

<https://helda.helsinki.fi>

Etiology and treatment patterns of ruptured extracranial carotid artery aneurysm

de Souza, Nicole Martins

2021-12

de Souza , N M , Vikatmaa , P , Tulamo , R & Venermo , M 2021 , ' Etiology and treatment patterns of ruptured extracranial carotid artery aneurysm ' , Journal of Vascular Surgery , vol. 74 , no. 6 , pp. 2097-+ . <https://doi.org/10.1016/j.jvs.2021.06.023>

<http://hdl.handle.net/10138/339740>

<https://doi.org/10.1016/j.jvs.2021.06.023>

cc_by_nc_nd

publishedVersion

Downloaded from Helda, University of Helsinki institutional repository.

This is an electronic reprint of the original article.

This reprint may differ from the original in pagination and typographic detail.

Please cite the original version.

Etiology and treatment patterns of ruptured extracranial carotid artery aneurysm

Nicole Martins de Souza, MD, Pirkka Vikatmaa, MD, PhD, Riikka Tulamo, MD, PhD, and Maarit Venermo, MD, PhD, *Helsinki, Finland*

ABSTRACT

Objective: Rupture of an extracranial carotid artery aneurysm (ECAA) is a very rare and life-threatening condition. To obtain a comprehensive view of previous and current management of ruptured ECAs (rECAs), we analyzed all cases reported since 1940 and two of our own cases.

Methods: We performed a comprehensive literature review of reports from the MEDLINE database on rECAs and included two patients treated in our department.

Results: A total 58 reports of 74 rECAs in 74 patients were analyzed. Their mean age was 50 years, and the male/female ratio was 2.2:1. Infection was the most common reported etiology (19 of 74; 26%), followed by connective tissue disorder (13 of 74; 18%), atherosclerosis (9 of 74; 12%), and previous trauma (5 of 74; 7%). For 28 patients (38%), information on the etiology was not available. Of the 74 patients, 24 (32%) had undergone reconstructive surgery, 10 (14%) had undergone endovascular treatment, 17 (23%) had undergone ligation, 2 (3%) had been treated conservatively, and 1 (1%) had died before receiving definite treatment. For 20 patients (27%), information on the treatment received was not available. The complications after reconstruction included carotid blowout (3 of 24 patients; 13%) and cranial nerve deficit (3 of 24 patients; 13%). Two patients (8%) had died of unrelated ECAA causes during long-term follow-up, and one patient (4%) had died of an ECAA-related cause within 30 days. After an endovascular approach, 1 of the 10 patients had developed a cranial nerve deficit. After ligation, five patients (29%) had experienced stroke, three of which were fatal. One conservatively treated patient had experienced no complications and one had died of an ECAA-related cause.

Conclusions: The most common reported etiology for rECAA was infection. Reconstructive surgery was the most common approach and was safer than ligation, which carried a high risk of stroke. Endovascular treatment showed promising results, especially for distally located aneurysms; however, the number of patients has remained low. (*J Vasc Surg* 2021;74:2097-103.)

Keywords: Aneurysm; Carotid aneurysm; Carotid artery injury; Rupture; Spontaneous

Rupture of an extracranial carotid artery aneurysm (ECAA) can be considered a very rare situation that a vascular surgeon encounters never or only a couple of times during their professional career.¹⁻³ Although rupture is not the most common complication of an ECAA, it can lead to life-threatening hemorrhage.⁴⁻⁶ The findings from modern imaging are more likely to reveal such aneurysms

more often and can guide the diagnosis and treatment method, even in the acute setting, differentiating ECAs from kinked carotid arteries, tumors, abscesses, and cysts, and providing a good anatomic view. Most reported studies have been case reports that have included only a few cases of ruptured ECAs (rECAs) among a majority of unruptured ECAs.

To obtain a comprehensive view of previous and current management of rECAs, the results of the treatment and the etiology of the ruptured aneurysms for all cases reported since 1940 and two of our own cases were analyzed.

METHODS

The present comprehensive literature review was performed in accordance with the recommendations of the PRISMA (preferred reporting items for systematic reviews and meta-analysis) statement.⁷ Two different searches were conducted, first in February 2019 and then repeated in October 2019. We searched the MEDLINE database through PubMed and retrieved pertinent studies found in the reference lists of the selected reports and from other sources. One search included the terms “carotid” AND “aneurysm*” and another “carotid” AND “pseudoaneurysm*” in the title.

From the Department of Vascular Surgery, Abdominal Center, University of Helsinki and Helsinki University Hospital.

Author conflict of interest: none.

Presented at the Finnish Congress of Surgery and Anesthesiology, Helsinki, Finland, November 13-15, 2019, and at the fast-track session for the Programme of the European Society for Vascular Surgery Month in 2020, on behalf of the European Society for Vascular Surgery, Virtual Congress, September 16, 2020.

Additional material for this article may be found online at www.jvascsurg.org.

Correspondence: Nicole Martins de Souza, MD, Department of Vascular Surgery, Abdominal Center, University of Helsinki, Helsinki University Hospital, PO Box 340, Helsinki FI-00029, Finland (e-mail: nicole.martins-de-souza@hus.fi).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214

Copyright © 2021 The Authors. Published by Elsevier Inc. on behalf of the Society for Vascular Surgery. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

<https://doi.org/10.1016/j.jvs.2021.06.023>

Both searches included “rupture” in all fields, with the exclusion criteria as detailed in Fig 1. Early rupture after trauma was defined by a period of <1 month between the trauma and aneurysm rupture or when the period had not been reported. The definition of aneurysm was determined by the use of term “aneurysm” in the original report, because most studies did not report the size of the aneurysm. Because no standardized definition is available for an ECAA, the anatomic criteria were used and comprised the common and external carotid arteries and the cervical portion of the internal carotid artery (below the intrapetrous portion). Rupture was considered present if the terms “rupture” or “tear” had been reported and/or if the clinical signs, radiologic, verification, or histopathologic confirmation of bleeding were reported. In addition, we included two patients treated at our department in the analysis.

RESULTS

A total of 74 rECAs in 74 patients were reported (72 from 56 studies and 2 of our own). The mean age was 50 years (range, 15-89 years), and the male/female ratio was 2.2:1. The mean ECAA maximum diameter was 4.6 cm (range, 0.5-10.0 cm). The etiologies were infection (n = 19; 26%), connective tissue disorder (CTD; n = 13; 18%), atherosclerosis (n = 9; 12%), and previous trauma (n = 5; 7%). For 28 patients (38%), the etiology was unknown or not reported. The mean age stratified by the etiology was 68 years (range, 54-83 years) for atherosclerosis, 32 years (range, 16-56 years) for CTD, 61 years (range, 20-89 years) for mycotic, and 39 years (range, 25-58 years) for a delayed post-traumatic lesion. The most common location was in zone II (41%), followed by zones III (23%) and I (8%; Fig 2).⁸

Of the 19 rECAs reported as mycotic, bacterial cultures were available for 16. The following pathogens were reported according to the bacterial culture findings of the lesion or blood culture: *Staphylococcus* (n = 8), *Mycobacterium* (n = 2), *Salmonella* (n = 2), *Proteus* (n = 1), *Streptococcus* (n = 1), *Klebsiella* (n = 1), and *Pseudomonas* (n = 1).

The reported treatment methods, aneurysm details, and outcomes are summarized in the Table and presented in detail in the Supplementary Table (online only). The aneurysm etiologies in relation to the year of publication are presented in the Supplementary Fig (online only).

Open reconstructive surgery was the most frequently reported treatment method (24 of 74; 32%). For the 24 open surgery cases, the most common etiology was infection (n = 9; 38%), and the most common location was zone II (n = 17; 71%). Three patients (13%) had developed one or more cranial nerve deficits (CNDs). Three patients (13%) had died after reconstructive surgery. One (4%) of an ECAA-related cause within 30 days and two (8%) of an ECAA-unrelated cause at 1 and 36 months after surgery. Three patients (13%) who had undergone reconstruction had experienced carotid artery blowout (one

polytetrafluoroethylene patch and two interpositions; one with a bovine pericardium tube and one with a great saphenous vein (GSV). One of these aneurysms was mycotic, one was due to CTD, and one had an unreported or unknown etiology. One of these patients had died of an ECAA-unrelated cause at 32 days postoperatively with a blowout hematoma diagnosed in the postmortem examination. The other two patients had undergone reoperation: one with ligation and one a GSV interposition. None of the patients who had undergone reconstructive surgery had experienced a stroke. Follow-up information was available for 17 patients who had undergone reconstruction, with a mean follow-up duration of 13 months (range, 1-48 months).

