

Superficial temporal artery pseudoaneurysm presenting as extradural hematoma: A case report and review of literature

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ABSTRACT

Superficial temporal artery (STA) pseudoaneurysm after a craniotomy is very rare with only nine cases reported in the literature. However, no cases have been reported in the literature about this entity presenting as an emergency in the form of extradural hematoma (EDH). This case being one of the rare ones and is also unique wherein the indication for craniotomy being intracranial tuberculoma that is not yet reported in the literature. We report the first case of a postcraniotomy STA pseudoaneurysm induced EDH following craniotomy for intracranial tuberculoma.

Key words: Extradural hematoma, pseudoaneurysm, superficial temporal artery, tuberculoma

Introduction

Pseudoaneurysms are caused by communication between the intima of the vessel and the hematoma cavity during the process of absorption. The intima is false and consists of the organized connective tissues of hematoma. There is no vascular wall structure. Superficial temporal artery (STA) pseudoaneurysm was first described in 1740 by Bartholin. Pseudoaneurysms of the STA have been reported in about 400 cases worldwide.^[1] Most patients are asymptomatic presenting with a painless, pulsatile, and expanding mass on the affected temporal area.^[2] It is most commonly caused by blunt trauma to the temporal region of the head.^[3] Other fewer frequent etiologies include penetrating scalp injuries, hair grafting, temporomandibular joint surgery, dental surgery, parotid surgery, Gardner traction, and internal carotid artery (ICA) ligation. STA pseudoaneurysm after a

craniotomy is very rare with only nine cases reported in the literature.^[4-12] We report the first case of a postcraniotomy STA pseudoaneurysm presenting as EDH following craniotomy for intracranial tuberculoma.

Case Report

A 57-year-old male patient presented with complaints of right upper limb focal seizures since 2 months, speech difficulty of 1-month and 20 days of bifrontal headache and right upper limb weakness. On examination, he was conscious, oriented, dysphasic and had left facial upper motor neuron paresis with right arm weakness of 4/5 power. Magnetic resonance imaging of the brain revealed a left insular irregular enhancing heterogenous lesion with mass effect [Figure 1]. Our provisional diagnosis was high-grade glioma. He underwent left frontotemporal craniotomy, excision of the lesion and discharged. On the 8th postoperative day, he presented with altered sensorium and worsening of hemiparesis. Computed tomography scan revealed a large temporal operated site extracalvarial and extradural hematoma (EDH) with midline shift [Figure 2]. Re-exploration and decompression were done. He deteriorated again on the 2nd postoperative day and had to again re-explore. Surprisingly, he found to have a recurrence of hematoma on the postoperative scan. Since we were quite sure during the third time that adequate hemostasis care was taken, we thought of the other possibilities for the cause and considered angiogram, which revealed an aneurysm along the parietal branch of STA. Hence, he was re-explored for the fourth time and underwent ligation and excision of the aneurysm along with evacuation of EDH.

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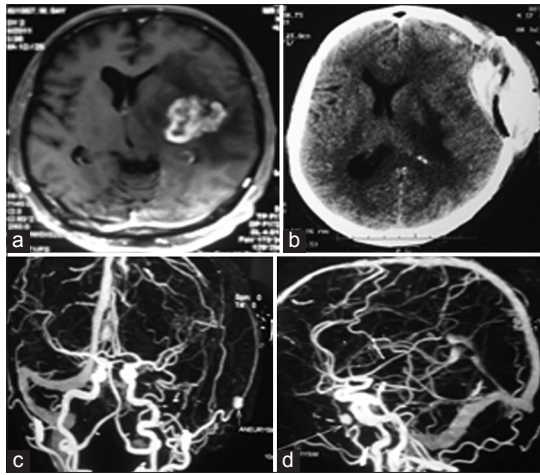


Figure 1: Pre and postoperative images of the patient. (a) Preoperative enhanced T1-weighted magnetic resonance imaging showing an enhancing lesion in the left insular lobe with a probable diagnosis of high-grade glioma. (b) Postoperative computed tomography-scan showed extradural hematoma beneath the craniotomy flap. (c) Anteroposterior and (d) lateral views of angiography that showed an aneurysm of the left superficial temporal artery

Histopathology was reported as Granuloma, and the intracranial biopsy revealed Tuberculoma. It was a pseudoaneurysm since there was no true wall noted in the aneurysm specimen. There were features of inflammation in the form of lymphocytes and histiocytes. He was treated with antitubercular drugs. He has been on regular follow-up since 1-year. He recovered normally and had no focal deficits.

