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Review Article

True superficial temporal artery aneurysm: A case after extracranial-intracranial bypass surgery and a systematic review

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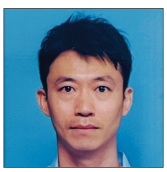

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ABSTRACT

Background: Nontraumatic true superficial temporal artery aneurysm (STAA) is rare, and its characteristics and pathogenesis are unclear.

Methods: We report a case of STAA and performed a systematic review of PubMed, Scopus, and Web of Science using the keyword "superficial temporal artery aneurysm" to include studies on STAA reported through July 2022. We excluded studies on STAA associated with trauma, arterial dissection, infection, or vasculitis.

Results: A 63-year-old woman who underwent left superficial temporal artery (STA)-middle cerebral artery bypass surgery 8 years previously was diagnosed with an aneurysm located at the left STA. The blood flow volume estimated by ultrasonography was higher in the left STA than in the contralateral counterpart (114 mL/min vs. 32 mL/min). She underwent clipping surgery to prevent aneurysmal rupture without sequela. The lesion was diagnosed as a true aneurysm by histology. The systematic review identified 63 cases (including the present case) of nontraumatic true STAA. The median age of the patients was 57 (interquartile range [IQR]: 41–70) years. Most (90.5%) cases were detected as a palpable mass. Aneurysmal rupture occurred in only 1 (1.6%) case, despite the large size of aneurysms (median size: 13 [IQR: 8–20] mm) and the high frequency (33.3%) of aneurysmal growth during observation. Most (93.7%) patients underwent surgical resection of STAA without sequela.

Conclusion: Our findings suggest that the pathogenesis of true STAA is promoted by hemodynamic stress. The systematic review clarified patients' and aneurysmal characteristics and treatment outcomes, providing further insight into the pathogenesis of nontraumatic true STAA.

Keywords: Bypass, Intracranial aneurysm, Segmental arterial mediolysis, Superficial temporal artery aneurysm, Systematic review

INTRODUCTION

Superficial temporal artery aneurysm (STAA) is an uncommon disease, and approximately 90% of STAAs are traumatic pseudoaneurysms.[59] Nontraumatic true STAA is much rarer than traumatic STAA and has mainly been presented in case reports.[25,26] The rarity of true STAA means that its characteristics and the underlying pathogenesis are not well clarified.

We report here a rare case of a true STAA that developed more than 2 years after superficial temporal artery (STA)-middle cerebral artery (MCA) bypass surgery. The findings in the present case suggest the potential pathogenesis leading to the development of true STAA and show a rare complication after STA-MCA bypass surgery. We also performed a systematic review of the literature to investigate the characteristics, treatment outcomes, and potential pathogenesis of nontraumatic true STAA.

MATERIALS AND METHODS

This study was conducted following the principles of the Declaration of Helsinki and its later amendments. The patient provided written informed consent for the treatment rendered. This systematic review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.[35] All datasets analyzed in this study are shown in Supplementary Tables 1 and 2. Ethical approval was unnecessary because the present study analyzed data from published literature.

Search strategy and selection criteria

We performed a systematic review of PubMed, Scopus, and Web of Science to identify eligible studies published between the date of the databases' inception and July 2022 using the keyword "superficial temporal artery aneurysm." We also searched the literature referenced by eligible studies for any possible missing studies. The present systematic review aimed to analyze the characteristics, treatment outcomes, and potential pathogenesis of nontraumatic true STAA. Therefore, the inclusion criteria were studies reporting STAA. The exclusion criteria were studies on STAA associated with trauma, arterial dissection, infection, or systemic inflammatory diseases such as vasculitis. Articles written in languages other than English or Japanese were translated using Google translate (https://translate.google.co.jp/?hl=ja).

Data extraction

Two investigators (M.I. and K.S.) independently completed the database search. Any discrepancies between investigators were resolved by discussion. We extracted data from the included studies using a standardized form consisting of the following factors: patients' characteristics (age, sex, and a history of hypertension); aneurysmal characteristics, such as size, shape (i.e., saccular or fusiform), location (e.g., the frontal branch of the STA), multiplicity, symptoms (e.g., tenderness), growth during observation, and rupture status; and factors regarding diagnosis or treatment, such as diagnostic modalities, histological findings regarding the etiology and wall thickness, treatment modalities, and treatment complications. In this study, multiplicity indicated if the patient had another aneurysm, such as an intracranial aneurysm (IA), visceral aneurysm (e.g., renal artery aneurysm), or aortic aneurysm [Supplementary Table 1].

