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Management strategy for extracranial carotid artery aneurysms
A single-center experience

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Abstract

This single-center, retrospective study aimed to describe the anatomic and clinical characteristics of extracranial carotid artery aneurysms (ECAAs) and to compare various ECAA management strategies in terms of outcomes.

A total of 41 consecutive patients, who underwent treatment for ECAAs between November 1996 and May 2020, were included in this study. The ECAAs were anatomically categorized using the Attigah and Peking Union Medical College Hospital (PUMCH) classifications. The possible study outcomes were restenosis or occlusion of the ipsilateral carotid artery after treatment and treatment-associated morbidity or mortality.

The 41 patients were stratified into three groups according to the management strategies employed: surgical (n = 25, 61.0%), endovascular (n = 10, 24.4%), and conservative treatment (n = 6, 14.6%). A palpable, pulsatile mass was the most common clinical manifestation (n = 16, 39.0%), and degenerative aneurysms (n = 29, 65.9%) represented the most common pathogenetic or etiological mechanism. According to the Attigah classification, type I ECAAs (n = 24, 58.5%) were the most common. Using the PUMCH classification, type I ECAAs (n = 26, 63.4%) were the most common. There was a higher prevalence of Attigah type I ECAAs among patients who underwent surgical treatment compared with those who underwent endovascular treatment (64.0% vs 40.0%, P = .09), whereas patients with PUMCH type IIa aneurysms were more likely to receive endovascular treatment (12.0% vs 30.0%). False aneurysms were more likely to be treated using endovascular techniques (20% vs 70%, P = 0.02). Except for two early internal carotid artery occlusions (one each among patients who underwent surgical and endovascular treatments, respectively), there were no early or late restenoses or occlusions during follow-up. Cranial nerve injuries were noted in three patients after surgical treatment, and late ipsilateral strokes occurred in two patients (one each among patients who underwent endovascular and conservative treatment, respectively). There were no other treatment-associated complications or deaths during the study period.

Conclusions: Both surgical and endovascular treatments could be performed safely for ECAAs with good long-term results according to anatomic location and morphology.

Abbreviations: CCA = common carotid artery, CEA = carotid endarterectomy, DUS = duplex ultrasound, ECAA = extracranial carotid artery aneurysm, ICA = internal carotid artery, IQR = interquartile range, PSV = peak systolic velocity, PUMCH = Peking Union Medical College Hospital.

Keywords: aneurysm, carotid artery, management

1. Introduction

Extracranial carotid artery aneurysms (ECAAs) represent an uncommon but important disease entity, which can result from various causes, such as atherosclerotic degeneration, local infection, traumatic injury, radiation, dissection, or as a complication after carotid endarterectomy (CEA).[1–3] Given

the risk of aneurysm rupture as well as the neurologic sequelae of cerebral thromboembolism, surgical intervention is recommended for ECAAs to prevent complications.[1–7] A variety of operative techniques for treating ECAAs have been described with good long-term outcomes.[4] Recently, due to advances in endovascular techniques, less-invasive endovascular treatment modalities have been introduced with high technical success rates and favorable short- and intermediate-term outcomes.[8,9] However, because ECAAs are rare, there are few well-designed cohort or prospective studies investigating ECAA-related topics.[10] Most publications reporting on ECAAs are case reports or case series, and there is no consensus on the management of ECAAs, in part because of a lack of supporting evidence.[11]

This single-center, retrospective study aimed to describe the anatomic and clinical characteristics of ECAAs and to compare various ECAA management strategies in terms of outcomes.

2. Patients and methods

2.1. Study design and data collection

This single-center, retrospective observational study was conducted using data extracted from the medical records of patients who were diagnosed with ECAAs at our hospital. Approval for data collection and publication was granted by our hospital's institutional review board (IRB No. 2020-1002), which waived the requirement for written informed consent because of the study's retrospective design. All methods were performed in accordance with the relevant guidelines and regulations.

