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Case Report

Postcraniotomy superficial temporal artery pseudoaneurysm in the setting of triple H therapy: A case report and literature review

Sergei Terterov, Nancy McLaughlin, Neil A. Martin

Department of Neurosurgery, David Geffen School of Medicine at UCLA, 757 Westwood Plaza, Suite 6236, Los Angeles, CA, USA

E-mail: * Neil A. Martin - neilmartin@mednet.ucla.edu; Nancy McLaughlin - nancymc50@hotmail.com; Sergei Terterov - sterterov@mednet.ucla.edu
Corresponding author

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Abstract

Background: Superficial temporal artery (STA) pseudoaneurysm after a craniotomy is very rare with only five cases reported in the literature, none manifesting in the setting of cerebral vasospasm treatment with triple H therapy.

Case Description: A 31-year-old male was admitted after a syncopal episode. Imaging documented a ruptured anterior communicating artery aneurysm. He was taken to the operating room for aneurysm clipping, but the procedure was aborted due to intraoperative aneurysm re-rupture, at which point the patient underwent emergent coil embolization of the aneurysm. The postoperative course was complicated by severe cerebral vasospasm requiring prolonged triple H therapy. On postoperative day 22, a growing left temporal mass with a bruit was noted. The suspected diagnosis of STA pseudoaneurysm was confirmed by femoral angiography, and it was treated with coils and Onyx embolization.

Conclusion: We report the first case of a postcraniotomy STA pseudoaneurysm in the setting of induced hypertension for the treatment of cerebral vasospasm. Endovascular embolization is a viable option for the treatment of an STA pseudoaneurysm.

Key Words: Craniotomy, pseudoaneurysm, superficial temporal artery, vasospasm

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INTRODUCTION

Since its first description in 1740 by Bartholin, pseudoaneurysms of the superficial temporal artery (STA) have been reported in about 400 cases worldwide.[14,20] Most patients are asymptomatic presenting with a painless, pulsatile, and expanding mass on the affected temporal area.[4] It is most commonly caused by blunt trauma to the temporal region of the head.[11] Other less frequent etiologies include penetrating scalp injuries, hair grafting, temporomandibular joint surgery, dental surgery, parotid surgery, Gardner traction, and internal carotid artery (ICA) ligation.[2,13,14] STA pseudoaneurysm after a craniotomy is very rare with only five cases reported in the literature and one additional case after ventriculostomy placement.[1,3,12,16,18,21] We report the first case of a postcraniotomy STA pseudoaneurysm in the setting of induced hypertension for the treatment of cerebral vasospasm.

CASE REPORT

A 31-year-old male, otherwise in good health, experienced a sudden onset of severe headache 5 days prior to admission. After experiencing a nonspecific lightheadedness, he was brought to an outside hospital.

Initial head computed tomography (CT) scan showed a subarachnoid hemorrhage in the basal cisterns, most extensively in the interhemispheric fissure, suggestive of a ruptured aneurysm. Upon arrival the patient was normotensive and had a nonfocal neurological exam. Magnetic resonance angiography (MRA) and computed tomography angiography (CTA) documented the presence of an elongated aneurysm arising from the anterior communicating artery. The aneurysm measured 6.2 mm in its greatest dimension and was directed superiorly. A second pouch measuring 2 mm projected inferiorly. Both studies documented multifocal narrowing of bilateral A1 segments, suggestive of angiographic vasospasm. The left A1 was dominant. Therapeutic options were presented to the patient including coil occlusion and surgical clipping. Given the patient's young age, the morphological characteristics of the aneurysm, and the risk of coil compaction over time, surgical treatment was elected.

The patient underwent a left fronto-orbital craniotomy to access the anterior communicating artery ruptured aneurysm. A lumbar subarachnoid drain was inserted prior to the craniotomy for intraoperative drainage of cerebrospinal fluid (CSF) and subsequent brain relaxation. After administration of mannitol, and CSF drainage, the sylvian fissure was dissected distally to proximally, following the proximal M1 segment toward the ICA. At that point, blood spontaneously filled the basal subarachnoid spaces, suggesting aneurysm re-rupture. Barbiturates were administered and burst suppression was rapidly achieved. The arterial blood pressure was lowered to 70 mmHg systolic. Although the bleeding stopped, the brain was swollen, obliterating the subdural and subarachnoid spaces, and bulged from the craniotomy. The temporalis muscle, galea, and scalp were closed in layers without replacing the bone flap. Intraoperative CT scan revealed evidence of new blood in the interhemispheric fissure without any intraparenchymal hematoma.