The extracted data are shown as the median (interquartile range [IQR]) or frequency (i.e., %). To assess the pathogenesis of STAA, we compared the characteristics of STAA with those of IA by referencing the International Study of Unruptured IA Investigators performed in the U.S.A, Canada, and Europe,[19] the Unruptured Cerebral Aneurysm Study by Japanese investigators,[37] and a histological analysis performed by Frösen *et al*.[15]

RESULTS

Case report

A 55-year-old woman with a medical history of hypertension and dyslipidemia visited our hospital complaining of weakness in the right upper extremity. A radiological examination showed cerebral infarction and hemodynamic compromise due to the left MCA stenosis. Therefore, she underwent revascularization by the left STA-MCA bypass. Two-year postoperative magnetic resonance angiography showed no aneurysm formation at the left STA [Figure 1a]. However, an 8-year postoperative magnetic resonance angiography demonstrated an aneurysm on the outer curvature at a sharp bend of the left STA [Figure 1b]. Digital subtraction angiography showed a saccular aneurysm that was 5 mm in size and an increase in the diameter of the left STA [Figures 1c-e]. The diameter and blood flow volume at bilateral STAs were measured in front of the external acoustic meatus by ultrasonography (LOGIQ S8 XDclear 2.0; GE Healthcare, USA) using a high-frequency linear probe (9 MHz). The diameter of the left STA was 2.6 mm, while the contralateral STA was 1.6 mm. The estimated blood flow volume was approximately 3.5 times higher in the left STA at the main trunk than in the contralateral counterpart (114 mL/min vs. 32 mL/min). Clipping surgery was performed to prevent aneurysmal rupture [Figures 1f-i]. The location of the aneurysm could be precisely identified by a 6-0 nylon suture used to ligate a branch from the parietal branch at the time of the previous bypass surgery [Figures 1c and h]. A histopathological investigation showed a lack of the internal elastic lamina, and a thin and hypocellular wall in the STAA. These histological features were evident compared with a control STA of a 79-year-old man [Figures 2a-h]. The three layers of the arterial wall (i.e., the intima, media, and adventitia) were preserved, consistent with the diagnosis of a true STAA. She was discharged on the 13th day postoperatively without sequela.

Search results

We included 45 studies in the present systematic review after full-text screening [Figure 3].[1,5,7-14,17,18,20-34,36,38-47,49,55,57,58,61-63]