A total of 41 consecutive patients, who underwent treatment for ECAAs between November 1996 and May 2020, were included in this study. Based on the definition proposed by de Jong et al,[12] an ECAA was defined as a bulb dilatation greater than 200% of the diameter of the internal carotid artery (ICA) or 150% of the diameter of the common carotid artery (CCA). In our study, the Attigah and Peking Union Medical College Hospital (PUMCH) classifications were used to categorize ECAAs. Aneurysm locations were classified according to the Attigah classification.[4,13] In terms of anatomic location and morphology, aneurysms were classified, according to the PUMCH classification, into four categories by whether the aneurysm was below or above the Blaisdell line, or whether it was kinked or not.[5]

The demographic characteristics, risk factors of interest, anatomic and clinical characteristics, management strategies, and outcomes for all consecutive patients were recorded in an Excel (Microsoft Corp, Redmond, WA) database and analyzed retrospectively.

2.2. Selection of treatment

The selection of management strategies was determined mainly based on the location and morphology of an ECAA, pathogenesis, and each patient's preferences and general health status. Basically, surgery was indicated if there was a degenerative ECAA associated with a tortuous ICA, if there was an unstable-looking thrombus within an aneurysm, or if there was a mycotic aneurysm. On the other hand, traumatic or radiation-induced false aneurysms, or large aneurysms involving the more distal ICA, were indicated for endovascular treatment.[14,15] Treatment choices were made using a multidisciplinary approach involving vascular surgeons, interventional radiologists, and neurologists.

Surgical treatment involved resection of the aneurysm with restoration of the cerebral blood flow using interposition bypass or end-to-end anastomosis, resection of the aneurysm with primary repair, trapping of the ICA with extracranial-to-intracranial bypass,[16] or resection of the aneurysm with ligation of the distal ICA.[17] Endovascular treatment was performed with the exclusion of the aneurysm sac and preservation of the cerebral blood flow, covered stent placement, embolization with or without stent placement, and flow-diverting braided stent placement or thrombin injection.[9] After surgical or endovascular treatments, all patients were given antiplatelet therapy with a statin in combination with stringent blood pressure control and close observation in an intensive care unit for at least 24 hours. All patients were followed up, both clinically and using computed tomographic angiography or carotid duplex ultrasound (DUS), before discharge. Patients who refused to undergo invasive treatments and patients who had small dissecting aneurysms (<1.5 cm) were treated conservatively with antiplatelet therapy with a statin and close observation.

2.3. Study outcomes and follow-up

The possible study outcomes were restenosis or occlusion of the ipsilateral carotid artery after treatment and treatment-associated complications including bleeding, cranial nerve injury, or fatal or nonfatal stroke ipsilateral to the treated carotid aneurysm. The complications were categorized as early (within 30 days after treatment) or late (>30 days after treatment). Restenosis was defined as the development of a ≥50% diameter reduction, diagnosed on the basis of DUS findings of luminal narrowing and velocity criteria with a peak systolic velocity (PSV) threshold ≥125 cm/s or an ICA/CCA PSV ratio >2.0.[24] Neurologic events were defined as previously detailed.[18,19]

Follow-up visits, with physical and laboratory evaluations and carotid DUS (iU22, Philips Ultrasound, Bothell, WA), were scheduled at 1, 6, and 12 post-treatment months and annually thereafter. Once stability had been established for over 3 years, surveillance was performed at longer intervals of about 2 years.

2.4. Statistical analyses

Continuous data are presented as medians and interquartile ranges (IQRs), and categorical data are presented as frequencies or percentages. Categorical variables were compared using the chi-square test, whereas continuous variables were compared using Student's t test. A P-value <.05 was considered statistically significant. Statistical analyses were performed using PASW Statistics for Windows, version 18.0 (SPSS Corp, Chicago, IL).

