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Review Article

Extracranial carotid aneurysms bridging gaps in diagnosis and treatment

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Abstract

Extracranial carotid artery aneurysms (ECAA) are uncommon and frequently overlooked conditions. This review seeks to encapsulate the prevailing insights on ECAA, focusing on its causes, clinical manifestations, diagnostic difficulties, and treatment alternatives. An extensive literature review was conducted, concentrating on studies that delve into the etiology, diagnostic processes, and management strategies for extracranial carotid aneurysms. Relevant articles published from 2000 to 2024 were sourced from databases including PubMed and Web of Science. ECAA is frequently asymptomatic and diagnosed incidentally, with pulsatile neck masses and cerebral ischemic events being the most common clinical presentations. Various etiologies, including atherosclerosis, infection, and trauma, contribute to the development of ECAA. Treatment strategies vary, ranging from conservative medical management to surgical and endovascular interventions, with no established guidelines (Central Illustration). ECAA remains a rare but significant clinical challenge due to the lack of consensus on its management. Further research is necessary to establish treatment guidelines and improve patient outcomes. Both surgical and endovascular options show promising results, but long-term data are needed.

Keywords: Extracranial carotid aneurysms, diagnosis, treatment, surgical management

INTRODUCTION

The extracranial carotid artery aneurysm (ECAA) is an infrequent peripheral arterial disorder with diverse symptoms and etiological factors. ECAA predominantly affects men, most commonly observed in individuals over 50 years of age. The most frequently noted finding is swelling in the neck. Primary treatment options encompass conservative management, open surgical procedures, endovascular approaches, and hybrid methods. Currently, treatment relies solely on expert opinion, supported by a limited number of case reports. As a result, there is limited information regarding the emergency and long-term outcomes of surgical treatment.

Aneurysms are an uncommon pathological condition of the carotid arteries. Despite the carotid artery's susceptibility to various diseases like atherosclerosis and aneurysm development, the internal carotid artery (ICA) segment is predominantly affected, showing an incidence of approximately 32% [1]. Aneurysmal disease accounts for an estimated 0.1% to 2% of carotid procedures, representing only 0.4% to 4% of all peripheral arterial aneurysms. [2-6]. Attigah et al. categorized carotid aneurysms into five anatomical types: Type I, isolated short aneurysms of the ICA located above the carotid bulb; Type II, long ICA aneurysms that extend from the carotid bulb to the Blaisdell line; Type III, aneurysms affecting the proximal ICA and carotid bifurcation; Type IV, aneurysms involving both the common carotid artery (CCA) and ICA, extending proximally and distally like Type III; and Type V, isolated aneurysms of the CCA [7].

The etiologies of ECAAs are diverse, including factors such as atherosclerosis, infections, fibromuscular dysplasia, connective tissue disorders linked to traumatic or spontaneous dissection,

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and pseudoaneurysms resulting from prior surgical repairs. [1]. In many cases, diagnosis may be incidental or made during evaluation for other pathologies. A pulsatile mass in the neck and mass effect are among the clinical presentations of ECAA [8].

Treatment options for ECAA encompass open surgery, endovascular procedures, hybrid techniques, and conservative management involving antiplatelet and anticoagulant agents; however, no formal treatment guidelines or expert consensus have been established to date.

Etiology and Pathophysiology

The existing literature on carotid artery aneurysms encompasses both true and false aneurysms, each with various underlying etiologies. ECAAs arise from diverse etiologies, including atherosclerosis, infections, fibromuscular dysplasia, connective tissue disorders associated with traumatic or spontaneous dissections, and pseudoaneurysms following prior surgical repairs [1] Morphologically, aneurysms are categorized into true aneurysms, pseudoaneurysms, and dissecting aneurysms; true aneurysms are primarily due to atherosclerosis, pseudoaneurysms commonly result from trauma, and dissecting aneurysms tend to develop at locations with the highest fluctuations in blood pressure. Beyond these primary causes, connective tissue disorders like Marfan syndrome and Ehlers-Danlos syndrome may also increase the risk of aneurysm development in patients. The two predominant groups account for a majority of cases in large series, with atherosclerotic and pseudoaneurysms constituting approximately 35% to 66% and 12.5% to 82% of aneurysms, respectively (Central Illustration) [4,7,9-12].

Diagnosis and Clinical Findings

Carotid aneurysm patients exhibit a variety of clinical presentations. The majority are male, with studies indicating a male predominance of 49% to 86%. Patient ages vary widely, from 19 to 95 years, depending on the aneurysm's etiology; however, the majority are in their sixth or seventh decade of life.