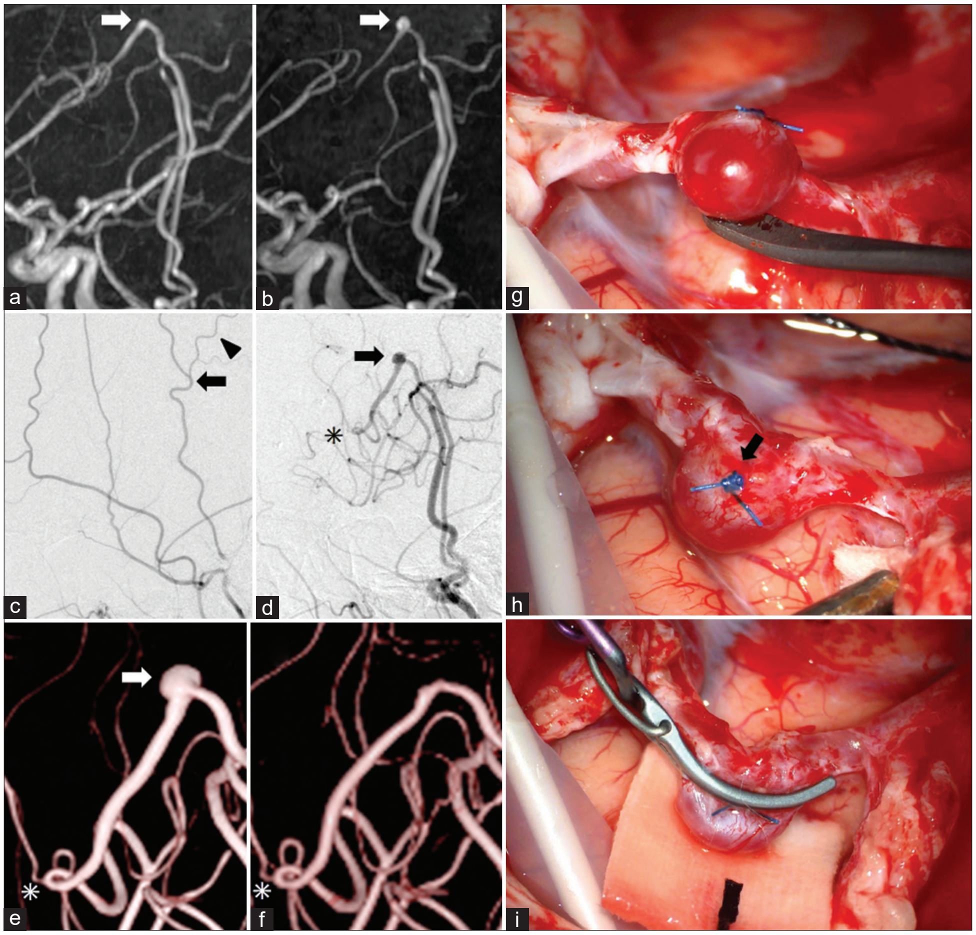

Figure 1: Radiological and intraoperative findings. (a and b) Two- (a) and 8-year (b) postoperative magnetic resonance angiography shows the development of a superficial temporal artery aneurysm (STAA) between 2 and 8 years after superficial temporal artery (STA)-middle cerebral artery bypass surgery. The STAA is localized at a sharp bend of the left STA (arrows in a and b) proximal to the anastomosis. (c and d) Preoperative (c) and 8-year postoperative (d) digital subtraction angiography show a *de novo* saccular STAA (5 mm in size) on the outer curvature of the left STA (arrows in c and d) and a postoperative increase in the diameter of the left STA. A small branch from the STA (arrowhead in c) was ligated using a 6-0 nylon suture and cut before the anastomosis. The site of the anastomosis is indicated by an asterisk in (d). (e and f) Three-dimensional rotational angiography before (e) and after (f) clipping surgery. The STAA is indicated by an arrow in (e), and the site of the anastomosis is indicated by asterisks in (e) and (f). (g-i) Intraoperative findings of the STAA. The appearance of the STAA resembles that of usual intracranial aneurysms. A 6-0 nylon suture (arrow in h) used to ligate the branch indicated by the arrowhead in (c) remains and was useful in identifying the precise location of the STAA.

According to the systematic review, 63 cases, including the present case, were identified after Brown first reported pathologically confirmed nontraumatic true STAA in 1942. All of the data acquired following the standardized form are shown in Supplementary Tables 1 and 2.

Patients' and aneurysmal characteristics of STAA and IA are summarized in Table 1 and Figure 4.[15,19,37] The median age of the patients was 57 (IQR: 41–70) years, and the median size of STAA was 13 (IQR: 8–20) mm. Approximately half of patients with STAA had a medical history of hypertension. The most common location of STAA was in the main trunk (27/56; 48.2%) followed by the frontal branch (19/56; 33.9%) [Figure 4a]. Most (61/63; 96.8%) of the cases were symptomatic. Fifty-seven (90.5%), 2 (3.2%), 1 (1.6%), and 1 (1.6%) case presented with a palpable mass, aneurysmal subarachnoid hemorrhage due to another aneurysm, rupture of STAA, and tenderness without a palpable mass, respectively. Atherosclerosis was the most frequent (13/21; 61.9%) etiology as indicated by histopathological findings [Table 1]. An age distribution histogram of STAA shows that its incidence

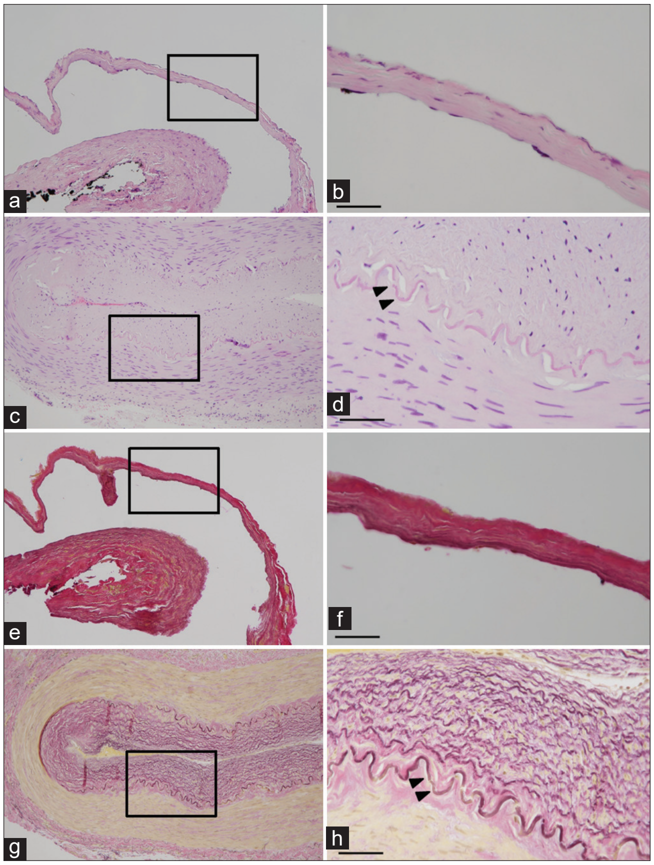

Figure 2: Histopathological investigation of the superficial temporal artery aneurysm (STAA) and a control superficial temporal artery (STA) in a 79-year-old man. Hematoxylin and eosin (a-d) and Elastica van Gieson (e-h) staining of the STAA (a, b, e, and f) and the control STA (c, d, g, and h) are shown. The magnified views indicated by the squares in (a, c, e, and g) are shown in (b, d, f, and h), respectively. The internal elastic lamina in the control STA is indicated by arrowheads in (d and h). The internal elastic lamina was absent in the STAA. The aneurysmal wall was thin and hypocellular. Scale bar, 50 µm.