3. Results

A total of 41 patients with ECAAs were enrolled in this. Eligible patients were stratified into three groups according to the management strategy: the surgical (n=25, 61.0%), endovascular (n=10, 24.4%), and conservative treatment groups (n=6, 14.6%) (Fig. 1).

The baseline and clinical characteristics of the study sample, including six patients who received conservative treatment, are presented in Table 1. Their mean age was 57.0 years (median, 57 years; IQR, 48–67 years), and 61.0% of the patients were females. Relevant comorbidities included hypertension (n=13, 31.7%), diabetes mellitus (n=2, 4.9%), and dyslipidemia (n=
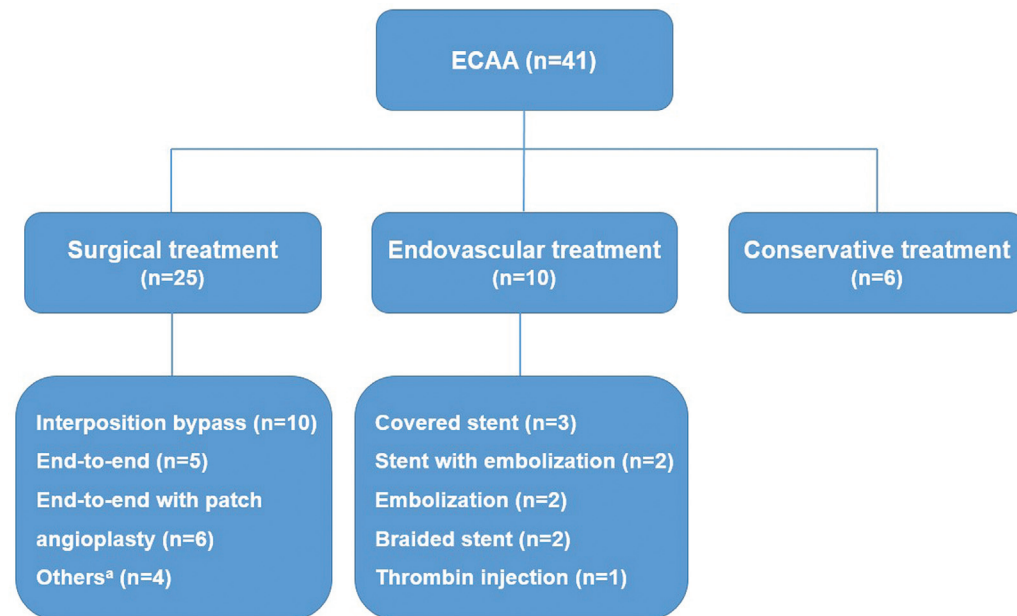
Figure 1. Flow chart of patient inclusion. Forty-one patients with the diagnosis of ECAA were included in the analysis. [a]Included two resected aneurysms with primary repair, one trapping of the internal carotid artery with extracranial-to-intracranial bypass, and one aneurysm resection with ligation of the distal internal carotid arteries. ECAA = extracranial carotid artery aneurysm.

17, 41.5%). There were 10 patients (24.4%) with previous cerebrovascular accidents: five were recent (within 6 months), and five were remote (>6 months). Their median aneurysm size was 2.5 cm (IQR, 1.4–3.2 cm). A palpable, pulsatile, submandibular mass was the most common clinical manifestation, noted in 16 patients (39.0%); headache was noted in seven patients (14.1%), and seven patients (14.1%) were incidentally diagnosed with ECAAs. Degenerative aneurysms (n=29, 65.9%) represented the most common pathogenetic mechanism.

There were no significant differences between patients who underwent surgical versus endovascular treatments in terms of demographic characteristics, risk factors, or clinical characteristics, except that the patients who underwent endovascular treatment were more likely to have had comorbid cancer (0% vs 30.0%, $P = .02$). With regard to causes of ECAAs, degenerative aneurysms were more likely to be common among patients who received surgical treatment than among those who received endovascular treatment (80.0% vs 40.0%, $P = .01$).

