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A spontaneous superficial temporal artery pseudoaneurysm possibly related to atherosclerosis: Case report and review of literature

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Abstract

Background: Spontaneous superficial temporal artery (STA) pseudoaneurysms are very rare; only four cases, including ours, have been reported to date. Therefore, the cause of them has not been studied.

Case Description: A 57-year-old woman was admitted to our hospital with a pulsatile mass in the left preauricular region. Her medical history included hypertension, dyslipidemia, and angina pectoris. She denied a history of head injury or minor head trauma. Three-dimensional computed tomography angiography showed a well-enhanced saccular aneurysm on the main trunk of the STA. To prevent rupture it was removed surgically. The histological diagnosis was pseudoaneurysm with atherosclerosis. By the 2nd postoperative day, she had completely recovered and was discharged home. There has been no relapse.

Conclusions: As all four documented patients were at high risk for atherosclerosis, we posit that a causal factor was weakening of the arterial wall due to atherosclerosis and chronic pressure on the STA from anatomical structures. Here, we present histological evidence to support this hypothesis.

Key Words: Pseudoaneurysm, spontaneous, superficial temporal artery

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INTRODUCTION

The first case report of a superficial temporal artery (STA) aneurysm was published in 1740; subsequently, about 400 additional cases have been reported.[1,2] Histologically most of the lesions were pseudoaneurysms that arose posttrauma.[2,3,5,7,10,12,13,15,18,19,21,22] Although most STA aneurysms of unknown etiology, i.e., spontaneous STA aneurysms, were true aneurysms in patients with atherosclerosis,[21] four were false aneurysms. The histologic diagnosis was dissecting aneurysm

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in one patient[5] and pseudoaneurysm in the other three.[14] We report a fourth patient with a spontaneous STA pseudoaneurysm and discuss its potential pathogenesis, the background of patients with these aneurysms, and the anatomical features of these lesions.

CASE REPORT

Presentation and clinical evaluation

This 57-year-old woman was referred to our hospital with a growing pulsatile mass in the left preauricular region. She had first noticed the gradually growing mass 5 years earlier. Her medical history included hypertension (HT), dyslipidemia (DL), and angina pectoris. She was treated with oral calcium- and angiotensin II receptor blockers. She had no recollection of head injury or minor head trauma.

Physical examination disclosed no neurological deficits. There was a 13 mm × 10 mm pulsatile mass in the left preauricular region; it was painless but annoying. Three-dimensional computed tomography angiography showed a well-enhancing saccular aneurysm on the main trunk of the STA [Figure 1a].

We were instructed her to manually compress the pseudoaneurysm (press a finger on the skin over the pulsatile area) for 30 min 3 times a day. However, it did not thrombosed. To prevent aneurysmal rupture we performed the surgical removal.

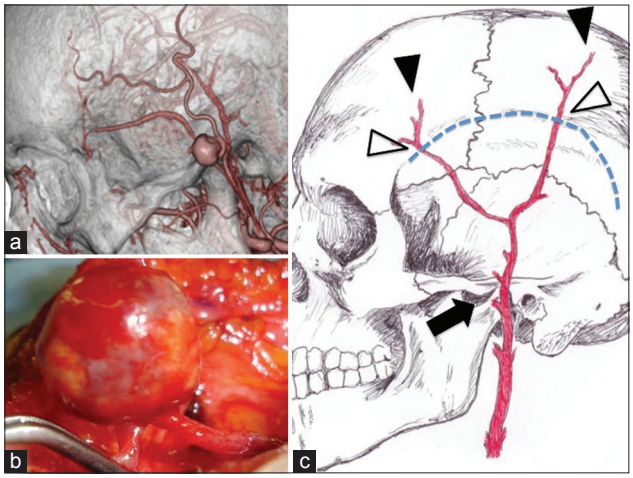

Figure 1: Pre- and intra-operative superficial temporal artery aneurysm imaging and figure. (a) Three-dimensional computed tomography angiogram showing an aneurysm on the main trunk of the superficial temporal artery and the preauricular region on the zygomatic arch. (b) Intraoperative photograph showing the superficial temporal artery aneurysm. There is a small distance between the feeding and the draining artery. (c) Lateral view of the cranial and superficial temporal artery schema showing that the main trunk of the superficial temporal artery crosses the zygomatic arch in the preauricular region, the site where the spontaneous superficial temporal artery pseudoaneurysm developed (arrow). The superficial temporal artery passes over the linea temporalis (white arrowhead) and includes its tip (black arrowhead)

Surgery

With the patient under general anesthesia, we made a skin incision about 4 cm above the main trunk of the left STA. The aneurysm was saccular and located lateral to the zygomatic arch. There was a small distance between the feeding and draining vessels [Figure 1b]. The distal and proximal regions of the aneurysm were ligated, and it was removed without any complications.