The endovascular (EV) approach was used for 10 patients (14%). Two of the procedures were hybrid procedures. The most common etiology in the EV approach group was late post-traumatic aneurysm (n = 4), of which three caused by a traffic accident (n = 3), and one by a foreign object in the nasal cavity (n = 1). The most common location was zone III (6 of 10). One hybrid procedure for a mycotic pseudoaneurysm included wrapping of the carotid wall with a polytetrafluoroethylene graft, and one had included stent insertion through an open approach. None of the patients experienced a stroke or had died. However, one patient had developed hypoglossal and glossopharyngeal nerve deficits, with persistent hypoglossal, but resolved, glossopharyngeal nerve injury at 18 months. Eight patients had a good recovery related to the ECAA and one patient was lost to follow-up. The mean follow-up duration was 11 months (range, 1-24 months).

Ligation was performed in 17 patients (23%). The most common etiology was infection (n = 6; 35%), and the most common location was zone III (n = 7; 41%). Of these 17 patients, 5 had experienced stroke, three of which were fatal. All the strokes had occurred in patients who had undergone ligation. Two patients, who were intravenous drug users, had had a ruptured mycotic ECAA and had undergone ligation twice because of persistent infection in both and recurrent profuse bleeding in one patient. Neither patient had developed neurologic deficits.

Two patients (3%) were treated conservatively. One patient had presented with dyspnea and hoarseness due to a ruptured pseudoaneurysm. The patient required tracheostomy to prevent airway obstruction. The patient was stable and the hematoma had resolved. After 1 month of follow-up, no complications were reported. The other patient had died of aneurysm rupture that had been treated palliatively.

Of the 74 patients, 9 (12%) had died. Of the nine deaths, six were related to the ECAA and three were not related to the ECAA. Of the six ECAA-related deaths, three had occurred after ligation of the carotid artery. One was due to CND and aspiration pneumonia, followed by GSV interposition with mandibulotomy. One of the patients

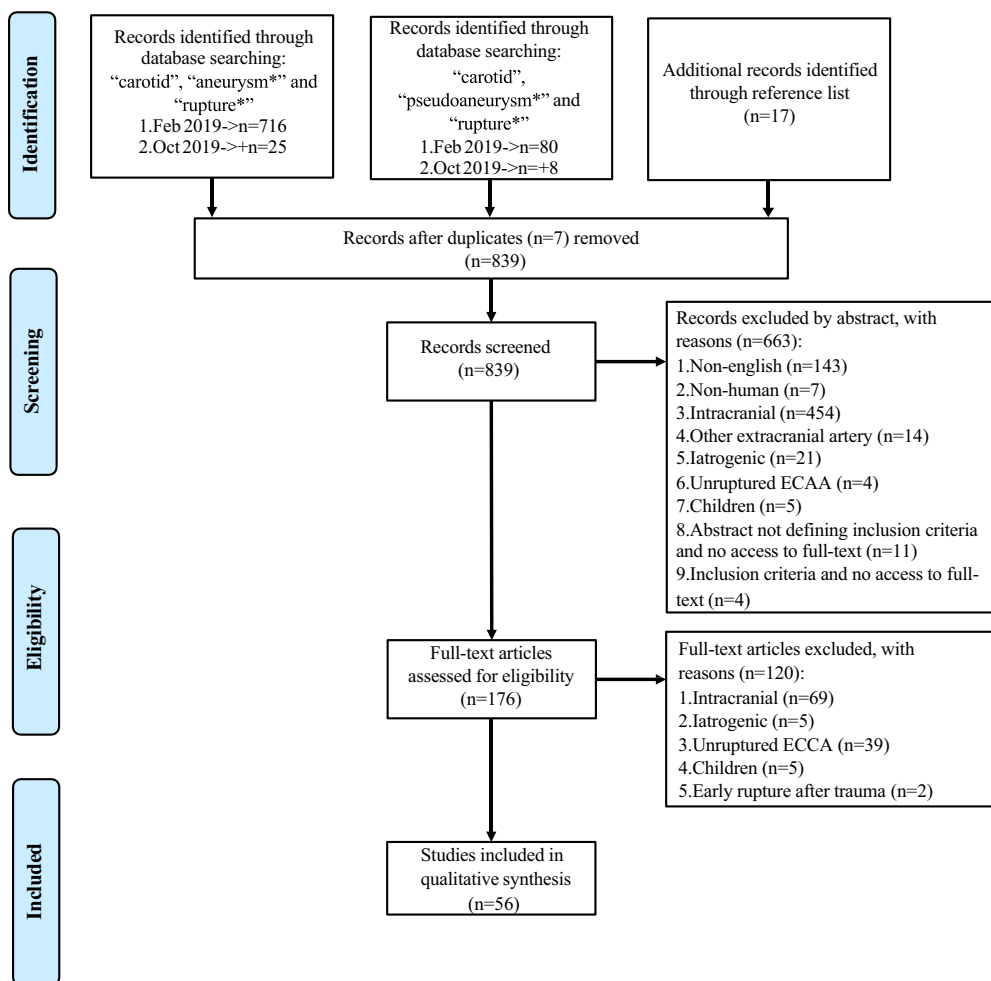


Fig 1. PRISMA (preferred reporting items for systematic reviews and meta-analysis) flow diagram of the comprehensive literature review for ruptured extracranial carotid artery aneurysms (rECAAs).

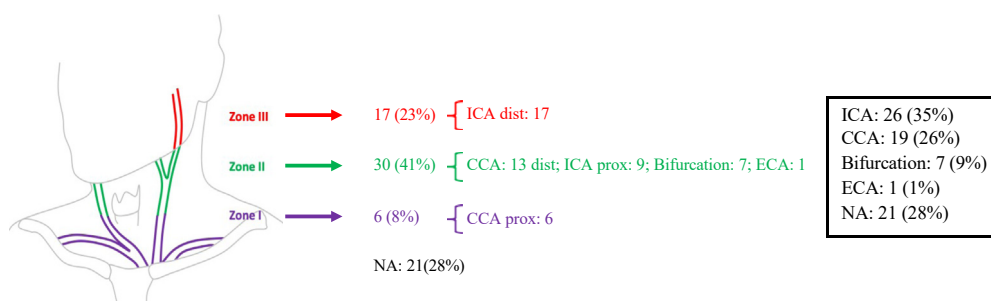


Fig 2. Sites of the ruptured extracranial carotid artery aneurysms (rECAAs) stratified by zones according to Núñez et al⁸ and location according to our comprehensive literature review. CCA, Common carotid artery; *dist*, distal; ECA, external carotid artery; ICA, internal carotid artery; NA, not available; *prox*, proximal.

had died of a palliatively treated ECAA that had ruptured. The final patient had died of severe hemorrhage.

CASE REPORT

Patient 1. The patient was 68-year-old woman with a medical history of chronic respiratory insufficiency, pulmonary embolism, and myocardial infarction. She had presented with pain and

bleeding from a peritonsillar ulceration caused by a 3.8-cm internal carotid artery (ICA) aneurysm located high in the right side of neck (Fig 3). EV treatment (EVT) was ruled out because of the tortuosity of the carotid artery, presumed difficulty in distal control, and local symptoms of the ECAA due to a mass effect. Thus, open surgery using a midline mandibulotomy and reconstruction of the ICA with an autologous (GSV) interposition

Table. Distribution of ruptured extracranial carotid artery aneurysms stratified by treatment