The anatomic locations and the morphology of the aneurysms among the study sample are presented in Table 2. With regard to the ECAA location classified by Attigah classification, the most common were type I ECAAs, located distal to the carotid bifurcation (n=24, 58.5%). Attigah type IV and V aneurysms involving the CCA were noted in 10 patients (24.4%), and most of them (n=8) were traumatic or radiation-induced false aneurysms. There was a higher prevalence of type I ECAAs among patients who underwent surgical treatment compared with those who underwent endovascular treatment, with a non-significant trend ($P = .09$). Based on the PUMCH classification, type I ECAAs, located below the Blaisdell line, were noted in 26 patients (63.4%), and type II ECAAs, located above the Blaisdell line, were noted in 15 patients (36.6%). Although the number of type IIa aneurysms (n=6) with documented locations above the Blaisdell line in the absence of kinking was too low to analyze the correlation of the anatomic location and morphology of the

aneurysm with management strategy, patients with type IIa aneurysms were more likely to have undergone endovascular treatment (12.0% vs 30.0%). Patients with false aneurysms were more likely to have been treated with endovascular techniques (20% vs 70%, $P = .02$).

Twenty-five patients underwent surgical treatments using various techniques of cerebral blood flow restoration according to the morphology of the aneurysm and the proximity of the two ends of the remnant carotid artery after aneurysm resection (Fig. 2). Ten patients underwent aneurysm resection with interposition bypass using autologous saphenous vein (n=6) or prosthetic (n=4) grafts. Aneurysm resection with end-to-end anastomosis was performed for 11 patients; among them, in six patients, patch angioplasty procedures were performed using processed bovine pericardium (Vascu-Guard; Bio-Vascular Inc, Saint Paul, MN) for the prevention of restenosis due to the small caliber of the anastomosis site. Other surgical treatments were resection of the aneurysm with primary repair (n=2), trapping of the ICA with extracranial-to-intracranial bypass (n=1), and resection of the aneurysm with ligation of the distal ICA (n=1). Endovascular treatment was performed for 10 patients: covered stent placement (n=3), embolization with or without stent placement (n=4), flow-diverting braided stent placement (n=2), and thrombin injection (n=1).

Study outcomes for the patients who underwent surgical or endovascular treatments are summarized in Table 3. Except for two early ICA occlusions (one each among patients who received surgical and endovascular treatments, respectively), there were no early or late restenoses or occlusions during follow-up. Early and late ipsilateral strokes did not occur among patients who received surgical treatment, whereas a late ipsilateral stroke (1 year after treatment) occurred in one patient who had undergone radiation therapy due to a recurrent tonsillar cancer; this patient had undergone endovascular treatment for a false aneurysm. Cranial nerve injury was noted in three patients after surgical

Table 1

Baseline and clinical characteristics of the study sample according to management strategy (surgical vs endovascular treatment).

	Total[*]	Surgical	Endovascular	P
Patients (n)	41	25 (61.0)	10 (24.4)	
Age (year)	57 (48–67)	56 (47–67)	63 (54–75)	.20
Female sex	25 (61.0)	18 (72.0)	4 (40.0)	.12
Atherosclerosis risk factor				
Hypertension	13 (31.7)	10 (40.0)	2 (20.0)	.43
Diabetes mellitus	2 (4.9)	1 (4.0)	1 (10.0)	.50
Dyslipidemia	17 (41.5)	7 (28.0)	6 (60.0)	.12
Medical history				
CAD	1 (2.4)	1 (4.0)	0	>.99
CVA[†]	10 (24.4)	5 (20.0)	2 (20.0)	>.99
CKD	2 (4.9)	1 (4.0)	1 (10.0)	.50
COPD	4 (9.8)	2 (9.1)	1 (10.0)	>.99
Comorbid cancer[‡]	3 (7.3)	0	3 (30.0)	.02
Aneurysm size (cm)	2.5 (1.4–3.2)	2.7 (1.5–3.1)	1.7 (0.5–5.1)	.32
Clinical or diagnostic feature				
Pulsatile mass	16 (39.0)	13 (52.0)	2 (20.0)	0.39
Headache	7 (14.1)	3 (12.0)	2 (20.0)	
Incidental	7 (14.1)	4 (16.0)	2 (20.0)	
Recent stroke (≤6 months)	5 (12.2)	2 (8.0)	1 (10.0)	
Pain	3 (7.3)	2 (8.0)	1 (10.0)	
Other[§]	3 (7.3)	1 (4.0)	2 (20.0)	
Cause				
Degenerative	29 (65.9)	20 (80.0)	4 (40.0)	.01
Mycotic	1 (2.4)	1 (4.0)	0	
Traumatic	5 (12.2)	2 (8.0)	2 (20.0)	
Vasculitis	2 (4.9)	2 (8.0)	0	
Radiation	3 (7.3)	0	3 (30.0)	
Unknown[‖]	3 (7.3)	0	1 (10.0)	
Follow-up (months)	33 (10–89)	52 (13–94)	14 (0–70)	.11