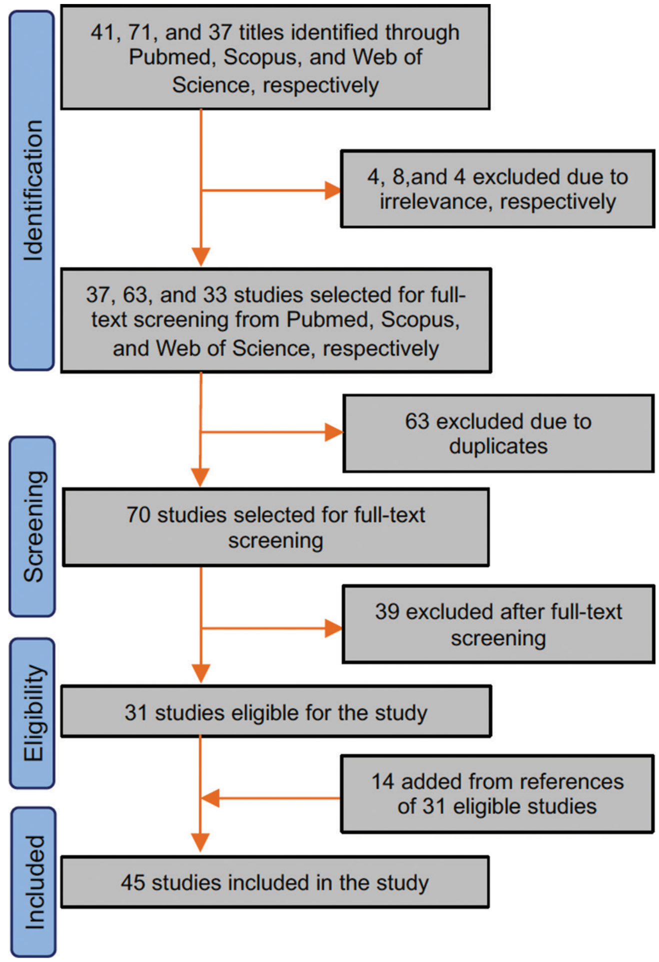

Figure 3: Study inclusion flowchart.

is positively associated with the patient's age, peaking at the 60–70s [Figure 4b]. With regard to treatment, surgical resection, clipping, and endovascular embolization were chosen in 59 (93.7%), 1 (1.6%), and 2 (3.2%) cases, respectively. One patient was lost to follow-up before treatment. Treatment complications have never been reported (0%).

Among the characteristics, the similarities of STAA with IA in the International Study of Unruptured IA Investigators and Unruptured Cerebral Aneurysm Study were observed for the patients' age, the frequency of a history of hypertension, and multiplicity of aneurysms [Table 1].[19,37] Histological features of a degenerative change in the aneurysm wall, such as thinning and loss of mural cells, were also observed in STAA with a similar frequency to that in IA.[15] However, there was a male predominance in STAA. Although an STAA 20 mm or larger in size and aneurysmal growth during observation were frequent (31% and 33.3%, respectively), aneurysmal rupture rarely occurred in STAA (1/63; 1.6%).

DISCUSSION

In this systematic review, nontraumatic true STAA not associated with dissection, infection, or systematic inflammatory diseases was exhaustively included and analyzed. The systematic review of 63 reported cases of STAA showed that most cases were detected as a palpable mass. Intriguingly, aneurysmal rupture was rarely reported, despite the large size of aneurysms (i.e., the median size was 13 mm) and the high frequency of aneurysmal growth during observation.[20] Surgical interventions might effectively prevent rupture of STAA because growth can be easily identified by palpation, unlike asymptomatic IA.[6] Cosmetic problems, tenderness, and potential rupture should be considered as indications for surgical interventions. Meanwhile, no surgical complications were reported, although the potential risk for facial nerve palsy due to skin incision should be noted. Therefore, patients with STAA are

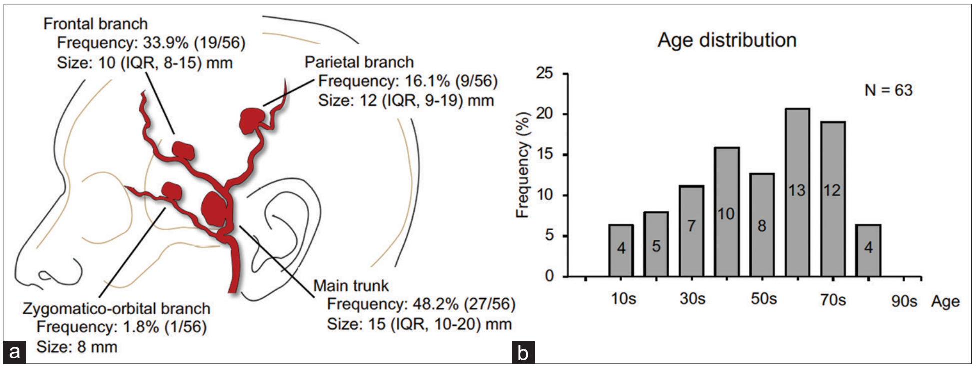

Figure 4: Characteristics of superficial temporal artery aneurysm (STAA) from a systematic review of 63 cases. (a) Incidence and size according to the location of STAA. Data are shown as the median and interquartile range (IQR). Among the 63 cases, data on the location and size are missing in 7 and 2 cases, respectively. (b) Age distribution histogram of patients with STAA. The numbers of patients belonging to each age group divided by every 10 years are shown in the histogram.