Treatment method	No. (%)	Technique	Etiology, No. (%)	Location (zone), No. (%)	Adverse outcome/status at follow-up, No. (%)
Reconstruction	24 (32)	Interposition graft: 16 ^a ; end-to-end anastomosis, 3; patch angioplasty, 2; ECA to ICA anastomosis, 1; brachiocephalic trunk to CCA bypass, 1; unspecified, 1	MYC, 9 (38); CTD, 5 (21); ASO, 4 (17); PT, 1 (4); NR, 5 (21)	II, 17 (71); III, 3 (13); I, 3 (13); NR, 1 (4)	Death, 3 (13): 2 ECAA-unrelated, 1 ECAA-related; CND, 3 (13) ^b ; blowout, 3 (13) ^c
EVT	10 (14)	Covered stent, 8 ^d ; coil embolization, 1; intravascular detachable balloon, 1	PT, 4 (40); CTD, 3 (30); ASO, 1 (10); MYC, 1 (10); NR, 1 (10)	III, 6 (60); II, 4 (40)	CND, 1 (10)
Ligation	17 (23)	NA	MYC, 6 (35); CTD, 5 (29); ASO, 2 (12); NR, 4 (24)	III, 7 (41); II, 6 (35); I, 2 (12); NR, 2 (12)	Stroke, 5 (29) ^e ; rebleeding, 1 (6) ^f ; persistent infection, 2 (12)
Conservative	2 (3)	NA	NR, 2 (100)	II, 1 (50); NR, 1 (50)	Death, 1 (50): ECAA-related, 1
No treatment	1 (1)	NA	MYC, 1 (100)	II, 1 (100)	Death, 1 (100): ECAA-related, 1
NR	20 (27) ^g	NA	NR, 16 (80); ASO, 2 (10); MYC, 2 (10)	I, 1 (5); II, 1 (5); III, 1 (5); NR, 17 (85)	Death, 1 (5): ECAA-unrelated, 1
Total	74 (100)	NA	MYC, 19 (26); CTD, 13 (18); ASO, 9 (12); PT, 5 (7); NR, 28 (38)	I, 6 (8); II, 30 (41); III, 17 (23); NR, 21 (28)	Death, 9 (12); stroke, 5 (7); CND, 4 (5); blowout, 3 (4); persistent infection, 2 (3); rebleeding, 1 (1)

ASO, Atherosclerosis obliterans; CCA, common carotid artery; CND, cranial nerve deficit; CTD, connective tissue disorder (Marfan syndrome, systemic lupus erythematosus, Behçet disease, Cogan syndrome, neurofibromatosis); ECA, external carotid artery; ECAA, extracranial carotid artery aneurysm; ICA, internal carotid artery; MYC, mycotic; NA, not applicable; NR, not reported; PT, post-traumatic.

^aTen great saphenous vein grafts, two polytetrafluoroethylene grafts, one polyethylene graft, one superficial femoral artery graft, and one bovine pericardium tube.

^bOne patient died of causes related to the ECAA.

^cOne patient died of causes unrelated to the ECAA.

^dOf the eight patients treated with stents, two were treated by a hybrid approach, and in two patients, the branching arteries were coiled.

^eThree strokes were fatal.

^fThe patient also had persistent infection.

^gThe cases of 19 patients had been reported in case series of unruptured ECAs and the case of 1 patient had been reported in a case report of ruptured ECAs.

graft was performed, as previously reported in a technical note.⁹ The patient had hypoglossal and glossopharyngeal nerve injuries and postoperative pharyngeal edema. On the 12th postoperative day, she was transferred to a local hospital. She died within 30 days postoperatively of aspiration pneumonia.

Case 2. The patient was 62-year-old man with hypertension, asthma, polymyalgia rheumatica, and chronic obstructive pulmonary disease. He had presented with swelling and pain in the left side of his neck of 5 months' duration and then abundant bleeding into the pharynx. At a local hospital, he underwent emergent tracheotomy. The computed tomography (CT) findings were suspicious for a pharyngeal tumor; however, biopsy findings were negative for neoplasm. CT angiography showed a 2.5-cm aneurysm or pseudoaneurysm at the left carotid bifurcation (Fig 4, A). An aneurysm–esophageal fistula was suspected, and the patient was admitted to the university hospital for surgery. Intraoperatively, the rupture site was thrombosed (Fig 4, B), and the aneurysm was resected with reconstruction from the common carotid artery (CCA) to ICA with a GSV interposition graft. The external carotid artery was also reconstructed with an additional GSV interposition

graft (Fig 4, C). After nasotracheal extubation on the operating table, the patient developed voluminous nasopharyngeal bleeding that was controlled by posterior nasal packing and cauterization. Esophagoscopy revealed a noncomplicated Zenker diverticulum. The patient had a good recovery without any neurologic deficits. The graft was patent on a CT scan at 1 month, and the patient was followed up for 1 year at his local hospital.

DISCUSSION

The most common etiology for an ECAA has remained controversial, and the risk of rupture with the different etiologies is not clear.^{1,2,10-12} Atherosclerosis has been previously reported as the most common etiology for unruptured ECAs.^{2,11,13-16} However, in the present study, the most common reported etiology for rECAs was infection (19 of 74; 26%). From the graphic presentation (Supplementary Fig, online only), the proportion of infectious etiologies for rECAs seems to have decreased during the time course. This could have been either a true decrease in the proportion of the infectious rECAs for various reasons or resulted from a relative increase in

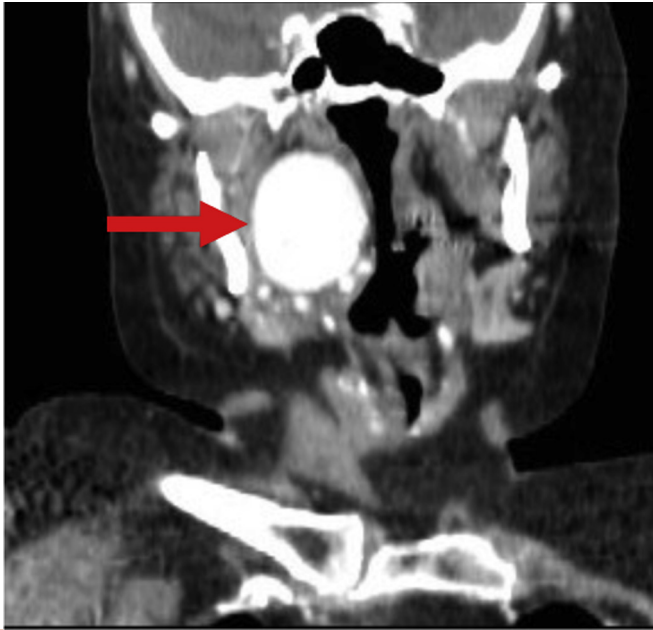


Fig 3. Computed tomography (CT) angiogram showing an aneurysm of the right internal carotid artery (ICA) located high in the neck (red arrow).

the number of rECAAs of other etiologies. The mean age per etiology suggested that for rECAAs, patients with atherosclerotic and infectious aneurysms will tend to be older compared with those with CTD and delayed post-traumatic lesions.

Both the bifurcation^{1,14,17} and the ICA^{2,11,12} have been suggested as the most common location for unruptured ECAAs. In the present review, the most common location for rECAAs was the ICA (35%), followed by the CCA (26%). In contrast, the bifurcation was the site for only 9% of the reported cases. This variation could have been affected by differences in the etiologies or differences in the descriptions provided by the various investigators of the anatomy in the unruptured and rECAAs.

For most unruptured ECAAs, open reconstructive surgery is the preferred treatment with satisfactory results.^{1,2,11,13} However, good results with nonoperative treatment have also been reported for patients with small, asymptomatic, and/or postdissection aneurysms or pseudoaneurysms.^{11,12,18,19}

For rECAAs, open surgery can be either ligation or reconstruction. In general, ligation has been reported to have a high incidence of ischemic stroke and/or death at 11% to 32% for unruptured aneurysms.^{1,20} From our literature review, the incidence of ischemic stroke and/or death was 29% for rECAAs, the incidence of rebleeding was 6%, and the incidence of persistent infection was 12%.

Because of the high incidence of complications associated with ligation, reconstruction seems to be a safer operative modality, although 26% had either experienced blowout (13%) or CND (13%) after reconstruction (Table). Regarding the use of prosthetic material in

reconstruction, we found only 3 reported cases (19%) compared with 10 (63%) that had used a GSV interposition. A recent study reported promising results using polytetrafluoroethylene in the treatment of unruptured ECAAs.²¹ However, the potential infectious etiology using the tissue samples from a rECAA often cannot be definitively ruled out at surgery. Thus, the possibility of an infectious etiology and the use of a biologic graft should be considered when treating rECAAs using open surgery.