Pathology

Several histologic sections were prepared from the surgically resected specimen. A cross-section of the aneurysm [Figure 2a] showed marked dilation of the artery. At the site of the aneurysm, the normal arterial wall structure was replaced by fibroblast proliferation and loose connective tissue [Figure 2b]; in the vicinity of the aneurysm, we observed atherosclerosis with intimal thickening and calcification in the media [Figure 2c]. No significant inflammation suggestive of arteritis was identified. The diagnosis was pseudoaneurysm with atherosclerosis.

Postoperative course

The patient was discharged home on the 2nd postoperative day. Her condition continues to be good and at the last follow-up 12 months after the operation, there was no evidence of recurrence.

DISCUSSION

Although no universally accepted classification of STA aneurysms based on their etiology has been established,

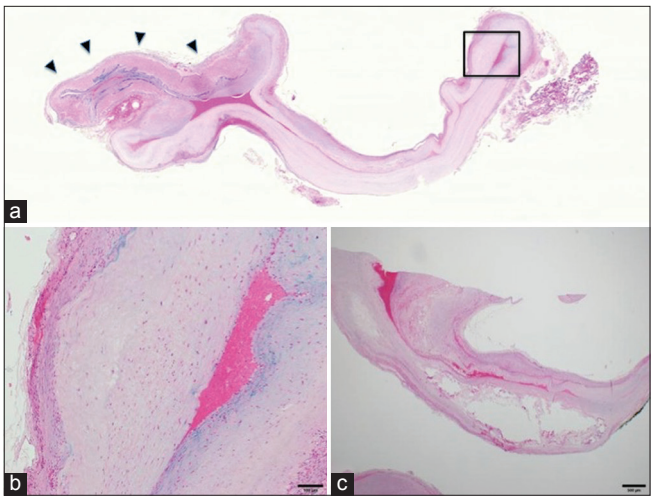

Figure 2: Histological features. (a) Low magnification of the lesion showing saccular dilation of the artery. The remnant of the internal elastic lamina is seen on the left (arrowheads). Victoria blue - H and E staining. (b) High magnification of the aneurysmal wall (encased by a rectangle in Figure 2a). Fibroblast proliferation and loose connective tissue are observed, and the normal arterial wall structure is lost (Victoria blue - H and E, ×100. (c) The arterial wall in the vicinity of the aneurysm shows intimal thickening and calcification in the media (H and E, ×20)

they have been classified as traumatic, iatrogenic, and spontaneous.[2,3,5,7,8,10,12-16,18,19,21,22] In our discussion, we use the designation traumatic and spontaneous (nontraumatic) aneurysms to describe traumatic- and iatrogenic aneurysms. Schechter and Gutstein[18] reported that 92% of STA aneurysms were traumatic, and the other 8% were spontaneous.

Traumatic aneurysms include those that arise following blunt head trauma, vehicular accidents, stab and gunshot wounds, and sports-related injuries,[2,3,6,10,11,13,15,22] while iatrogenic STA aneurysms are attributed to external ventricular drainage catheters, pin-type head-holder devices, craniotomy, hair transplants, and the injection of botulinum toxin.[1,2,4,11,16,17,20]

Histologically, most traumatic STA aneurysms exhibit the features of pseudoaneurysms.[5] Spontaneous aneurysms, on the other hand, are true aneurysms that tend to be associated with arteriosclerosis.[14,21] Very rarely, spontaneous STA aneurysms are histologically dissecting aneurysms or pseudoaneurysms. Fujii *et al.*[5] reported a spontaneous STA aneurysm whose histologic diagnosis was dissecting aneurysm, and Nii *et al.*[14] encountered three spontaneous STA pseudoaneurysms.[14] To the best of our knowledge, ours is the fourth spontaneous STA pseudoaneurysm with a definitive histologic diagnosis.