Continuous data are presented as medians and interquartile ranges; categorical data are presented as numbers (%).

CAD = coronary artery disease, CKD = chronic kidney disease, COPD = chronic obstructive pulmonary disease, CVD = cerebrovascular disease.

[*] Includes six patients who received conservative treatment.

[†] Includes five patients with remote stroke events (>6 months).

[‡] One for each of laryngeal cancer, tonsillar cancer, and pharyngeal cancer.

[§] Includes tinnitus, oral bleeding, and dysphagia.

[‖] Dissecting aneurysms of unknown etiology.

Table 2

Anatomic classification of the study sample according to management strategy (surgical versus endovascular treatment).

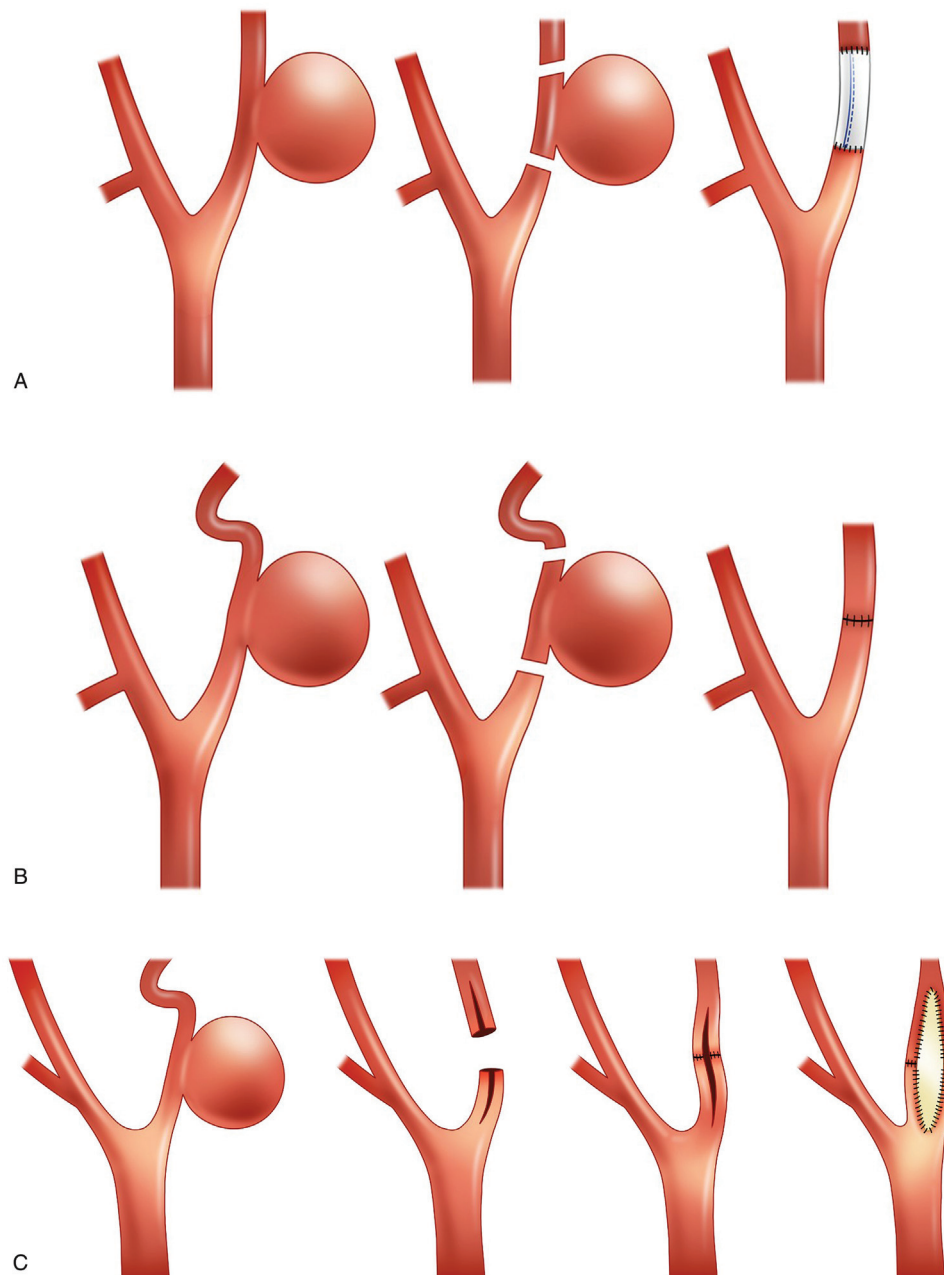|	Total[*]	Surgical	Endovascular	P
Patients (n)	41	25 (61.0)	10 (24.4)	
Attigah classification				
I	24 (58.5)	16 (64.0)	4 (40.0)	.09
II	5 (12.2)	3 (12.0)	0	
III	2 (4.9)	2 (8.0)	0	
IV	4 (9.8)	1 (4.0)	3 (30.0)	
V	6 (14.6)	3 (12.0)	3 (30.0)	
PUMCH classification				
Ia	12 (29.3)	7 (28.0)	5 (50.0)	.12
Ib	14 (34.1)	12 (48.0)	1 (10.0)	
IIa	10 (24.4)	3 (12.0)	3 (30.0)	
IIb	5 (12.2)	3 (12.0)	1 (10.0)	
Structure				
True	26 (63.4)	20 (80.0)	3 (30.0)	.02
False	15 (36.6)	5 (20.0)	7 (70.0)	

Data are presented as numbers (%).

PUMCH = Peking Union Medical College Hospital.

[*] Includes six patients who received conservative treatment.

4. Discussion