Although the reported cases showed promising results with EVT, such treatment is also not free of complications, with 1 of the 10 patients developing CND after EVT.²² A similar incidence of complications related to unruptured ECAA treatment was reported by Welleweerd et al.¹¹ They reported that 12% had developed CND after surgery and none after EVT. In other studies, the rate of CND after surgical treatment was similar, with an incidence of $\leq 23\%$.^{10,13,17}

The location of the aneurysm has an influence on the type of treatment chosen. Aneurysms located in zone II can usually be easily treated using open surgery with reconstruction. However, aneurysms located in zone I (below the cricoid) will usually require sternotomy, and those in the zone III (above the angle of mandible) will require mandibulotomy or other special techniques.^{9,23} In the present review, the most common location for the reconstructive approach was zone II (71%; Table). In zone III, EVT with covered stents is a fair and safe option to treat rECAAs, whenever feasible.²⁴⁻³⁰ However, considering the kinks and curves often detected in the CCA, especially in zone I, the number of suitable EV grafts will be limited, and their performance could be suboptimal. To reduce the working length, the hybrid approach, in which the EV graft is placed through an open access on the neck, seems to be an alternative.²⁴ Hopefully, improving the stent technology and design will produce more options for stenting in the future.

The risk of publication bias in the present comprehensive literature review is obvious, because the occurrence of ECAAs is rare, and unsuccessful outcomes are less likely to be reported. Concurrently, the reports were very diverse owing to the absence of standardization. A meta-analysis was not feasible owing to the insufficient patient data, which were obtained from case series and case reports. Inevitably, the limited information provided in the reports resulted in a high amount of missing data, most significantly regarding the aneurysm size, and the short- and long-term follow-up data. However, we believe that even the limited data we have presented can increase our understanding of the disease and its treatment options and, thus, be a guide for the management of rECAAs, which at present have no treatment guidelines.

The natural history of ECAAs is also unclear because most asymptomatic aneurysms will not be reported and, if detected, will not be followed up, but repaired. Given the small number of patients with ECAA, a clinical trial is not

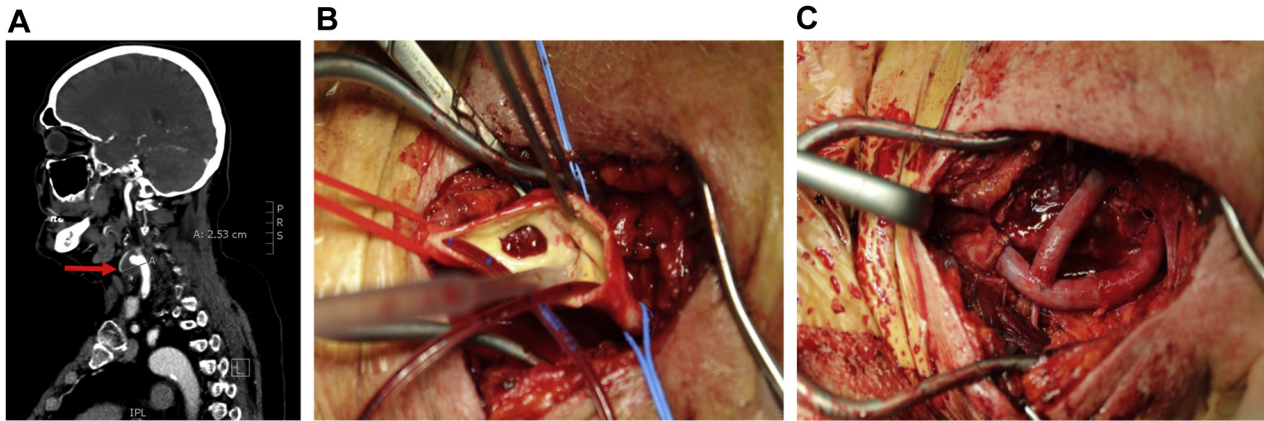


Fig 4. **A.** Computed tomography (CT) angiogram showing a 2.5-cm aneurysm at the bifurcation of the left carotid artery (red arrow). **B.** Intraoperative view of the aneurysm before aneurysmectomy. **C.** Reconstruction from the common carotid artery (CCA) to internal carotid artery (ICA) with a great saphenous vein (GSV) interposition graft. The external carotid artery was also reconstructed with an additional GSV interposition graft.

feasible. Therefore, a reasonable solution would be to collect all cases of carotid aneurysms into an existing international database with systematic and comparable data variables.⁵¹ The same type of patient registry has been successfully described for cystic fibrosis, providing annual reports and a better understanding of the disease.^{52,53}

CONCLUSIONS

Rupture of an ECAA is a rare and dangerous condition. The most common reported etiology for rECAAs was infection. Reconstructive surgery was the most common approach and a safer operative modality compared with ligation, which carried a high risk of stroke. EVT showed promising results, especially for distally located aneurysms; however, the number of patients has remained low.

AUTHOR CONTRIBUTIONS

Conception and design: NMdS, PV, RT, MV
 Analysis and interpretation: NMdS, PV, RT, MV
 Data collection: NMdS
 Writing the article: NMdS, PV, RT, MV
 Critical revision of the article: NMdS, PV, RT, MV
 Final approval of the article: NMdS, PV, RT, MV
 Statistical analysis: Not applicable
 Obtained funding: Not applicable
 Overall responsibility: NMdS

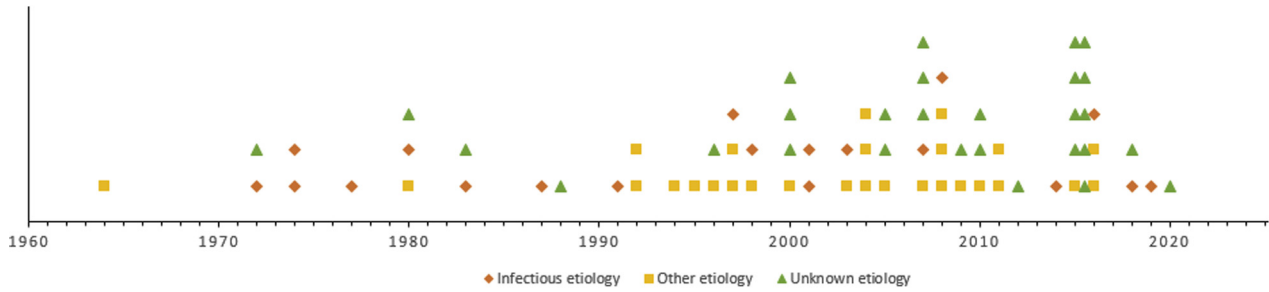
REFERENCES

1. El-Sabroun R, Cooley DA. Extracranial carotid artery aneurysms: Texas Heart Institute experience. *J Vasc Surg* 2000;31:702-12.
2. Radak D, Davidović L, Vukobratov V, Ilijevski N, Kostić D, Maksimović Z, et al. Carotid artery aneurysms: Serbian multicentric study. *Ann Vasc Surg* 2007;21:23-9.
3. Donas KP, Schulte S, Pitoulias GA, Siebertz S, Horsch S. Surgical outcome of degenerative versus postreconstructive extracranial aneurysms. *J Vasc Surg* 2009;49:93-8.
4. Rittenhouse EA, Radke HM, Sumner DS. Carotid artery aneurysm: review of the literature and report of a case with rupture into the oropharynx. *Arch Surg* 1972;105:786-9.
5. Pomara C, Bello S, Serinelli S, Fineschi V. A rare and lethal case of right common carotid pseudoaneurysm following whiplash trauma. *Forensic Sci Med Pathol* 2015;11:69-73.
6. Baranoski JF, Wanebo JE, Heiland KE, Zabramski JM. Superficial femoral artery interposition graft for repair of a ruptured mycotic common carotid artery pseudoaneurysm: case report and review of the literature. *World Neurosurg* 2019;129:130-2.
7. Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med* 2009;6:e1000097.
8. Núñez DB Jr, Torres-León M, Múnera F. Vascular injuries of the neck and thoracic inlet: helical CT-angiographic correlation. *Radiographics* 2004;24:1087-100.
9. Vikatmaa P, Mäkitie AA, Railo M, Törnwall J, Albäck A, Lepäntalo M. Midline mandibulotomy and interposition grafting for lesions involving the internal carotid artery below the skull base. *J Vasc Surg* 2009;49:86-92.
10. Faggioli GL, Freyrie A, Ila A, Pedrini L, Gargiulo M, Tarantini S, et al. Extracranial internal carotid artery aneurysms: results of a surgical series with long-term follow-up. *J Vasc Surg* 1996;23:587-95.
11. Welleweerd JC, den Ruijter HM, Nelissen BG, Bots ML, Kappelle LJ, Rinkel GJ, et al. Management of extracranial carotid artery aneurysm. *Eur J Vasc Endovasc Surg* 2015;50:141-7.
12. Fankhauser CT, Stone WM, Fowl RJ, O'Donnell ME, Bower TC, Meyer FB, et al. Surgical and medical management of extracranial carotid artery aneurysms. *J Vasc Surg* 2015;61:389-93.
13. Moreau P, Albat B, Thévenet A. Surgical treatment of extracranial internal carotid artery aneurysm. *Ann Vasc Surg* 1994;8:409-16.
14. Zhang Q, Duan ZQ, Xin SJ, Wang XW, Dong YT. Management of extracranial carotid artery aneurysms: 17 years' experience. *Eur J Vasc Endovasc Surg* 1999;18:162-5.
15. Zhou W, Lin PH, Bush RL, Peden E, Guerrero MA, Terramani T, et al. Carotid artery aneurysm: evolution of management over two decades. *J Vasc Surg* 2006;43:493-6.
16. Bush RL, Long P, Atkins MD. Carotid artery aneurysms. In: Sidawy AN, Perler BA, editors. *Rutherford's Vascular Surgery and Endovascular Therapy*. 9th ed. Philadelphia: Elsevier; 2019. p. 1242-54.
17. Zwolak RM, Whitehouse WM Jr, Knake JE, Bernfeld BD, Zelenock GB, Cronenwett JL, et al. Atherosclerotic extracranial carotid artery aneurysms. *J Vasc Surg* 1984;1:415-22.
18. Pourier VEC, Welleweerd JC, Kappelle LJ, Rinkel GJE, Ruigrok YM, van der Worp HB, et al. Experience of a single center in the conservative approach of 20 consecutive cases of asymptomatic extracranial carotid artery aneurysms. *Eur J Neurol* 2018;25:1285-9.