The patient underwent an urgent cerebral angiogram. In addition to the two lobulations documented preoperatively, there was an additional lobulation, likely representing the rupture site. Both A1s, right more so then left, were spastic. The aneurysm was almost completely occluded with Guglielmi detachable coils (GDC), leaving only partial filling at the base of the aneurysm, which is protective in the acute period.[22] Immediately after the coiling, an external ventricular drain was inserted to aid in the management of intracranial pressures.

Throughout his postoperative course, daily transcranial Doppler studies (TCDs) were performed to follow arterial velocities. On postoperative day 4, pharmacological angioplasty with intraarterial verapamil injection in both ICAs and left A1 was performed. Although improved, the TCDs remained elevated, and the patient remained on aggressive triple H therapy maintaining blood pressures above 180 mmHg systolic. On postoperative day 22, a mass was noted in the left temporal region as seen in Figure 1. A head CT scan was performed initially and showed a partially hypodense-isodense fluid-filled mass just superior to the zygoma and adjacent to the lower part of the craniotomy site [Figure 2a]. The following day (POD23), the mass had progressed in size and revealed avascular bruit on auscultation and arterial pulsation on the duplex Doppler. The magnetic resonance imaging/magnetic resonance angiogram (MRI/MRA) showed a temporal mass isointense on T1WI, heterogeneous on T2WI with pulsation artifacts and homogeneous enhancement [Figure 2b and c]. The suspected diagnosis of an STA pseudoaneurysm was confirmed by transfemoral angiography [Figure 2d and e]. Interventional approach was elected and the STA was occluded with coil embolization and sealed with liquid Onyx (ev3 Inc.). On the final postintervention angiogram, no contrast opacification was seen within the pseudoaneurysm [Figure 2f]. No other extracranial arterial supply was observed on the last sequences following embolization. No intimal flaps or intraluminal filling defects were seen within the parent artery. A follow-up angiogram was performed 8 days later (POD31), confirming persistence of the occlusion of the STA pseudoaneurysm. Over time, the temporal subcutaneous mass progressively decreased in size. The patient had a slow postoperative recovery. A follow up angiogram is planned in 3 months.

DISCUSSION

In reviewing the literature, we found reports of only five cases of postcraniotomy STA pseudoaneurysms.[3,12,16,19,21] The age at presentation ranged from 26 to 73 years. Four of the cases were males, and the gender of the fifth case was not specified. Three of the cases presented with aneurysmal subarachnoid hemorrhage that underwent surgical clipping, but none had postoperative clinically significant vasospasm. One underwent bilateral frontal sinus reconstruction and one had a frontal meningioma resected. The STA pseudoaneurysm presentation was delayed in all cases, ranging from 17 days to 3 months. One of the five cases was initially misdiagnosed as fluid or infectious collection and accessed percutaneously with a needle, yielding brisk arterial flow. Four were treated surgically and one was treated with percutaneous injection of thrombin glue.

In the presented case, the patient developed a pulsatile temporal mass approximately 22 days after surgery, while undergoing aggressive prolonged treatment for symptomatic vasospasm during the postoperative period. Similar to the other postoperative cases of pseudoaneurysm of the STA, the initial arterial injury

