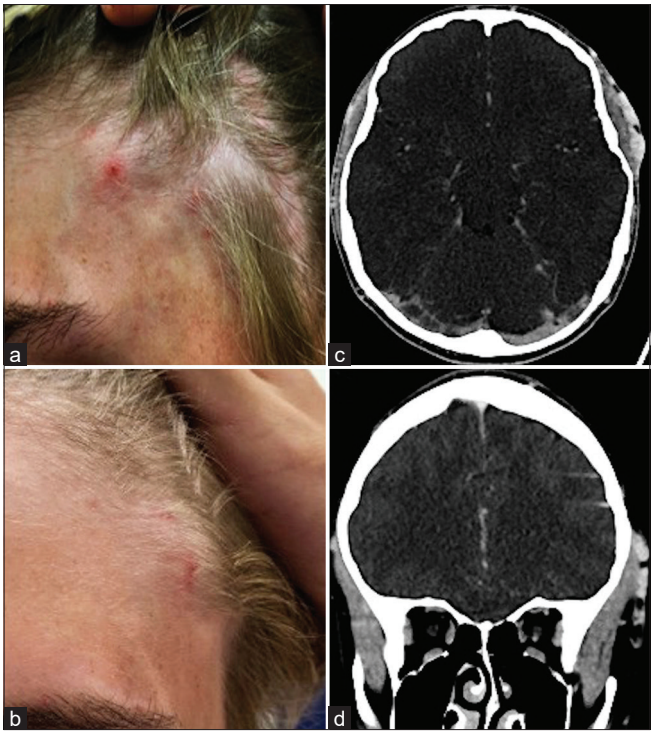

Figure 2: Engorged left temporal vessels 7 years following traumatic epidural hematoma evacuation. (a and b) Clinical presentation of the left anterolateral scalp swelling. (c and d) CTA head 7 years after initial trauma demonstrated scalp engorged vessels.

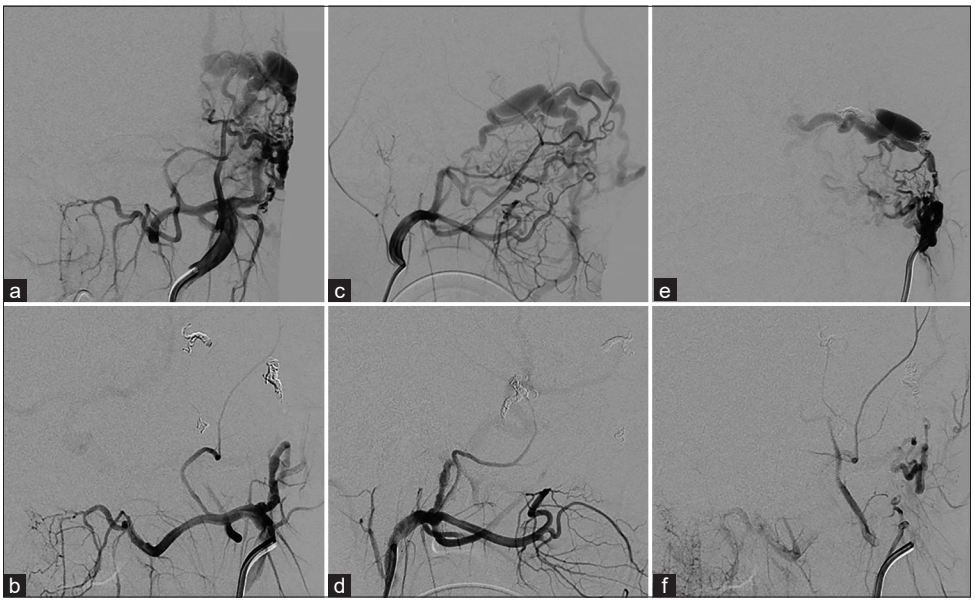

Figure 3: Pre- and post-embolization angiogram. (a, b, c) AP, lateral, and selective internal maxillary angiogram demonstrating the deep temporal arterial feeders, venous pouch, and venous drainage. (d, e, f) Postoperative angiogram demonstrating obliteration of the AV fistula after embolization.