19. Paraskevas KI, Batchelder AJ, Naylor AR. Fate of distal false aneurysms complicating internal carotid artery dissection: a systematic review. *Eur J Vasc Endovasc Surg* 2016;52:281-6.
20. Brackett CE Jr. The complications of carotid artery ligation in the neck. *J Neurosurg* 1953;10:91-106.
21. Illuminati G, Pizzardi G, Pasqua R, Nardi P, Calio' FG, Ricco JB. Long-term results of polytetrafluoroethylene versus saphenous vein repair of degenerative carotid artery aneurysm. *J Vasc Surg* 2020;72:1413-20.
22. Smith BL, Munschauer CE, Diamond N, Rivera F. Ruptured internal carotid aneurysm resulting from neurofibromatosis: treatment with intraluminal stent graft. *J Vasc Surg* 2000;32:824-8.
23. Malikov S, Thomassin JM, Magnan PE, Keshelava G, Bartoli M, Branchereau A. Open surgical reconstruction of the internal carotid artery aneurysm at the base of the skull. *J Vasc Surg* 2010;51:323-9.
24. Kubaska SM III, Greenberg RK, Clair D, Barber C, Srivastava SD, Green RM, et al. Internal carotid artery pseudoaneurysms: treatment with the Wallgraft endoprosthesis. *J Endovasc Ther* 2003;10:182-9.
25. Siablis D, Karnabatidis D, Katsanos K, Mastronikolis N, Zabakis P, Kraniotis P. Extracranial internal carotid artery aneurysms: report of a ruptured case and review of the literature. *Cardiovasc Intervent Radiol* 2004;27:397-401.
26. Akiyama Y, Nakahara I, Tanaka M, Iwamuro Y, Hayashi J, Harada K, et al. Urgent endovascular stent-graft placement for a ruptured traumatic pseudoaneurysm of the extracranial carotid artery. *J Trauma* 2005;58:624-7.
27. Ohshima T, Miyachi S, Hattori K, Iizuka H, Izumi T, Nakane Y, et al. A case of giant common carotid artery aneurysm associated with vascular Behçet disease: successfully treated with a covered stent. *Surg Neurol* 2008;69:297-301.
28. Yeh CH, Lin MS, Chiu MC, Chen CH, Pai YL. Endovascular treatment of a huge cervical carotid artery pseudoaneurysm with Wallgraft prosthesis. *Ann Vasc Surg* 2011;25:265.e1-3.
29. Mazzaccaro D, Stegher S, Occhiuto MT, Malacrida G, Righini P, Tealdi DG, et al. Hybrid endovascular and surgical approach for mycotic pseudoaneurysms of the extracranial internal carotid artery. *SAGE Open Med Case Rep* 2014;2.
30. Katsevman GA, Braca JA III, Welch KC, Ashley WW Jr. Delayed presentation of an extracranial internal carotid artery pseudoaneurysm and massive epistaxis secondary to a nasal foreign body: case report and review of the literature. *World Neurosurg* 2016;92:585.e13-9.
31. Welleweerd JC, De Borst GJ. Extracranial carotid artery aneurysm: optimal treatment approach. *Eur J Vasc Endovasc Surg* 2015;49:235-6.
32. European Cystic Fibrosis Society. ECFS Patient Registry. Available at: <https://www.ecfs.eu/projects/ecfs-patient-registry/project>. Accessed September 29, 2019.
33. Cystic Fibrosis Foundation. Patient Registry. Available at: <https://www.cff.org/Research/Researcher-Resources/Patient-Registry>. Accessed September 29, 2019.

Submitted Nov 29, 2020; accepted Jun 13, 2021.

Additional material for this article may be found online at www.jvascsurg.org.



Supplementary Fig (online only). Etiologies of ruptured extracranial carotid artery aneurysms (rECAAs) stratified by year of publication.

Supplementary Table (online only). Details of ruptured extracranial artery aneurysms from our comprehensive literature review

Investigator	Country	rECAA, No.	Sex	Age, years	Maximal diameter, cm	Location	Zone	Etiology	Treatment	Adverse outcome (<30 days, related)	Follow-up, months	Status at follow-up
Razdan et al, ¹ 1964	India	1	M	25	5	CCA distal	2	PT	Arterial cadaveric graft interposition	No	8.5	Graft working and hoarseness improved considerably; hoarseness present at admission
Margolis et al, ² 1972	USA	1	M	20	6	ICA distal	3	MYC	Ligation	No	NR	NR
Rittenhouse et al, ³ 1972	USA	1	M	74	1.5	Bifurcation	2	NR	Modified bovine heterograft interposition	No neurologic deficit	>1	Patent (ultrasound arteriogram showed patency of graft 32 days postoperatively); shortly thereafter, patient died (unrelated) and had blowout
Ledgerwood et al, ⁴ 1974	USA	2	M	52	10	CCA proximal	1	MYC (<i>S. aureus</i> MRSA)	Ligation (sternotomy)	No neurologic deficit; persistent infection (remained febrile) and recurrent profuse bleeding from puncture; ligation of distal CCA, subclavian; repeat ligation of innominate and collaterals and necrosis evacuated from abscess (MRSA); postoperative course was uneventful; discharge 4 weeks later, with irrigation continued at home	NR	2 Subsequent admissions required for drainage of suprasternal abscess; returned to previous job; cured of heroin addiction
			M	49	8	CCA proximal	1	MYC (<i>Pseudo monas</i>)	Ligation (sternotomy)	Persistent infection (remained febrile); no neurologic deficit; no hemoptysis; 1 week later, CCA was ligated distal to mass; daily irrigations were performed, and the wound healed; patient progressed satisfactorily and was discharged 4 weeks after admission with no neurologic deficits	NR	NR
Howell et al, ⁵ 1977	USA	1	M	62	10	CCA	2	MYC (<i>Klebsiella</i>)	Ligation	No neurologic deficits; wound infection and drainage from incisional drain site continued for 4 weeks	1.3	-5 Weeks postoperatively was discharged from hospital; wound had healed completely
Busuttill et al, ⁶ 1980	USA	1	NR	NR	NR	NR	NR	NR	Ligation	Stroke	NR	NR
Pratschke et al, ⁷ 1980	Germany	2	NR	NR	NR	NR	NR	MYC	NR	NR	NR	NR
			NR	NR	NR	NR	NR	ASO	NR	NR	NR	NR
Welling et al, ⁸ 1983	USA	2	NR	NR	NR	ICA proximal	2	NR	NR	NR	NR	NR
			NR	NR	NR	NR	NR	MYC (<i>S. aureus</i>)	GSV interposition	NR	NR	NR

(Continued on next page)

Supplementary Table (online only). Continued.