Compared with atherosclerotic occlusive disease of the carotid arteries or aneurysms involving the intracranial carotid arteries and their branches, ECAAs are rare, with the true incidence accounting for less than 1% of all carotid pathologies.[20] ECAAs can result from various causes, such as atherosclerotic degeneration, local infection, traumatic injury, dissection, or as a complication after CEA.[1–3] Owing to the rarity and many different causes of ECAAs, controversy exists about the optimal management strategy, and multiple treatment options are available based on the location, size and morphology, and cause of the aneurysm, as well as consideration of the overall condition of the patient. Although results vary widely depending on the anatomic and morphological characteristics of the aneurysm, surgical treatment of most ECAAs is feasible and is associated with high success rates and acceptable rates of neurologic complications. Attigah et al reported a single-center study of 64 carotid reconstructions in 57 patients with long-term follow-up over 24 years; they showed that surgical therapy for ECAAs is feasible with good long-term results.[4] The treatment of ECAAs has evolved during the last decade, and endovascular treatment has been recognized as an effective and less-invasive treatment modality for select patients, with the advantage of avoiding potential difficulties with surgical dissection and eliminating the need for high cervical exposure, thus reducing the risk of cranial nerve injuries and other possible surgery-associated complications.[8–11] The natural history of ECAAs managed by observation is poorly defined. Given the likelihood of neurologic symptoms and the risk of aneurysm-associated permanent adverse neurologic events, a conservative approach to ECAAs cannot be justified in the vast majority of cases.