With advancements in imaging, many asymptomatic carotid aneurysms are now detected incidentally. In contrast, larger aneurysms are more likely to produce symptoms. The percentage of patients presenting with a pulsatile mass ranges from 12% to 93%. Furthermore, cerebral ischemic events are reported in 12% to 51% of cases. Less common symptoms include cranial nerve deficits, infections, difficulty in swallowing, tinnitus, bruit, bleeding, hoarseness, tracheal obstruction, and dizziness [4,7,9,10]

Therapeutic Approaches

Treatment is individualized for each patient, as no single approach is universally recommended for all carotid aneurysms. Management options for ECAAs include open surgery, endovascular therapy, hybrid procedures, and conservative treatment with antiplatelet and anticoagulant agents; however, there are currently no established guidelines or expert consensus.

The primary goal of treatment is to prevent local mass effects, rupture, and neurological deficits resulting from embolization or thrombosis. Personalized treatment approaches should take into account the aneurysm's size, location, and morphology, alongside patient comorbidities and the potential risks of intervention-related complications. Over the years, a variety of treatments have been attempted, ranging from supervised medical management to open surgical interventions and more recently to endovascular options. A thorough understanding of the natural progression of different treatments and the associated risk of complications is essential for determining the most suitable treatment option.

Fankhauser and colleagues conducted a 15-year review at the Mayo Clinic, analyzing 141 carotid aneurysms diagnosed in 132 patients [12]. Out of the 141 aneurysms, seventy-five were managed non-surgically. Treatment strategies included antiplatelet therapy, anticoagulation, or periodic imaging, based on the clinician’s assessment. Most of these patients were asymptomatic, with prior imaging indicating aneurysm stability. During the study period, none of the patients who received medical management experienced significant morbidity or mortality related to the aneurysm.

Open surgical techniques vary based on the patient’s anatomy and the underlying pathology, and may include ligation, resection with primary repair, resection with interposition grafting, or patch repair of the artery. Previous studies indicate that resection with interposition is performed in approximately 14% to 57% of cases, resection with primary anastomosis in 6% to 31%, patch angioplasty in 9.5% to 66%, and ligation in a considerably lower range of 0% to 1.6% [4,7,9,10].

Ligation of the internal carotid artery is generally reserved for emergency situations, such as arterial rupture, particularly when infection is the primary cause. In these cases, patients are often placed on anticoagulation therapy to prevent embolization as the artery fills with thrombus. Although there is no standardized anticoagulation duration, some groups recommend treatment periods ranging from 2 weeks to 3 months [13,14].

El-Sabrout and Cooley documented favorable outcomes in cases of pseudoaneurysms associated with prosthetic patches by restoring the arteries to a healthy state and performing re-patch angioplasty [4]. Open surgical procedures generally achieve high technical success rates, though surgical risks can vary widely. Reported early mortality rates range from 0% to 7%, perioperative stroke from 0.7% to 11%, cranial nerve injuries from 0% to 66%, hematoma formation from 0% to 5%, acute renal failure from 0% to 1.5%, thrombosis from 0% to 6%, myocardial infarction from 0% to 1.7%, and infection from 0% to 1.7% [4,7,9,10]. Cranial nerve injuries can affect the facial, vagus, spinal accessory,

hypoglossal, and glossopharyngeal nerves. Careful dissection and minimal manipulation of the aneurysm are recommended during carotid endarterectomy to reduce the risk of embolization [15].

Endovascular techniques are often favored for aneurysms located in the distal segment of the ICA. With advancements in technology, the endovascular approach has gained popularity in managing carotid aneurysm disease. However, the presence of unstable thrombus within an aneurysm or aneurysms in highly tortuous carotid arteries are relative contraindications for endovascular repair. Li and colleagues recently performed a systematic review focused on the endovascular treatment of carotid artery aneurysms. They found that the average aneurysm diameter was 26.3 mm, with the internal carotid artery as the most frequently affected site. Covered stents were used in 68% of cases, treating 83% of true aneurysms and 67% of pseudoaneurysms with covered stents. Success rates were recorded at 100% for true aneurysms and 92.4% for pseudoaneurysms. The average follow-up duration was 15.4 months. The patency rate for covered stents was 91.8%, and the thrombosis rate of the aneurysm sac was 95.8%. In comparison, the patency rate for bare metal stents was 97.1%, with a thrombosis rate of 70.6%. Late complications occurred at rates of 8.3% and 23.5%, respectively, while the overall occlusion rate was 6.3%. The stroke rate with covered stents was 2.5%, whereas no strokes occurred with bare metal stents [16].

In our clinic, we prefer open surgical repair. The aneurysm originated from the internal carotid artery, 2 cm above the bifurcation. We resected the aneurysm sac and restored continuity with a PTFE graft. The procedure was completed without complications, and no issues were encountered during the 18-month follow-up of the patient.