Investigator	Country	rECAA No.	Sex	Age, years	Maximal diameter, cm	Location	Zone	Etiology	Treatment	Adverse outcome (<30 days, related)	Follow-up, months	Status at follow-up
Grossi et al, ⁹ 1987	USA	1	F	68	6	ICA proximal	2	MYC (<i>Salmonella enteritidis</i>)	End-to-end	14 Days postoperatively: patent anastomosis	8	Free from problems related to aneurysm procedure
Nicholson et al, ¹⁰ 1988	UK	1	F	72	2.8	ICA proximal	2	NR	GSV interposition	No	6	Patent; hoarseness had resolved and hypoglossal nerve palsy had improved; hoarseness and hypoglossal nerve palsy present at admission
Petrovic et al, ¹¹ 1991	Yugoslavia	1	M	67	NR	CCA prox	1	MYC (tuberculosis)	NR (Emergency surgical procedure + sternotomy)	Persistent stroke (regressive hemiparesis) and persistent CND (Horner). Ps: stroke and Horner syndrome are symptoms at admission	3	Dead, unrelated (no signs of wound infection)
Crow et al, ¹² 1992	USA	1	M	26	3	ICA distal	3	PT	Intravascular detachable balloon	No evidence of cerebral ischemia (ARDS unrelated)	1	ARDS resolved; thrombosis of ICA and aneurysm; no neurologic events; no bleeding
Kubo et al, ¹³ 1992	Japan	1	F	56	6	ICA distal	3	CTD	End-to-end	New CND; hoarseness and Horner syndrome at admission, persistent; complications unrelated (>5 days: hemorrhagic shock due to hepatic ruptured aneurysm, surgery, patent ICA, septic shock; >11 days: surgery intracranial aneurysm; 1.5 months after operation, patient had rib fractures)	NR	NR (>3 months: incisional abdominal hernia, unrelated)
Liapis et al, ¹⁴ 1994	Greece	1	F	NR	NR	CCA	2	ASO	End-to-end	No neurologic deficit	36	Dead, unrelated
Faggioli et al, ¹⁵ 1996; Stella et al, ¹⁶ 1995	Italy	1	F	83	4.5	ICA distal	3	ASO	ICA/ECA transposition	No	24	Patent
Karov, ¹⁷ 1996	Bulgaria	1	F	65	NR	ICA distal	3	NR	NR	NR	NR	NR
Neelakandhan et al, ¹⁸ 1996	India	1	NR	NR	NR	NR	NR	ASO	Ligation	NR (no death)	NR	NR
Coffin et al, ¹⁹ 1997	France	1	NR	NR	NR	ICA distal	3	CTD	Ligation	NR (no death)	NR	NR
Rice et al, ²⁰ 1997	USA	1	M	70	0.8	ICA proximal	2	MYC (<i>Salmonella</i> group D)	Patch angioplasty (GSV)	No	12	Symptom free
Tüzün et al, ²¹ 1997	Turkey	1	M	25	NR	CCA	2	CTD	Ligation	NR	1	Alive
Buerger et al, ²² 1998	Germany	1	F	55	NR	Bifurcation	2	MYC (<i>S. aureus</i>)	GSV interposition	No new neurologic deficit (persistent hoarseness since admission); 3 days postoperatively patent; 12 days postoperatively carotid blowout, GSV interposition; no further complications and ATB for 3 months	NR	NR

Supplementary Table (online only). Continued.

Investigator	Country	rECAAs No.	Sex	Age, years	Maximal diameter, cm	Location	Zone	Etiology	Treatment	Adverse outcome (<30 days, related)	Follow-up, months	Status at follow-up
Sasaki et al, ²³ 1998	Japan	1	M	16	NR	CCA	2	CTD	Patch angioplasty (PTFE)	NR	36	Blowout at 2 months; CCA ligation; no neurologic deficit; alive at 3 years
Nair et al, ²⁴ 2000	South Africa	3	NR	NR	NR	NR	NR	NR	NR	NR (no death or new CND)	NR	NR
			NR	NR	NR	NR	NR	NR	NR	NR (no death or new CND)	NR	NR
			NR	NR	NR	NR	NR	NR	NR	NR (no death or new CND)	NR	NR
Smith et al, ²⁵ 2000	USA	1	F	28	NR	ICA dist	3	CTD	EV, covered stent + coil	CND	18	Patent, persistent hypoglossal nerve disorder, resolution of glosso pharyngeal injury, no other lesions
Machens et al, ²⁶ 2001	Germany	2	F	60	NR	CCA	2	MYC (<i>S. aureus</i> MSSA)	No	Death from aneurysm rupture	NR	Dead
			M	52	0.5	CCA proximal	1	MYC (<i>S. aureus</i> MRSA)	GSV interposition	NR	5	Osteomyelitis ensued 5 months after cervical reoperation; osteomyelitis subsided on ATB; no current evidence of infection found
Angle et al, ²⁷ 2003	USA	1	M	88	3	ICA proximal	2	MYC (<i>Proteus mirabilis</i>)	GSV interposition	No neurologic deficit	NR	NR
Kubaska et al, ²⁸ 2003	USA	1	F	34	NR	ICA distal	3	CTD	Hybrid, covered stent (Wallgraft)	NR	14	Patent
Davidovic et al, ²⁹ 2004; Davidovic et al, ³⁰ 2016	Serbia	2	NR	NR	NR	ICA distal	3	ASO	Ligation	Stroke and death	NR	Dead
			M	NR	NR	ICA proximal	2	ASO	Reconstruction, unspecified	NR	NR	NR
Siablis et al, ³¹ 2004	Greece	1	M	68	4.1	ICA proximal	2	ASO	EV, covered stent	No (postinterventional angiography: patent)	12	1 Month: no flow in aneurysmal cavity; 1 year: symptom free
Akiyama et al, ³² 2005	Japan	1	M	48	3	CCA	2	PT	EV, covered stent	No	6	Patent
Aleksic et al, ³³ 2005	Germany	1	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Masuda et al, ³⁴ 2005	Japan	1	F	46	3	CCA	2	NR, pseudo aneurysm	Conservative	NR	1	Neck swelling and laryngeal edema since admission had resolved
Low et al, ³⁵ 2007	Singapore	1	M	17	NR	CCA	2	CTD	GSV interposition	No	24	Complications unrelated after 4 months; 24 months, no evidence on serial MR angiography of recurrent aneurysms or progressive vascular stenosis

(Continued on next page)

Supplementary Table (online only). Continued.

Investigator	Country	rECAA No.	Sex	Age, years	Maximal diameter, cm	Location	Zone	Etiology	Treatment	Adverse outcome (<30 days, related)	Follow-up, months	Status at follow-up		
Radak et al, ³⁶ 2007	Serbia	4	NR	NR	NR	ICA distal	3	NR ^a	Ligation	Stroke and death	NR	Dead		
			NR	NR	NR	ICA distal	3	NR ^a	Ligation	Stroke and death	NR	Dead		
			NR	NR	NR	ICA distal	3	NR ^a	Ligation	Stroke	NR	NR		
			NR	NR	NR	NR	NR	NR	NR	NR	NR	NR		
Ku et al, ³⁷ 2008	Taiwan	1	F	28	5	ICA proximal	2	CTD	Ligation	None (other than symptoms at admission)	NR	NR		
Ohshima et al, ³⁸ 2008	Japan	1	M	56	6.5	CCA	2	CTD	EV, covered stent + coil	Cervical mass had disappeared; residual hematoma sac aspirated after 3 weeks; symptoms dramatically improved	12	Patent		
Shrestha, ³⁹ 2008	Nepal	1	M	54	10	Bifurcation	2	ASO	Polyethylene interposition (Dacron)	CND (facial nerve)	3	Improvement of Horner syndrome (present at admission); persistent facial nerve injury		
Song et al, ⁴⁰ 2008	China	1	M	55	3.6	Bifurcation	2	MYC (<i>Streptococcus</i>)	GSV interposition	No	48	Symptom free		
Donas et al, ⁴¹ 2009	Germany	1	NR	NR	NR	NR	NR	ASO	NR	NR	NR	NR		
Pinjala, ⁴² 2010	India	1	M	25	NR	CCA	2	NR	Ligation	No	NR	NR		
Reis et al, ⁴³ 2010	Brazil	1	NR	15	NR	ICA distal	3	NR, pseudo aneurysm	EV, coil	No	3	Occlusion of artery; no blood flow to rupture, clinical improvement present at discharge maintained		
Sayed et al, ⁴⁴ 2010	Egypt	1	M	32	NR	ICA distal	3	CTD	Ligation	NR (no death or stroke)	NR	NR		
O'Brien et al, ⁴⁵ 2011	USA	1	M	40	4.7	ICA proximal	2	CTD	GSV interposition	No neurologic deficit	4	Persistent vocal cord paralysis (present at admission)		
Yeh et al, ⁴⁶ 2011	China	1	M	38	6.3	ICA distal	3	PT	EV, covered stent	Size of aneurysm reduced	24	30 Days postoperatively: passage of airway in nasopharynx improved; 24 months postoperatively: no aneurysm		
Garg et al, ⁴⁷ 2012	USA	1	NR	NR	NR	CCA proximal	1	NR	Bypass from BCT to CCA (sternotomy)	NR (no death or stroke)	NR	NR (no death or stroke)		
Mazzaccaro et al, ⁴⁸ 2014	Italy	1	M	81	4	Bifurcation	2	MYC (<i>S. aureus</i> MRSA)	Hybrid, covered stent (PTFE nitinol) + PTFE graft to reinforce carotid wall	No	7	Patent and no wound infection		
Fankhauser et al, ⁴⁹ 2015	USA	9	NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR	NR ^b	
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR	NR ^b	
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR	NR ^b	
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR	NR	NR ^b
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR	NR	NR ^b