Table 1: Characteristics of 63 STAAs from a systematic review.

	Missing*	STAA (*n*=63)	Intracranial aneurysm (IA)		
			ISUIA[19] Group 1† (*n*=727)	UCAS[37] (*n*=6697)	Frösen *et al.*[15] Unruptured IA (*n*=24)
Age, yr	0	57 (41–70)	56	62.5±10.3	52
Female, %	0	28 (45.2)	517 (71.1)	4532 (67.7)	17 (70.8)
Hypertension, %	32	16 (51.6)	/‡(44.2)	2969 (44.3)	/‡
Size	2	13 (8-20)	10.9 (/‡)	5.7±3.7	7.5 (/‡)
Multiplicity, %	1	13 (21.0)	182 (25.0)	1770 (26.4)	11 (45.8)
Shape	17				
Saccular		27 (58.7)			
Fusiform		19 (41.3)			
Pathology-etiology	42				
Atherosclerosis		13 (61.9)			
Segmental arterial mediolysis		3 (14.3)			
Mucoid degeneration		2 (9.5)			
Papillary endothelial hyperplasia		2 (9.5)			
Cystic medial necrosis		1 (4.8)			
Pathology wall thickness	32				
Thinning/hypocellular		9 (29.0)			5 (20.8)
Tenderness	3	12 (20.0)			
Growth during observation	12	17 (33.3)			
Rupture	0	1 (1.6)			

Data are presented by the median (interquartile range), mean±standard deviation, or number (%). *Missing indicates the number of missing data among 63 cases in the systematic review. †Group 1 included patients with unruptured intracranial aneurysms without a history of aneurysmal subarachnoid hemorrhage. ‡Data were unavailable. STAA: Superficial temporal artery aneurysm, ISUIA: International Study of Unruptured Intracranial Aneurysms Investigators, UCAS: Unruptured cerebral aneurysm study

subjected to little risk by the surgical interventions, which justify the indications for the treatments. Among the 63 reported cases, our case is the first to show the development of true STAA as a rare complication after STA-MCA bypass surgery. Besides, our case is the first to demonstrate the feasibility of neck clipping of STAA in the situation where preserving blood supply to the brain through the STA-MCA bypass is necessary.

The present systematic review showed that several vascular risk factors (i.e., aging and hypertension) were similarly associated with the development of aneurysms both in STA and intracranial arteries [Table 1].[19,37] In addition, histological analysis showed that atherosclerosis was the most prevalent type of histological feature in STAA. Lipid accumulation in the arterial wall may be associated with degenerative changes, leading to the development of STAA, as suggested in IA.[16]

Segmental arterial mediolysis, which is a rare arteriopathy causing aneurysms in various locations,[56] was the second frequent pathological finding identified in STAA. Therefore, the pathogenesis of STAA as indicated by histological findings is in line with that of aneurysms in other locations.

Hemodynamic stress loaded on the arterial wall, which depends on blood flow volume and the morphology of the vessel,[50] may also be involved in the pathogenesis of STAA. In our case, STAA developed more than 2 years after the surgical intervention and in the context of an increase in blood flow volume and sharp bend of the STA. Therefore, a hemodynamic stress-dependent mechanism is likely to have been involved in the pathogenesis of STAA, although the effect of direct mechanical stress by the surgical intervention should also be considered. Similarly, STAA can develop in a feeder of arteriovenous malformation in the scalp or in association with porocarcinoma or hemihyperplasia, where the blood flow volume in the STA potentially increases.[23,32,41] These findings are consistent with the previous studies investigating IA in humans and animal models.[4,52,54]

The prevalence of true saccular aneurysms is diverse between locations throughout the body because of undetermined reasons, which are presumably associated with the pathogenesis. The prevalence of saccular aneurysms is highest in intracranial arteries followed by visceral arteries (e.g., the splenic artery) (3.2% vs. 0.01–0.2%).[3,60] The incidence rate of STAA is much lower than that of visceral artery aneurysms. Differences in the arterial structure should be considered as a reason for the differences in the prevalence of saccular aneurysms between locations. Intracranial arteries have a thinner media and adventitia, fewer elastic medial fibers, and lack the external elastic lamina and vasa vasorum.[48] However, our case is insightful in that an increase in blood demand compatible with that of an intracranial artery can also induce an aneurysm in the STA. Indeed, the previous animal studies showed that saccular aneurysms were reproducibly induced under similar conditions in intracranial and extracranial arteries.[2,51,53] These findings suggest that the blood demand of each organ may be a factor affecting the prevalence of aneurysms through the regulation of the blood flow volume *in situ*.