In the present study, we determined optimal intervention strategies based on aneurysm location, size, morphology, and cause, in addition to the overall condition of the patient. Although there is no universally accepted ECAA classification model due to the rarity of ECAAs, two ECAAs classifications were developed with respect to aneurysm location and morphology: the Attigah[4] and PUMCH[5] classifications.

treatment, and there were no other treatment-associated complications, including bleeding or death among patients who underwent surgical and endovascular treatments during the study period.

Among the six patients who received conservative treatment, three patients with degenerative aneurysms had refused to undergo invasive treatments. Another three patients with small dissecting aneurysms (<1.5 cm) were determined to receive medical treatments. Their median aneurysm size was 1.3 cm (IQR, 0.8–2.4 cm). Clinical details, including medications and outcomes, are summarized in Table 4. During follow-up, one ipsilateral stroke occurred in a patient with a degenerative aneurysm. One traumatic dissecting aneurysm resolved spontaneously during follow-up, and two small dissecting aneurysms of unknown causes showed no morphological changes and no complications during follow-up periods of 33 months and 7 months, respectively. Compared with patients who underwent either surgical or endovascular treatment, the risk of an ipsilateral stroke was significantly higher among patients who refused to undergo invasive treatments (1/35, 2.9% vs 1/3, 33.3%, P = .02).

Figure 2. Schematic representative figures of surgical treatment. (A) Resection of aneurysm with interposition bypass, (B) resection of aneurysm with end-to-end anastomosis, and (C) resection of aneurysm with patch angioplasty.

Attigah et al proposed five different types of ECAAs based on the anatomic location and length of the aneurysm.[4] The PUMCH classification defined four different types of ECAAs based on the anatomic location of the aneurysm and the tortuosity of the proximal artery.[5] In our analysis, there was a higher prevalence of Attigah type I ECAAs—isolated and short aneurysms of the ICA above the carotid bulb—among patients who underwent surgical treatment compared with those who underwent endovascular treatment, whereas patients with PUMCH type IIa aneurysms—aneurysms above the Blaisdell line in the absence of proximal artery kinking—were more likely to have undergone

endovascular treatment. In terms of the cause of aneurysm, degenerative aneurysms were commonly indicated for surgical treatment, whereas endovascular treatments were generally performed for traumatic or radiation-induced false aneurysms.

Surgical treatment has been the mainstay treatment modality for ECAAs for years, but endovascular treatment has also been shown to be effective.[7] For surgical treatment, whenever possible, resection of the aneurysm and restoration of the cerebral blood flow should be carried out.[5] In our series, one patient underwent ICA ligation because of an infected false aneurysm of the distal ICA. The decision on whether to use an

Table 3

Clinical outcomes of the study sample according to management strategy (surgical versus endovascular treatment).

	Surgical (n=25)	Endovascular (n=10)
ICA occlusion		
≤30 days	1 (4.0)	1 (10.0)
>30 days	0	0
Ipsilateral stroke		
≤30 days	0	0
>30 days	0	1 (10.0)
Cranial nerve injury		
Transient	2 (8.0)	0
Permanent	1 (4.0)	0
30-days mortality	0	1 (10.0)

Data are presented as numbers (%).
ICA = internal carotid artery.