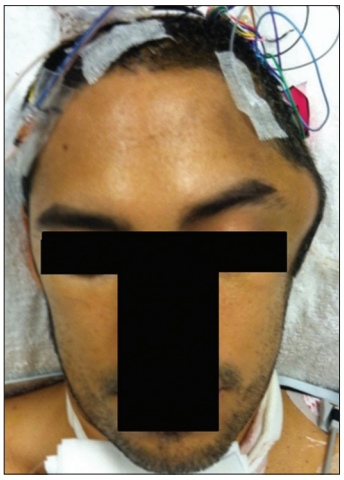

Figure 1: Subcutaneous enlarging mass in the left temporal region

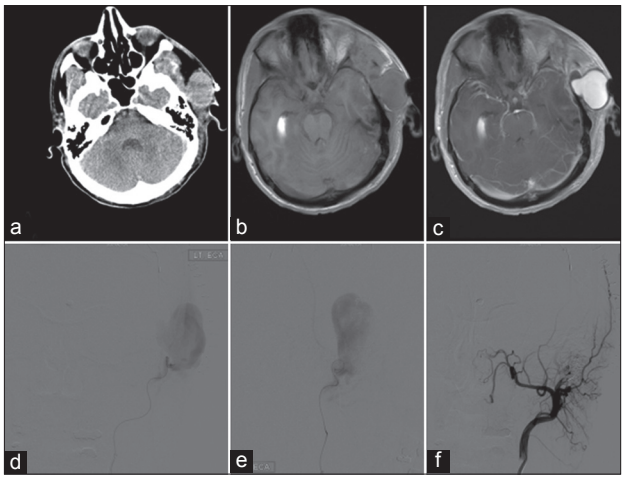

Figure 2: (a) Noncontrast axial CT image revealing a partially hypodense-isodense complex fluid collection superior to the zygoma, 3.6 cm in its greatest dimension. (b) Axial T1-weighted MRI revealing a 3.6 cm hyperintense fluid collection without and (c) with gadolinium, that is homogeneously enhancing. (d) Left selective STA injection, antero-posterior and (e) lateral projections show circular area of contrast blush. (f) Postcoiling angiogram, left ECA injection, antero-posterior projection, shows coils in superficial temporal artery and resolution of contrast blush

likely occurred during the fronto-orbital craniotomy. However, we propose that the prolonged periods of induced hypertension and hyperperfusion, as treatments for vasospasm, may have played a significant role in the development of the pseudoaneurysm. Furthermore, an underlying arterial wall predisposition may be a prerequisite since two thirds of the described cases of STA pseudoaneurysm occurred in the setting of ruptured intracranial aneurysms, while such reports have not been made in similar craniotomies used for tumor resection or other nonvascular procedures.

The goals of treatment of a STA pseudoaneurysm are to prevent hemorrhage, correct a cosmetic deformity, and alleviate pain as well as related headaches.[5] Medical management is generally not recommended primarily due to the persistent risk of aneurysmal rupture, as well as persistence with possible worsening of the cosmetic deformity.[11] However, one author described a case of a semi-conservative management in a critically ill patient with manual compression of the STA, which led to thrombosis of the aneurysm after 20 minutes.[6] Historically, the recommended treatment was surgical ligation and aneurysmoraphy.[5,11] This results in immediate aneurysm control, resolution of the cosmetic deformity, and prevention of recurrence. However, it usually requires induction of general anesthesia and sometimes results in a visible facial scar. Furthermore, surgical exploration of more proximal STA pseudoaneurysms may lead to facial nerve injury.[9] Recently, less invasive techniques have been explored as therapeutic options for STA pseudoaneurysms. Percutaneous ultrasound-guided thrombin injection has been described with resultant successful obliteration of such aneurysms.[3,5,15,17,19] However, experience with thrombin injections in other types of aneurysms has shown that this technique may be associated with recanalization, systemic intravascular thrombosis, and an allergic reaction to thrombin.[10] As an alternate obliteration technique, percutaneous ultrasound-guided coil embolization of an STA pseudoaneurysm has been successfully reported.[7] Recently, effective endovascular coil embolization of a STA pseudoaneurysm has been described.[8,9] Endovascular coil embolization of the STA pseudoaneurysm was also employed in the present case. Selective injection of the STA allowed assessing if other extracranial branches have been recruited to supply the pseudoaneurysm. Such complementary information is essential for tailored and complete endovascular treatment of these lesions. In addition to the use of coils, we sealed the origin of the pseudoaneurysm with Onyx (ev3 Inc.) liquid embolic material. Endovascular techniques may delay cosmetic results and require follow-up angiographic imaging to confirm persistence of obliteration and absence of recruitment.[3]

CONCLUSION

STA pseudoaneurysm is a rare entity usually occurring in the setting of blunt trauma. They are exceedingly rare in the setting of a craniotomy with only five cases reported in the literature, of which three occurred in the setting of a craniotomy for ligature of a ruptured intracranial aneurysm. We propose that prolonged periods of induced hypertension and hyperperfusion, as treatments for vasospasm, may contribute to the development of the pseudoaneurysm, especially in patients predisposed to vascular wall anomalies. In cases of vascular neurosurgery with potential for postoperative cerebral vasospasm necessitating use of triple H therapy, we recommend taking extra care during the craniotomy to avoid STA injury, thus

reducing the risk of developing a STA pseudoaneurysm. A postcraniotomy temporal mass should be considered a STA pseudoaneurysm until proven otherwise, and needle decompression should not be attempted. In addition to surgical ligation and resection, therapeutic options for STA pseudoaneurysms should include endovascular coil embolization, which based on our experience, is an effective option, especially for proximal STA pseudoaneurysms.

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