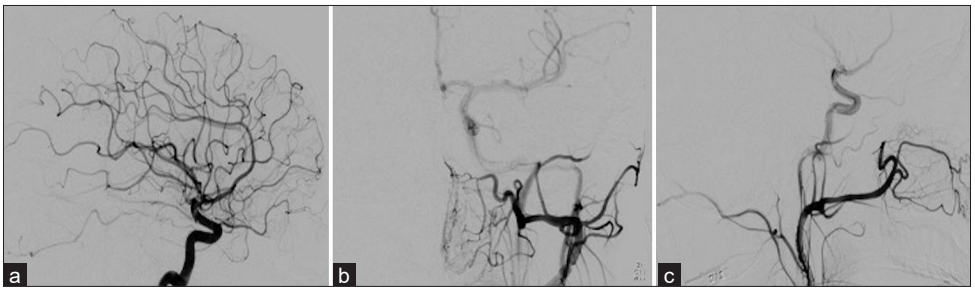

Figure 4: Six-month follow-up angiogram. (a) Lateral image of the left ICA. (b) AP image of the left internal maxillary artery (IMA). (c) Oblique view of IMA.

from the sinus thrombosis suffered by our patient. However, this is unlikely, since we did not observe any intracranial fistulae, feeders, or venous engorgement. Moreover, the sinus thrombosis was medically treated and intracranial pressures normalized, which was evident by resolve of papilledema. The inciting trauma was likely from surgery since there was no scalp hematoma noted on the initial head CT. Although placing a clip around the superficial temporal artery could theoretically prevent scalp AV fistulae, this is not common in our practice given the rarity of these postoperative complications.

Our case presents a unique situation because of the delayed presentation 7 years after initial trauma and prior surgery in the region of the AV fistula. The delay in presentation was likely due to the relatively smaller size of deep temporal arteries compared to the superficial temporal and occipital arteries, which are more common feeders. We predict that this would result in slower reconstruction, enlargement, and development of swelling from the AV fistula.

Diagnostic workup is critical to define the management of AV fistula. Recent systematic reviews have developed diagnostic and treatment algorithms for these vascular pathologies depending on size and arterial feeders.[8,9] CTA or MRI/MRA aids in identifying the vascular lesion and localizing it anatomically. Angiography is necessary to characterize a vascular lesion particularly to check for intracranial arterial feeders and number of feeding vessels. This will also help differentiate it from a slow flow pathology such as lymphatic or venolymphatic malformation. Our workup consisted of a head CTA to initially diagnose the malformation and study its relationship to the skull and intracranial compartment. A diagnostic angiogram was then performed to define the size, arterial blood supply, and venous drainage allowing us to clearly diagnose the AV fistula. The two main treatment options are surgery, intra-arterial embolization, or a combination of both.

We performed endovascular embolization, because surgery carried increased risk for wound healing and infection

since there is poorer vascular supply from prior surgery. Paradoxically, other literature reports endovascular treatment carries a higher risk of scalp necrosis.[5] Nonetheless, we believe that it was safest to do endovascular embolization of the AV fistula. To avoid skin necrosis, our technique consisted of deploying the coils within the venous pouch in the distal and proximal portion. Hence, arterial branches from the arterial side of AV fistula should not be occluded with this strategy. We successfully embolized the scalp AV fistula. The patient had immediate relief of symptoms and at 6-month follow-up, there were no signs of recurrence or tissue necrosis.

CONCLUSION

This report highlights that traumatic scalp AV fistulas may present years after the initial injury, and it is incumbent on the treating physician to include this in the differential diagnosis. Typical presenting symptoms include headache, scalp swelling, pulsatile tinnitus, chemosis, and proptosis. Angiography is the gold standard for diagnoses. Endovascular embolization is a safe and effective treatment of these lesions. We recommend it for small lesions in a previously operated area to reduce wound healing problems. Moreover, deploying the coils or other embolization material at the pathology will reduce the risk of scalp necrosis. One of the limitations of this report is that not all of published case reports/series include the time to onset of symptoms from the inciting event and some report time of symptom duration before presenting for treatment. This could be from limited resources, symptom tolerance, or misdiagnoses.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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