end-to-end anastomosis was based on tissue quality and the possibility of tension-free adaptation.[5] Aneurysm resection with end-to-end anastomosis was performed for 11 patients in our series; among them, six patients underwent additional patch angioplasty procedures to prevent restenosis due to the small caliber of the anastomosis site, and there were no early or late restenoses or occlusions during follow-up. The most frequently encountered neurologic complications associated with surgical ECAA treatment are cranial nerve injuries, which occur transiently in 11% to 22% of cases and permanently in 3% to 13% of cases.[2,4–7] In our series, the transient and permanent cranial nerve injury rates were 8.0% and 4.0%, respectively. The reported incidence of transient focal neurologic events ranges from 3% to 17%, and the incidence of permanent, non-incapacitating neurologic deficits ranges between 1.5% and 6%.[2,5,20–22] Although only a small number of patients who underwent surgical treatment were included in our series, there were no early or late ipsilateral neurologic events in our analysis. Even though the surgical treatment of ECAAs poses treatment challenges due to possible surgical complications and frequently inaccessible locations, our results suggest that surgical treatment is a safe and effective management strategy for accessible ECAAs.

With recent advances in endovascular techniques, endovascular treatment is a less-invasive procedure and has shown promising technical success for most arterial aneurysmal diseases.[23,24] Several endovascular techniques have been reported for the treatment of ECAAs.[8,23,24] A previous

systematic review by Li et al. demonstrated a 2% perioperative incidence of stroke and a 93% stent patency rate during a mean follow-up of 15 months.[8] However, similar to most existing reports of endovascular treatment—most of which are retrospective case series or case reports—the number of patients studied in our series was small, and further prospective studies are needed to evaluate the long-term outcomes and durability of endovascular treatment for this disease.

Untreated ECAAs can lead to compression of cranial nerves causing focal neurologic symptoms,[13] or they can progress to distal embolization or complete occlusion, causing devastating neurologic deficits.[25,26] Conservative treatment for ECAAs with anticoagulants or antiplatelet agents might lower the incidence of ischemic cerebrovascular events; however, it does not resolve the actual lesion, maintaining the substantial risk of thrombus formation and distal embolization.[23,27,28] Therefore, a more invasive procedure, either surgical or endovascular, may be warranted for optimal late-term outcomes. In our series, among the three patients who had degenerative ECAAs and refused to receive invasive treatment, one ipsilateral stroke occurred at 6 months after the ECAA diagnosis.

This study had some limitations. First, the retrospective nature of this single-center study made it subject to selection and information biases. Therefore, the decisions to perform surgical or endovascular treatment and the choice of method among various operative or endovascular techniques for ECAA were mainly made by the physician based on the expected level of technical difficulty of the procedure. Second, our current findings were obtained at a single center, resulting in a small sample size in each of the treatment groups. The low number of cases precluded the execution of detailed statistical analysis, and a small number of events, which limits the overall relevance of our results; this study was likely underpowered to provide sufficient supporting evidence for our results. Finally, our study cohort comprised only subjects of Asian descent; thus, because there may be genetic disparities in the pathophysiology and prevalence of ECAAs, our findings should be cautiously interpreted with respect to different racial or ethnic groups.

In conclusion, despite the potential limitations, our results suggest that both surgical and endovascular treatments could be performed safely with good long-term results for treating ECAAs according to their anatomic location and morphology.

Table 4

Clinical details and outcomes of the six patients who received conservative treatment.

	Sex/age	Pathogenesis	Attigah classification	PUMCH classification	Clinical symptoms	Follow-up (months)	Clinical outcomes
1*	M/65	Degenerative	II	IIa	Visual disturbance	68	No change
2	F/66	Degenerative	I	IIb	Mass	139	No change
3*,†,‡	F/64	Degenerative	I	Ib	Incidental	6	Stroke
4*	M/53	Traumatic	I	IIa	Headache	150	Resolved
5*	M/37	Dissecting	I	IIa	Headache	33	No change
6*,†	F/53	Dissecting	II	IIa	Visual disturbance	7	No change

PUMCH = Peking Union Medical College Hospital.
* Received antiplatelet agent.
† Received statin.
‡ Received antihypertensive agent.

Author contributions

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