CONCLUSION

To the best of our knowledge, this is the first case to show a rare complication after STA-MCA bypass surgery (i.e., *de novo* aneurysm formation in the STA). In addition, the present case suggests that the pathogenesis of true STAA is promoted by hemodynamic stress, as with IA. Our systematic review clarified patients' and aneurysmal characteristics of STAA and treatment outcomes. The analysis of 63 reported cases of STAA suggests that its characteristics are generally in line with that of saccular aneurysms in other locations, such as IA. A male predominance and high frequency of aneurysmal growth during observation in STAA should be regarded as potential differences from IA. This study provides comprehensive data regarding STAA and further insight into the pathogenesis of saccular aneurysms, including STAA and IA.

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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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SUPPLEMENTARY TABLES

Supplementary Table 1: Standardized form for patients' and aneurysmal characteristics.

	Language	Case No	Patient characteristics			Aneurysm characteristics							
			Age, year	Sex	HT	Size, mm	Shape	Location	Multiplicity	Pain	Initial presentation	Growth	Rupture
Brown, 1942	English	1	34	M	–	32	Fusiform	T	–	–	Palpable mass	+	–
Martin, 1955	English	2	60	M	+	10	Fusiform	Fr	–	–	Palpable mass	+	–
Buckspan and Rees, 1986	English	3	70	M	/	40	/	T	–	–	Palpable mass	+	–
Locatelli *et al.*, 1988	English	4	10	M	–	10	Fusiform	P	–	–	Palpable mass	–	–
Ikeda and Watanabe, 1988	Japanese	5	15	M	–	10	Saccular	T	–	–	Palpable mass	+	–
Nishioka *et al.*, 1988	Japanese	6	14	M	–	15	Saccular	P	–	–	Palpable mass	+	–
Dailey *et al.*, 1994	English	7	50	F	/	5	Saccular	/	–	+	Palpable mass	–	–
Nishimura and Kubota, 1996	Japanese	8	47	F	–	30	Saccular	T	–	+	Palpable mass	+	–
Uchida and Sakuma, 1999	English	9	34	M	–	20	Saccular	T	–	–	Palpable mass	+	–
Endo *et al.*, 2000	English	10	85	M	+	10	Saccular	T	STAA	–	Palpable mass	–	–
		11	85	M	+	3	Fusiform	Fr	STAA	–	Asymptomatic	–	–
Porcellini *et al.*, 2001	English	12	24	F	/	/	/	P	–	/	Palpable mass	–	–
Ohta *et al.*, 2003	English	13	55	F	+	6.5	Saccular	T	Two IA	/	Aneurysmal SAH	–	–
Riaz *et al.*, 2004	English	14	65	M	/	20	Fusiform	/	–	+	Palpable mass	–	–
		15	77	M	/	15	/	/	–	–	Palpable mass	–	–
Coenegrachts *et al.*, 2005	English	16	59	M	+	27	Fusiform	T	ICAA	–	Palpable mass	+	–
Silverberg and Teodorescu, 2005	English	17	46	M	–	24	Saccular	T	–	–	Palpable mass	+	–
Ysa *et al.*, 2008	English	18	59	F	+	15	Saccular	T	–	–	Palpable mass	–	–
Gull *et al.*, 2009	English	19	80	F	/	8	Fusiform	T	–	+	Palpable mass	–	–
Piffaretti and Castelli, 2009	English	20	63	F	+	13	Fusiform	T	–	–	Palpable mass	–	–
		21	47	M	+	15	Fusiform	/	STAA	–	Palpable mass	–	–
Kawabori *et al.*, 2009	English	22	78	F	/	47	Saccular	T	–	–	Palpable mass	–	–
Wise and Gibson, 2009	English	23	49	F	–	4.5	Fusiform	Fr	STAA	–	Palpable mass	–	–
		24	49	F	–	2.3	/	/	STAA	+	Palpable mass	–	–
Karam *et al.*, 2010	English	25	34	F	/	16	/	T	–	–	Palpable mass	–	–
Bozkurt *et al.*, 2011	English	26	62	M	/	30	Saccular	/	–	–	Palpable mass	–	–
Labropoulos *et al.*, 2011	English	27	26	M	/	14	/	/	/	–	Palpable mass	/	–
Moriyama *et al.*, 2011	English	28	67	F	/	10	Fusiform	Fr	–	–	Palpable mass	–	–
Sakamoto *et al.*, 2011	English	29	77	F	/	16	Saccular	T	–	–	Palpable mass	–	–
Nair *et al.*, 2011	English	30	84	F	/	15	Saccular	T	–	–	Palpable mass	–	–
Mousa *et al.*, 2011	English	31	72	F	+	20	Saccular	P	–	+	Palpable mass	+	–
Wei and Akkersdijk, 2011	English	32	72	M	/	10	Fusiform	T	–	–	Palpable mass	–	–
Lim *et al.*, 2012	Korean	33	50	F	–	10	Saccular	T	–	–	Palpable mass	–	–
Molinaro, 2012	English	34	33	M	–	8	Fusiform	ZO	–	–	Palpable mass	+	–
Sloane *et al.*, 2013	English	35	32	M	/	37	Saccular	Fr	–	+	Palpable mass	+	–
Joshi and Klimczak, 2014	English	36	60	M	/	20	Saccular	Fr	–	–	Rupture of STAA	+	+