Discussion

Carotid aneurysms are rare pathological conditions with diverse etiologies, yet comprehending their clinical characteristics and treatment options remains crucial. ECAAs are rarely seen, constituting less than 1% of all aneurysms, and have mostly been published as case reports. Most ECAAs are commonly found at the carotid bifurcation or further along in the ICA, with causes that include atherosclerosis, infections, fibromuscular dysplasia, connective tissue disorders, trauma, prior surgeries, and radiation exposure [12-17]. The most common underlying cause of ECAA is atherosclerosis, which has been shown to be a leading factor in 37-42% of aneurysms in previous series [18,19]. In some series, it has been observed that 35% to 51% of aneurysms are due to trauma [16-20]. Other series report that pseudoaneurysms in previously operated endarterectomy sites account for 26-57% of aneurysms [4,19]. Connective tissue disorders that impact the carotid arteries include Marfan syndrome, Ehlers-Danlos syndrome, osteogenesis imperfecta, and pseudoxanthoma elasticum [21,22]. While polycystic kidney disease and neurofibromatosis are not formally categorized as connective tissue disorders, they can also contribute to the development of carotid artery disease [23,24]. If a condition predisposing a patient to carotid aneurysm is suspected, further investigation is necessary, including a review of family history, pathology consultation, and imaging of affected arterial regions.

Regarding symptoms, cerebral thromboembolism and partial compression are frequently reported as initial signs, whereas ECAA rupture remains underreported [25,26]. ECAA is typically diagnosed through clinical symptoms and imaging studies. Previous studies have reported a pulsatile neck mass in 93% of patients and neurological symptoms in approximately 50% of patients [7,9,27].

The increased use of imaging devices has led to a rise in the diagnosis of asymptomatic ECAAs. Grant T et al. reported that 49% of ECAAs in their case series were discovered incidentally [12]. Ultrasound is a reliable and effective tool for differentiating between a solid tumor and vascular disease, while CT angiography can be used to guide the choice of surgical procedure [28]. Additional imaging techniques, such as magnetic resonance angiography (MRA) and computed tomography angiography (CTA), offer more detailed anatomical insights, which are essential for surgical planning.

Treatment options, including medical management, open surgery, and endovascular intervention, should be customized to each patient to achieve the best possible outcomes [29]. Medical treatment, including drug selection for ECAAs, remains underexplored and requires further investigation. Conservative treatment for ECAAs is rarely reported. McCollum et al. proposed that asymptomatic ECAAs could undergo conservative treatment via antiplatelet therapy or anticoagulation [5]. Until more data on medical treatment for ECAA patients becomes available, it is advised to follow treatment guidelines for the most probable underlying condition. Surgical intervention is defended for all ECAAs due to the high risk of mortality in non-operated cases [5,30-32]. Surgical intervention is recommended for all ECAAs, whether symptomatic or asymptomatic, due to the high mortality risk associated with untreated cases [33].

The surgical management of carotid aneurysms depends on the aneurysm's etiology, size, and location, as well as the patient's comorbid conditions. Open surgical intervention has been the standard treatment since Cooper's pioneering surgery in 1805 (Figure 1). Garg et al. outlined five key surgical options: aneurysm clipping, primary anastomotic excision, interposition graft excision, extracranial-intracranial bypass, and carotid ligation [20]. Ligation carries a stroke risk of up to 25% and a mortality risk of up to 20% [29]. Therefore, it tends to be used only as a last resort. While open surgery has been successful, protecting cranial nerves from injury during surgery has become a growing concern. Furthermore, aneurysms near the cranial base

present additional challenges due to the difficulty in exposing the surgical area. The advent of endovascular therapy has helped to overcome these issues by minimizing the exposure needed for open repair, preserving cranial nerves, and enabling the procedure to be performed under regional anesthesia, thus reducing surgical invasiveness and shortening hospital stays [32,34].

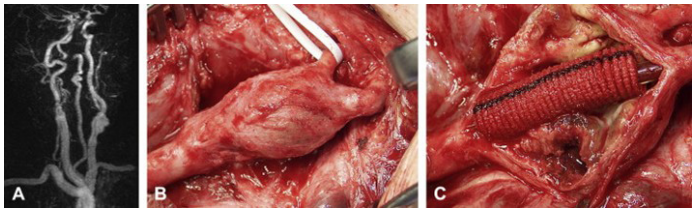

Figure 1. Surgical exposure and resection of an extracranial carotid artery aneurysm. The aneurysm sac is being isolated, and continuity is restored using a dacron graft. This procedure demonstrates the open surgical technique for managing extracranial carotid aneurysms, highlighting the meticulous dissection required to avoid cranial nerve injury and ensure successful vascular reconstruction [7]