Discussion

In reviewing the literature, we found reports of only nine cases of postcraniotomy STA pseudoaneurysms.^[4-12] The age at presentation ranged from 17 to 73 years. Six of the cases were males, two were females and the gender of one case was not specified. Five of the cases presented with aneurysmal subarachnoid hemorrhage that underwent surgical clipping,^[9,10] one underwent bilateral frontal sinus reconstruction,^[6] one had a frontal meningioma resected, one following posttraumatic intracerebral hematoma^[13] and another following ventriculostomy.^[11] 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. Six cases were treated surgically,^[9,10] two endovascular^[7,13] and one was treated with percutaneous injection of thrombin glue.^[4]

In the present case, the patient developed a pulsatile temporal mass approximately 8 days after surgery. Similar to the other postoperative cases of pseudoaneurysm of the STA, the initial arterial injury likely occurred during the craniotomy. However, we propose that the prolonged periods of tuberculosis (TB) induced vasculitis may have played a significant role in the

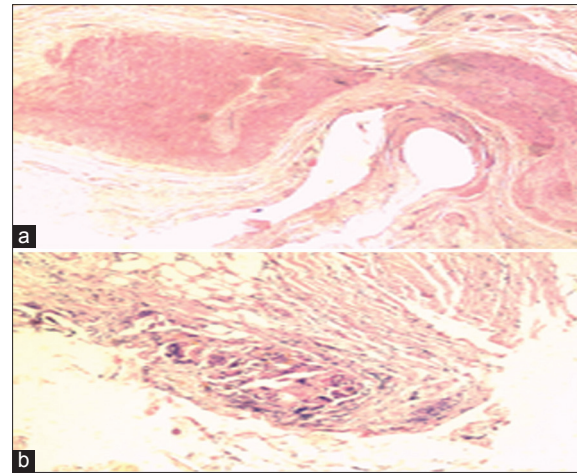


Figure 2: Histopathology of the resected aneurysm. Micro-slides were stained with H and E. (a) No true aneurysm wall could be observed. (b) Inflammatory and giant cells are observed under light microscopic magnification

development of the pseudoaneurysm. Vasculitis secondary to TB meningitis (TBM) can cause infarcts and rarely aneurysm formation^[13] in the intracranial vasculature. In TBM, inflammatory changes occur in the vessel wall of the arteries bathed in the exudate leading to narrowing of the lumen or occlusion by thrombus formation. The vessels at the base of the brain are most severely affected, including the ICA, proximal middle cerebral artery and perforating vessels of the basal ganglion. In these cases, the infection probably spreads from the adventitia toward the internal elastic lamina, weakening the vessel wall, with subsequent formation of an infectious aneurysm. The aneurysm in this case is probably a traumatic cause and not vasculitis induced, as there is no evidence of it in the histopathological examination. Furthermore, an underlying arterial wall predisposition has been noted in two thirds of the described cases of STA pseudoaneurysm in the setting of ruptured intracranial aneurysms^[9,10] while such reports have not been made in similar craniotomies used for tumor resection or other nonvascular procedures. Though the surgery around the STA is regularly done in neurosurgery, the development of pseudoaneurysms in very rare cases raises suspicion about any underlying secondary pathology of the vessel wall *per se*. Hence, we hypothesize that the mechanism underlying the development of this aneurysm could be an insensible trauma during craniotomy probably because of the weakened adventitia and internal elastic lamina due to extensive vasculitis affecting the intra and extracranial vasculature.

The goals of treatment of a STA pseudoaneurysm are to prevent hemorrhage, correct a cosmetic deformity and relieve pain. Our case actually required an emergency intervention as there was an acute EDH with mass effect that is not seen in any of the reported cases. This also emphasizes the importance of this entity. 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. Historically, the recommended treatment was surgical ligation and aneurysmorrhaphy. This results in immediate aneurysm control, resolution of the cosmetic deformity, and prevention of recurrence. In addition, surgical exploration of more proximal STA pseudoaneurysms may lead to facial nerve injury. Recently, fewer 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. As an alternate obliteration technique, percutaneous ultrasound-guided coil embolization of an STA pseudoaneurysm has been successfully reported. Recently, effective endovascular coil embolization of a STA pseudoaneurysm has been described. We followed the gold standard treatment of proximal and distal ligation and excision of the aneurysm and still feel that this is the best mode of treatment.

Conclusion

Postcraniotomy STA pseudoaneurysms being rarer with only nine cases reported. Careful dissection of the STA is required during any craniotomy especially in cases of aneurysm/arteriovenous malformation surgeries/diffuse vascular diseases. This case being one of the rarer ones is also unique wherein the presentation was an emergency in the form of EDH, which is not yet reported in the literature. Though the surgery around the STA is regularly done in neurosurgery, the development of pseudoaneurysms in very rare cases raises suspicion about any underlying secondary pathology of the vessel wall *per se*. Hence, we hypothesize that the mechanism underlying the development of this aneurysm could be an insensible trauma during craniotomy probably because of the weakened adventitia and internal elastic lamina due to extensive vasculitis affecting the intra and extracranial vasculature. Thus, the authors recommend histologic examination in all STA pseudoaneurysm cases explored surgically. Surgeons' familiarity with this entity is important for diagnosis and treatment as it can also present as an emergency condition that is very much curable.

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