Supplementary Table (online only). Continued.

Investigator	Country	rECAA, No.	Sex	Age, years	Maximal diameter, cm	Location	Zone	Etiology	Treatment	Adverse outcome (<30 days, related)	Follow-up, months	Status at follow-up
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR ^b
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR ^b
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR ^b
			NR	NR	NR	NR	NR	NR, pseudo aneurysm	NR	NR	NR	NR ^b
Lyazidi et al, ⁵⁰ 2015	Morocco	1	M	20	9	CCA proximal	1	CTD	PTFE interposition (sternotomy)	No	1	Patent
Katsevman et al, ⁵¹ 2016	USA	1	M	58	1	ICA distal	3	PT	EV, covered stent	No further episodes of epistaxis	NR	NR (patient did not return)
Rogers et al, ⁵² 2016	Ireland	1	M	61	2.4	Bifurcation	2	MYC (coagulase-negative <i>Staphylococcus</i>)	Ligation	NR	3	CTA showed no signs of infection; resolution of osteomyelitis
Souldi et al, ⁵³ 2016	Morocco	1	F	32	NR	ECA proximal	2	CTD	Ligation	No	12	No vascular complications
Kim et al, ⁵⁴ 2018	Korea	1	M	89	4.8	CCA	2	MYC (<i>S. aureus</i>)	PTFE interposition	No	3	No wound infection
Kumar et al, ⁵⁵ 2018	India	1	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Baranoski et al, ⁵⁶ 2019	USA	1	M	51	1	CCA distal	2	MYC	SFA interposition	No new deficits	6	Patent, stable, free of recurrent disease
Vikatmaa et al, ⁵⁷ 2009 (present case 1)	Finland	1	F	68	4	ICA distal	3	NR	GSV interposition (mandibulotomy)	CND and death	NR	Dead
Present case 2, 2020	Finland	1	M	62	2.5	Bifurcation	2	NR	GSV interposition	Bleeding through mouth during surgery; resolved after nasal packing and cauterization	1	Patent

ARDS, acute respiratory distress syndrome; ASO, atherosclerosis obliterans; ATB, antibiotic; BCT, brachiocephalic trunk; CCA, common carotid artery; CND, cranial nerve deficit; CTA, computed tomography angiography; CTD, connective tissue disorder (Marfan syndrome, systemic lupus erythematosus, Behçet disease, Cogan syndrome, neurofibromatosis); ECA, external carotid artery; EV, endovascular; F, female; GSV, great saphenous vein; ICA, internal carotid artery; M, male; MR, magnetic resonance; MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-sensitive *Staphylococcus aureus*; MYC, mycotic; NR, not reported; PT, post-traumatic; PTFE, polytetrafluoroethylene; rECAA, ruptured extracranial carotid aneurysm; *S. aureus*, *Staphylococcus aureus*; SFA, superficial femoral artery.

^aOne patient had a mycotic aneurysm caused by tuberculous lymphadenitis of the neck.

^bOne patient died of an aneurysm treated palliatively that ruptured.

SUPPLEMENTARY REFERENCES

- Razdan AN, Sharma BD, Kak VK. Traumatic Aneurysm of the common carotid artery treated by excision and arterial homograft. *J Int Coll Surg* 1964;42:126-33.
- Margolis MT, Stein RL, Newton TH. Extracranial aneurysms of the internal carotid artery. *Neuroradiology* 1972;4:78-89.
- Rittenhouse EA, Radke HM, Sumner DS. Carotid artery aneurysm: review of the literature and report of a case with rupture into the oropharynx. *Arch Surg* 1972;105:786-9.
- Ledgerwood AM, Lucas CE. Mycotic aneurysm of the carotid artery. *Arch Surg* 1974;109:496-8.
- Howell HS, Baburao T, Graziano J. Mycotic cervical carotid aneurysm. *Surgery* 1977;81:357-9.
- Busuttill RW, Davidson RK, Foley KT, Livesay JT, Barker WF. Selective management of extracranial carotid arterial aneurysms. *Am J Surg* 1980;140:85-91.
- Pratschke E, Schäfer K, Reimer J, Stiegler H, Stelter WJ, Becker HM. Extracranial aneurysms of the carotid artery. *Thorac Cardiovasc Surg* 1980;28:354-8.
- Welling RE, Taha A, Goel T, Cranley J, Krause R, Hafner C, et al. Extracranial carotid artery aneurysms. *Surgery* 1983;93:319-23.
- Grossi RJ, Onofrey D, Tvetenstrand C, Blumenthal J. Mycotic carotid aneurysm. *J Vasc Surg* 1987;6:81-3.
- Nicholson ML, Horrocks M. Leaking carotid artery aneurysm. *Eur J Vasc Surg* 1988;2:197-8.
- Petrovic P, Avramov S, Pfau J, Fabri M, Obradovic J, Vukobratov V. Surgical management of extracranial carotid artery aneurysms. *Ann Vasc Surg* 1991;5:506-9.
- Crow WN, Scott BA, Guinto FC Jr, Chaljub G, Wright G, Rabassa AE, et al. Massive epistaxis due to pseudoaneurysm: treated with detachable balloons. *Arch Otolaryngol Head Neck Surg* 1992;118:321-4.
- Kubo S, Nakagawa H, Imaoka S. Systemic multiple aneurysms of the extracranial internal carotid artery, intracranial vertebral artery, and visceral arteries: case report. *Neurosurgery* 1992;30:600-2.
- Liapis CD, Gugulakis A, Misiakos E, Verikokos C, Dousaitou B, Sechas M. Surgical treatment of extracranial carotid aneurysms. *Int Angiol* 1994;13:290-5.
- Faggioli GL, Freyrie A, Ila A, Pedrini L, Gargiulo M, Tarantini S, et al. Extracranial internal carotid artery aneurysms: results of a surgical series with long-term follow-up. *J Vasc Surg* 1996;23:587-95.
- Stella A, Tarantini S, Faggioli GL, Gargiulo M, Pilato A. Rupture of a non-specific aneurysm of the extracranial internal carotid artery. *G Ital Chir Vasc* 1995;2:157-65.
- Karov I. Spontaneous rupture of an internal carotid artery aneurysm diagnosed as a peritonsillar abscess, a tonsillar and epipharyngeal carcinoma with metastasis. *Folia Med (Plovdiv)* 1996;38:65-7.