Although Nii *et al.*[14] suggested that spontaneous STA pseudoaneurysms were attributable to minor trauma or arterial dissection, none of their three aneurysms presented with histopathologic evidence of dissection. As all three patients wore eyeglasses, this raised the suspicion that repetitive or continuous compression and friction on the STA by the arms of the glasses damaged the vessel walls, eventually resulting in the STA pseudoaneurysms. Our patient, on the other hand, did not wear eyeglasses, and there was no histologic evidence of a dissecting aneurysm.

Most traumatic aneurysms in the head arise on the STA due to its anatomical location.[11] All four spontaneous STA pseudoaneurysms, including ours, developed in the preauricular region [Table 1] where the STA crosses over the zygomatic arch. As there is only thin connective tissue between the STA and the zygomatic arch in this region, the STA can be compressed chronically and damaged [Figure 1c, arrow]. According to Kawabori *et al.*,[9] true STA aneurysms tend to occur in younger patients in the preauricular region.

They suggested the congenital vulnerability of the arterial wall in this patient population, rather than atherosclerosis, as an important causal factor and the anatomic features of the preauricular region as a contributing factor. From a different perspective, this portion of the STA is not vulnerable to traumatic injury because it runs inside a line from the auricle to the zygomatic arch [Figure 1c, arrow].

There are two specific sites on the STA that are vulnerable to trauma. One is its peripheral tract; the temporalis muscle is the only protective tissue between the STA and the outer table of the skull. The peripheral portion is particularly vulnerable to injuries due to the lack of protective temporal and frontal muscle tissues [Figure 1c, black arrowhead].[11] The linea temporalis is also vulnerable to trauma because it passes over the frontal or parietal osseous ridge in the galea aponeurotica formed by deep and superficial temporal muscle fasciae; its location results in tethering to the galea aponeurotica [Figure 1c, white arrow head].[20] Anatomically, traumatic STA pseudoaneurysm may arise at sites peripheral to the frontal and parietal branches of the STA.

Other common findings in the four patients with spontaneous STA pseudoaneurysms were their demographics and medical history. All were middle-aged or older women with a history of HT and DL, putting them at high risk for atherosclerosis [Table 1]. Moreover, the histologic findings were consistent with pseudoaneurysm with atherosclerosis [Figure 2]. This observation is noteworthy because many previously-reported traumatic STA pseudoaneurysms arose in younger males.[12,15,19] Most STA aneurysms are addressed by surgical resection due to their low risk for recurrence and from a cosmetic point of view. Small lesions can be treated using an endovascular approach.[5,7,11,19] All four of the spontaneous STA pseudoaneurysms were surgically resected, and none recurred.

Based on our and previously reported observations in patients with spontaneous STA pseudoaneurysms, we suggest that atherosclerosis, which weakens the arterial wall, and chronic pressure on the STA from anatomical structures, play a role. To support our hypothesis, more

Table 1: Demographic and medical data on the four documented patients with spontaneous superficial temporal artery pseudoaneurysms

Authors and year	Age	Sex	Past history	Side	Site	Aneurysm type	Treatment	Recurrence
Nii, *et al.*, 2011	75	Female	HT, DL	Right	PA	Fusiform	Removal	No
	76	Female	HT, DL, DM	Left	PA	Fusiform	Removal	No
	76	Female	HT, DL	Right	PA	Fusiform	Removal	No
Present case	57	Female	HT, DL, AP	Left	PA	Saccular	Removal	No

HT: Hypertension, DL: Dyslipidemia, DM: Diabetes mellitus, AP: Angina pectoris, PA: Preauricular lesion

case reports and series are needed to identify patient demographics devoid of bias as we cannot rule out the possibility that patients forgot minor trauma to the affected site.

CONCLUSION

To the best of our knowledge, this is the first report of the possible association between atherosclerosis and a spontaneous STA pseudoaneurysm. HT and DL must be treated to prevent cardiovascular events and to prevent spontaneous STA pseudoaneurysms, and physical stress on temporal areas must be avoided, especially in middle-aged and older women with risk factors for atherosclerosis.

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Conflicts of interest

All authors who are members of the Japan Neurosurgical Society (JNS) have registered online self-reported conflict of interest disclosure statement forms through the website for JNS members.

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