Currently, positive procedural outcomes are reported in case series advocating for the endovascular approach in the treatment of ECAA; however, there is a lack of medium- or long-term follow-up. A recent systematic review analyzed 959 ECAA cases, with 750 treated using open surgery (perioperative complications included cranial nerve injury in 9%, stroke in 4%, and death in 2%), while 85 cases were managed with endovascular therapy, showing no perioperative complications, despite one case of restenosis [35]. A systematic review of 224 patients treated with endovascular therapy demonstrated that stenting for ECAA is technically feasible, with a high procedural success rate of 92.8%. The complication rate was relatively low, including post-procedure endoleak in 8.1%, stroke in 1.8%, and cranial nerve injury in 0.5%. The average follow-up period was 15.4±15.3 months, with a stent patency rate of 93.2% [16].

In conclusion, the main objective in managing ECAA is to prevent permanent neurological deficits caused by thromboembolism and thrombosis, as well as to eliminate the risk of rupture. ECAA management has evolved with advancements in surgical techniques, with the complete removal of the aneurysm and restoration of blood flow to the affected arteries being considered the optimal approach. Open surgical intervention has long been the primary treatment for carotid aneurysm disease; however, evidence also supports the effectiveness of endovascular treatment. Although some data on endovascular treatment exist, long-term outcomes are not well-established, and the patient population studied remains relatively small. Most evidence for both open and endovascular interventions comes from retrospective case series or case reports. There are no definitive guidelines, expert consensus, or treatment algorithms for managing this condition. Current literature indicates variability in intervention thresholds based on aneurysm diameter for extracranial carotid artery aneurysms (ECAA) (Table 1). Some studies suggest surgical or endovascular intervention for aneurysms larger than 2-3 cm, while others propose a threshold of 1.5 cm, highlighting the lack of consensus on standardized criteria for intervention [7,17]. The absence of a diameter-based classification system poses challenges in clinical decision-making and may lead to inconsistent management practices. A diameter-based classification could help guide clinical practice more effectively. For instance, small aneurysms (<1.5 cm) might be monitored conservatively, medium-sized aneurysms (1.5-2.5 cm) could warrant intervention based on symptoms and growth rate, and large aneurysms (>2.5 cm) would likely require surgical or endovascular treatment. Developing such a framework would address a critical gap in the management of ECAA, providing clinicians with a more structured approach to risk stratification and treatment selection. As treatment approaches continue to evolve, there is an urgent need for substantial randomized studies to determine the most effective treatment strategies.

Table 1. Summary of studies on ECAA treatments

Author	Year	Number of cases	Treatment method	Outcomes
Mc Collum et al.	1979	21	Surgical	High success rate, low complication rates
El-Sabrout and Cooley	2000	21	Surgical	High success rate, perioperative stroke up to 11%
Attigah et al.	2009	50	Surgical	90% success, low mortality and complication rates
Donas et al.	2009	224	Endovascular	91.8% patency, low complication rates
Zhou et al.	2010	80	Endovascular and surgical	High success, low complications with minimally invasive approach
Li et al.	2011	85	Endovascular (stenting)	92.8% technical success, 93.2% stent patency, low stroke rate
Pulli et al.	2013	60	Surgical	95% success, low complication rates
Fankhauser et al.	2015	141	Surgical and medical management	Surgery: high success; medical: stable patients
Garg et al.	2012	100	Surgical (ligation, bypass, etc.)	25% stroke risk after ligation, variable success
Hoffman et al.	2022	85	Endovascular and surgical	Fewer complications with endovascular, high success with surgery

CONCLUSION

ECAA are rare yet potentially serious pathologies that can lead to significant clinical consequences. In this review, we have examined the current understanding of ECAA, including its etiology, clinical presentation, diagnostic methods, and treatment strategies. ECAA is often asymptomatic and frequently diagnosed incidentally during evaluations for other conditions. The most common symptoms include pulsatile neck masses and cerebral ischemic events.

Current treatment approaches encompass surgical, endovascular, and conservative methods; however, there is no established standard treatment guideline for ECAA (Central Illustration). This lack of consensus leads to variability and uncertainty in clinical practice. More clinical research is essential to enhance the management of ECAA. It is recommended to conduct multicenter studies to establish long-term outcomes and develop effective treatment methods. Proper management of ECAA will not only improve patients' quality of life but also reduce morbidity and mortality rates. Given the variability in clinical presentation and the lack of standardized treatment protocols, further multicenter randomized trials are essential to determine the most effective management strategies for ECAA. Such studies would help establish evidence-based guidelines and improve both short-term and long-term patient outcomes.

Future studies should focus on the pathophysiology, risk factors, and response to treatment in ECAA to address existing knowledge gaps in this field.

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