(Contd...)

Supplementary Table 1: *(Continued)*.

	Language	Case No	Patient characteristics			Aneurysm characteristics							
			Age, year	Sex	HT	Size, mm	Shape	Location	Multiplicity	Pain	Initial presentation	Growth	Rupture
López-Ruiz *et al*., 2014	English	37	67	M	/	25	Fusiform	Fr	–	+	Pain	–	–
Kawai *et al*., 2014	English	38	65	M	–	10	Saccular	Fr	IA	–	Palpable mass	+	–
		39	76	F	+	17	Saccular	T	–	–	Palpable mass	–	–
		40	57	F	+	6	Saccular	Fr	STAA, OAA	–	Palpable mass	–	–
Kim, 2014	English	41	70	F	/	8	/	Fr	–	–	Palpable mass	/	–
		42	68	F	/	9	/	Fr	–	–	Palpable mass	/	–
		43	23	M	/	8	/	Fr	–	–	Palpable mass	/	–
		44	43	F	/	14	/	T	–	+	Palpable mass	/	–
		45	29	M	/	11	/	Fr	–	–	Palpable mass	/	–
		46	67	F	/	23	/	P	–	+	Palpable mass	/	–
		47	43	M	/	10	/	T	–	–	Palpable mass	/	–
		48	53	F	/	8	Saccular	T	–	–	Palpable mass	+	–
		49	30	M	/	15	/	Fr	–	+	Palpable mass	/	–
		50	46	M	/	13	/	Fr	–	–	Palpable mass	/	–
		51	39	F	/	12	/	P	–	–	Palpable mass	/	–
		52	25	M	/	7	/	T	–	–	Palpable mass	/	–
Pejkić *et al*., 2014	English	53	53	M	+	8	Saccular	P	–	–	Palpable mass	–	–
Matkovski *et al*., 2015	English	54	69	M	/	10	Saccular	Fr	–	–	Palpable mass	–	–
Delen *et al*., 2016	English	55	79	F	+	5	Saccular	T	Two IA	/	Aneurysmal SAH	–	–
Kato *et al*., 2017	Japanese	56	76	F	–	26	Saccular	T	AAA	–	Palpable mass	–	–
Kotsis *et al*., 2018	English	57	67	M	/	20	Fusiform	Fr	–	–	Palpable mass	–	–
Chapman *et al*., 2019	English	58	75	M	+	7	Fusiform	Fr	–	+	Palpable mass	–	–
Kitamura and Horiuchi 2020	English	59	70	F	/	20	Saccular	T	Three RAA	–	Palpable mass	+	–
Abdelhedy *et al*., 2020	English	60	43	M	–	20	Fusiform	T	–	–	Palpable mass	+	–
Mahajan *et al*., 2021	English	61	13	M	–	/	Fusiform	P	Two IA	–	Palpable mass	–	–
Brummund *et al*., 2021	English	62	49	M	+	6	Fusiform	Fr	–	–	Palpable mass	–	–
Present case, 2022	English	63	63	F	+	5	Saccular	P	–	–	Asymptomatic	–	–

AAA: Abdominal aortic aneurysm, F: Female, Fr: Frontal branch, HT: Hypertension, IA: Intracranial aneurysm, ICAA: Internal carotid artery aneurysm (extracranial), M: Male, OAA: Occipital artery aneurysm, P: Parietal branch, RAA: Renal artery aneurysm, SAH: Subarachnoid hemorrhage, STAA: Superficial temporal artery aneurysm, T: Main trunk, ZO: Zygomatico-orbital branch

Supplementary Table 2: Standardized form for examinations and treatments.