18. Neelakandhan KS, Matta R. Extracranial carotid artery aneurysms. *Int Angiol* 1996;15:316-20.
19. Coffin O, Maïza D, Galateau-Sallé F, Martel B, Vignon C, Neri E, et al. Results of surgical management of internal carotid artery aneurysm by the cervical approach. *Ann Vasc Surg* 1997;11:482-90.
20. Rice HE, Arbabi S, Kremer R, Needle D, Johansen K. Ruptured *Salmonella* mycotic aneurysm of the extracranial carotid artery. *Ann Vasc Surg* 1997;11:416-9.
21. Tüzün H, Beşirli K, Sayin A, Vural FS, Hamuryudan V, Hizli N, et al. Management of aneurysms in Behçet's syndrome: an analysis of 24 patients. *Surgery* 1997;121:150-6.
22. Buerger T, Meyer F, Halloul Z. Ruptured cervical aneurysm of the carotid artery—case report of a rare disease. *Vasa* 1998;27:122-4.
23. Sasaki SH, Yasuda K, Takigami K, Shiya N, Matsui Y, Sakuma M. Surgical experiences with peripheral arterial aneurysms due to vasculo-Behçet's disease. *J Cardiovasc Surg (Torino)* 1998;39:147-50.
24. Nair R, Robbs JV, Naidoo NG. Spontaneous carotid artery aneurysms. *Br J Surg* 2000;87:186-90.
25. Smith BL, Munschauer CE, Diamond N, Rivera F. Ruptured internal carotid aneurysm resulting from neurofibromatosis: treatment with intraluminal stent graft. *J Vasc Surg* 2000;32:824-8.
26. Machens A, Dralle H. Mycotic aneurysm of common carotid artery induced by *Staphylococcus aureus* infection after cervical reoperation. *World J Surg* 2001;25:1113-6.
27. Angle N, Dorafshar AH, Ahn SS. Mycotic aneurysm of the internal carotid artery: a case report. *Vasc Endovasc Surg* 2003;37:213-7.
28. Kubaska SM III, Greenberg RK, Clair D, Barber G, Srivastava SD, Green RM, et al. Internal carotid artery pseudoaneurysms: treatment with the Wallgraft endoprosthesis. *J Endovasc Ther* 2003;10:182-9.
29. Davidovic L, Kostic D, Maksimovic Z, Markovic D, Vasic D, Markovic M, et al. Carotid artery aneurysms. *Vascular* 2004;12:166-70.
30. Davidovic L, Koncar I, Dragas M, Ilic N, Banzic I, Pavlovic SU, et al. Treatment strategies for carotid artery aneurysms. *J Cardiovasc Surg (Torino)* 2016;57:872-80.
31. Siablis D, Karnabatidis D, Katsanos K, Mastronikolis N, Zabakis P, Kraniotis P. Extracranial internal carotid artery aneurysms: report of a ruptured case and review of the literature. *Cardiovasc Intervent Radiol* 2004;27:397-401.
32. Akiyama Y, Nakahara I, Tanaka M, Iwamura Y, Hayashi J, Harada K, et al. Urgent endovascular stent-graft placement for a ruptured traumatic pseudoaneurysm of the extracranial carotid artery. *J Trauma* 2005;58:624-7.
33. Aleksic M, Heckenkamp J, Gawenda M, Brunkwall J. Differentiated treatment of aneurysms of the extracranial carotid artery. *J Cardiovasc Surg (Torino)* 2005;46:19-23.
34. Masuda K, Ozawa H, Fujii M, Kimura S, Nagasaki K, Matsunaga T, et al. Critical spontaneous rupture of a common carotid artery pseudoaneurysm. *Lancet* 2005;366:2034.
35. Low AH, Su JW, Sin KY, Fong KY, Thumboo J. Cogan's syndrome with recurrent carotid and aortic aneurysms: a potentially fatal disorder mimicking Marfan syndrome. *Scand J Rheumatol* 2007;36:71-3.
36. Radak D, Davidović L, Vukobratov V, Ilijevski N, Kostić D, Maksimović Z, et al. Carotid artery aneurysms: Serbian multicentric study. *Ann Vasc Surg* 2007;21:23-9.
37. Ku YK, Chen HW, Chen HW, Fu CJ, Chin SC, Liu YC. Giant extracranial aneurysms of both internal carotid arteries with aberrant jugular veins in a patient with neurofibromatosis type 1. *AJNR Am J Neuroradiol* 2008;29:1750-2.
38. Ohshima T, Miyachi S, Hattori K, Iizuka H, Izumi T, Nakane Y, et al. A case of giant common carotid artery aneurysm associated with vascular Behçet disease: successfully treated with a covered stent. *Surg Neurol* 2008;69:297-301.
39. Shrestha UK. Ruptured aneurysm of extracranial carotid artery. *JNMA J Nepal Med Assoc* 2008;47:232-4.
40. Song JQ, Zhang J, Yin MD, Shan SY, Wu B, Duan ZQ, et al. Vascular reconstruction of a ruptured and infected aneurysm of extracranial carotid artery. *Chin Med J (Engl)* 2008;121:671-2.
41. Donas KP, Schulte S, Pitoulias GA, Siebertz S, Horsch S. Surgical outcome of degenerative versus postreconstructive extracranial aneurysms. *J Vasc Surg* 2009;49:93-8.
42. Pinjala RK. Ruptured large carotid artery aneurysm. *Indian J Surg* 2010;72(Suppl 1):321-2.
43. Reis F, Reis LF, Ferraz Filho JR, de Godoy JM. Involvement of the oral cavity in a carotid pseudoaneurysm of an adolescent treated by an endovascular procedure. *Acta Otolaryngol* 2010;130:190-2.
44. Sayed A, Elwan H, Fouad F, Taha A, Elhindawi K, Khairy H, et al. Behçet extracranial carotid aneurysms: is there still a role for ligation? *Eur J Vasc Endovasc Surg* 2010;39:17-22.
45. O'Brien PJ, Peterson D, Cox MW. Spontaneous rupture of a carotid artery aneurysm. *Vasc Endovasc Surg* 2011;45:202-4.
46. Yeh CH, Lin MS, Chiu MC, Chen CH, Pai YL. Endovascular treatment of a huge cervical carotid artery pseudoaneurysm with Wallgraft prosthesis. *Ann Vasc Surg* 2011;25:265.e1-3.
47. Garg K, Rockman CB, Lee V, Maldonado TS, Jacobowitz GR, Adelman MA, et al. Presentation and management of carotid artery aneurysms and pseudoaneurysms. *J Vasc Surg* 2012;55:1618-22.
48. Mazzaccaro D, Stegher S, Occhiuto MT, Malacrida G, Righini P, Tealdi DG, et al. Hybrid endovascular and surgical approach for mycotic pseudoaneurysms of the extracranial internal carotid artery. *SAGE Open Med Case Rep* 2014;2: 2050313X14558081.
49. Fankhauser GT, Stone WM, Fowl RJ, O'Donnell ME, Bower TC, Meyer FB, et al. Surgical and medical management of extracranial carotid artery aneurysms. *J Vasc Surg* 2015;61:389-93.
50. Lyazidi Y, Abissegue GY, Chtata HT, Taberkant M. Ruptured carotid aneurysm revealing a Behçet's disease. *Ann Vasc Surg* 2015;29:1317.e1-4.
51. Katsevman GA, Braca JA III, Welch KC, Ashley WW Jr. Delayed presentation of an extracranial internal carotid artery pseudoaneurysm and massive epistaxis secondary to a nasal foreign body: case report and review of the literature. *World Neurosurg* 2016;92:585.e13-9.
52. Rogers AC, Bourke M, Galbraith AS, Ryan AG, Cross KS, McMonagle MP. Mycotic aneurysm of the extracranial internal carotid artery, resect and ligate or reconstruct? *Ann Vasc Surg* 2016;35:203.e5-10.
53. Souldi H, Bajja MY, Chenguir M, Abada R, Rouadi S, Roubal M, et al. Ruptured left external carotid artery aneurysm presenting as upper airway obstruction in von Recklinghausen's disease. *Int J Surg Case Rep* 2016;26:170-2.
54. Kim DI, Park BS, Kim DH, Kim DJ, Jung YJ, Lee SS, et al. Ruptured mycotic aneurysm of the common carotid artery: a case report. *Vasc Specialist Int* 2018;34:48-50.
55. Kumar A, Prabhakar A, Gupta V, Khandelwal N, Ahuja CK, Singhal M, et al. Endovascular management of internal carotid artery pseudoaneurysms: a single-centre experience of 20 patients. *Neurol India* 2018;66:1067-74.
56. Baranoski JF, Wanebo JE, Heiland KE, Zabramski JM. Superficial femoral artery interposition graft for repair of a ruptured mycotic common carotid artery pseudoaneurysm: case report and review of the literature. *World Neurosurg* 2019;129:130-2.
57. Vikatmaa P, Mäkitie AA, Railo M, Törnwall J, Albäck A, Lepäntalo M. Midline mandibulotomy and interposition grafting for lesions involving the internal carotid artery below the skull base. *J Vasc Surg* 2009;49:86-92.