First author, yr	Case No.	Radiological Modalities	Pathological examination	Etiology (Histological type)	Wall thickness	Treatment	Treatment complication
Brown, 1942	1	–	+	/	Thinning	Resection	–
Martin, 1955	2	–	+	Atherosclerosis	Thickening	Resection	–
Buckspan, 1986	3	US	+	Atherosclerosis	Thickening	Resection	–
Locatelli, 1988	4	Angiography	+	/	Thinning	Resection	–
Ikeda, 1988	5	Angiography	+	Cystic medial necrosis	Thickening	Resection	–
Nishioka, 1988	6	Angiography	+	/	Thickening	Resection	–
Dailey, 1994	7	–	+	Atherosclerosis	Thickening	Resection	–
Nishimura, 1996	8	Angiography	–	/	/	Resection	–
Uchida, 1999	9	Angiography	+	Atherosclerosis	Thickening	Resection	–
Endo, 2000	10	Angiography	+	Atherosclerosis	Thickening	Resection	–
	11	Angiography	+	Atherosclerosis	Thickening	Resection	–
Porcellini, 2001	12	–	+	/	/	Resection	–
Ohta, 2003	13	Angiography	+	Segmental arterial mediolysis	Thickening	Resection	–
Riaz, 2004	14	US	+	/	Thickening	Resection	–
	15	US	+	/	/	Resection	–
Coenegrachts, 2005	16	US, CTA, DSA	–	/	/	Embolization	–
Silverberg, 2005	17	MRA	+	/	Thickening	Resection	–
Ysa, 2008	18	US, MRA	+	Atherosclerosis	/	Resection	–
Gull, 2009	19	US	+	/	Thinning	Resection	–
Piffaretti, 2009	20	US	+	Atherosclerosis	/	Resection	–
	21	US	+	/	/	Resection	–
Kawabori, 2009	22	CTA	+	/	Thickening	Resection	–
Wise, 2009	23	US, DSA	+	/	Thickening	Resection	–
	24	US, DSA	+	/	/	Resection	–
Karam, 2010	25	CTA	+	/	Thickening	Resection	–
Bozkurt, 2011	26	–	+	Atherosclerosis	/	Resection	–
Labropoulos, 2011	27	/	–	/	/	Resection	–
Moriyama, 2011	28	CTA	+	Intravascular papillary endothelial hyperplasia	Thickening	Resection	–
Sakamoto, 2011	29	MRA	+	/	Thickening	Resection	–
Nair *et al.*, 2011	30	US	–	/	/	Resection	–
Mousa *et al.*, 2011	31	US	+	/	/	Resection	–
Wei and Akkersdijk, 2011	32	CTA	–	/	/	Resection	–
Lim *et al.*, 2012	33	CTA	+	/	Thinning	Resection	–
Molinaro, 2012	34	US, CTA	+	/	/	Resection	–
Sloane *et al.*, 2013	35	US	+	Mucin and myxoid degeneration	Thickening	Resection	–
Joshi and Klimczak 2014	36	CTA	+	/	/	Resection	–
López-Ruiz *et al.*, 2014	37	US	+	Atherosclerosis	/	Resection	–
Kawai *et al.*, 2014	38	US, MRA, DSA	+	/	Thinning	Resection	–
	39	US, MRA, CTA	+	Mucoid degeneration in the media	Thinning	Resection	–
	40	US, CTA	+	/	Thickening	Resection	–
Kim, 2014	41	US, CTA	+	/	/	Resection	–
	42	US, MRA, CTA	+	/	/	Resection	–

(Contd...)

Supplementary Table 2: *(Continued)*.

First author, yr	Case No.	Radiological Modalities	Pathological examination	Etiology (Histological type)	Wall thickness	Treatment	Treatment complication
	43	US, CTA	+	/	/	Resection	–
	44	CTA	+	/	/	Resection	–
	45	US, CTA	+	/	/	Resection	–
	46	US, MRA, CTA	+	/	/	Resection	–
	47	CTA	+	/	/	Embolization	–
	48	CTA	+	/	Thickening	Resection	–
	49	US, DSA	+	/	/	Resection	–
	50	US, DSA	+	/	/	Resection	–
	51	US, DSA	+	/	/	Resection	–
	52	US, DSA	+	/	/	Resection	–
Pejkić *et al.*, 2014	53	US, CTA	+	Segmental arterial mediolysis	Thinning	Resection	–
Matkovski *et al.*, 2015	54	US	+	Atherosclerosis	Thickening	Resection	–
Delen *et al.*, 2016	55	CTA	–	/	/	Resection	–
Kato *et al.*, 2017	56	CTA	+	Atherosclerosis	Thinning	Resection	–
Kotsis *et al.*, 2018	57	US	+	Atherosclerosis	/	Resection	–
Chapman *et al.*, 2019	58	–	+	Intravascular papillary endothelial hyperplasia	Thickening	Resection	–
Kitamura and Horiuchi, 2020	59	Angiography	+	Segmental arterial mediolysis	Thickening	Resection	–
Abdelhedy *et al.*, 2020	60	US, CTA	+	/	/	Resection	–
Mahajan *et al.*, 2021	61	Angiography	–	/	/	Lost to follow	/
Brummund *et al.*, 2021	62	CTA	+	/	/	Resection	–
Present case, 2022	63	MRA, CTA, DSA	+	/	Thinning	Clipping	–

CTA: Computed tomography angiography, DSA: Digital subtraction angiography, MRA: Magnetic resonance angiography, US: Ultrasonography