Chapter 12 Extracranial Carotid Artery Aneurysms

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Introduction

Aneurysms in the extracranial carotid artery are rare and account for less than 1% of all arterial aneurysms [\[1](#page-12-0)[–4](#page-13-0)]. Large case series describing more than 50 patients are limited to 5–6 reports in the medical literature. In an angiographic study of 5000 patients, House and Baker et al., found extracranial ICA aneurysms only in 8 (0.16%) patients [[5\]](#page-13-1). Given the rarity of such lesions, limited knowledge is available about their true incidence and natural history. Despite this, the lesion is considered to be of significant clinical importance, mostly for its potential for giving rise to embolic and occlusive strokes, which may occur in as high as 50% of cases [[6\]](#page-13-2).

Various etiologies have been described for the occurrences of these rare aneurysms including atherosclerosis, trauma, connective tissue disorders, etc. In most instances, the presenting symptoms are either due to stroke or local compression. Recently, a significant number of asymptomatic patients are being diagnosed incidentally. Although all segments of the extracranial carotid artery (ECCA) can be

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M. S. Park et al. (eds.), *Carotid Artery Disease*, https://doi.org/10.1007/978-3-030-41138-1_12

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affected, the internal carotid artery is the most common location with the external carotid artery as the rarest. Treatment depends on the symptoms and etiology, along with the location. While small and/or asymptomatic lesions are likely best managed conservatively, symptomatic and/or enlarging asymptomatic aneurysms may require intervention to reduce the risk of stroke and rupture. Depending on anatomic and physiological issues related to collateral circulation, both reconstructive and deconstructive techniques with or without bypass may be needed. The treatment plan should consider all open microsurgical and endovascular techniques and tools.

Classification

ECCA aneurysms can be classified as true or false (pseudo) aneurysms based on their etiology and pathological characteristics. True aneurysms are surrounded by all three vessel wall layers (intima, media, and tunica) but, have a dilation of at least 50% when compared to the normal diameter [\[7](#page-13-3)]. Pseudoaneurysms are caused by the disruption of the arterial wall and may contain a hematoma in the arterial lumen. Common causes of pseudoaneurysms are trauma, dissection, or local infection. Literature showing the relative prevalence of extracranial true vs pseudo carotid artery aneurysms varies between studies. In a study, describing 42 cases, the incidence of pseudo vs true aneurysms were nearly equal [[8\]](#page-13-4). However, in another larger study published by the Mayo Clinic describing 141 patients, 82% were pseudoaneurysms as compared to 18% of true aneurysm [\[9](#page-13-5)]. A classification scale based on anatomical segment has been established which has importance in treatment planning (Fig. [12.1](#page-1-0) and Table [12.1\)](#page-2-0).

Fig. 12.1 Classification of external carotid artery aneurysms. (Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved)

Normal	Type 1	Type 2	Type 3	Type 4	Type 5
Natural	Isolated	Aneurysms of the	Aneurysms of Combined		Aneurysm of
looking		Aneurysm of internal carotid	the carotid	aneurysms of the the common	
artery	internal	artery with	bifurcation	internal and	carotid artery
	carotid	involvement of the		common carotid	
		bifurcation		artery	

Table 12.1 Classification of external carotid artery aneurysms

Although any segment of the extracranial carotid artery can give rise to aneurysms, the segment around the carotid bifurcation has a higher tendency for aneurysm formation, because of the higher burden of atherosclerotic disease. This segment is also most exposed to external trauma and surgery (carotid endarterectomy, neck dissection for tumors) which increases its vulnerability to pseudoaneurysm formation. Although, the carotid bifurcation is not commonly involved per se, the diseased artery at the bifurcation can lead to increased flow velocity and stress in the internal carotid artery just distal to the bifurcation and may pose risk for aneurysm formation. In the Mayo Clinic series, 81% of the aneurysms were located in the internal carotid, 8% in the common carotid, 10% at the bifurcation, and 1% in the external carotid [[9\]](#page-13-5). In a literature review of 1332 ECCA aneurysms, Welleweerd et al. found that the ICA was the most common location comprising 46% of cases followed by the bifurcation which accounted for 20% of cases [[10\]](#page-13-6).

Etiology and Risk Factors

The etiology of aneurysm formation in the extracranial carotid arteries is multifactorial and is broadly due to hemodynamic changes from atherosclerotic changes or from weakness of the arterial wall due to direct or indirect injury. Atherosclerosis is the most likely cause for formation of true aneurysms in elderly individuals. In the review by Welleweerd et al., atherosclerosis accounted for up to 38% of the aneurysms [\[10](#page-13-6)]. Although arterial wall degeneration has been shown as the most common mechanism, another study from these same authors analyzing the histological characteristics of these aneurysm identified arterial dissection as another important etiology [\[11](#page-13-7)]. No patients presented with a prior history of arterial dissection, but the histological findings of arterial dissection suggests that some degree of dissection, even in asymptomatic patients, may lead to the future development of aneurysms. A more frequent association is also seen with coronary artery disease and chronic obstructive pulmonary disease [\[12](#page-13-8)]. Other etiologies for formation of true aneurysms especially in younger patients include fibromuscular dysplasia, connective tissue disorders, inflammatory diseases, Marfan syndrome, Ehler-Danlos Syndrome, tuberous sclerosis, and cystic medial necrosis [\[9](#page-13-5), [13](#page-13-9)].

Pseudoaneurysms are normally caused by prior intervention or infections. Both true and false aneurysms can be caused by blunt or penetrating trauma depending on the severity of the arterial wall injury [[12\]](#page-13-8). Primary and secondary infection of the neck can also lead to ECCA aneurysms. Primary infections are rare with only 100 cases reported in the literature [\[14](#page-13-10)]. Carotid endarterectomy has been historically shown to cause less than 1% of pseudoaneurysms of all procedures performed [[15\]](#page-13-11). However, in the Mayo Clinic series, prior endarterectomy was implicated in the formation of 24% of the psuedoaneurysms [[9\]](#page-13-5). Blunt injury and penetrating cerebrovascular injury can also lead to pseudoaneurysms. Additionally, the timing of delayed aneurysm formation can be as long as 20 years [\[9](#page-13-5)]. Spontaneous carotid dissections can also give rise to aneurysm formation [[16\]](#page-13-12). Nearly 30% of patients with spontaneous dissections ultimately developed aneurysms [\[17](#page-13-13)]. Radiation, another risk factor for formation of ECCA aneurysms, may result in radiationinduced changes of the arterial wall with subsequent degeneration and weakening [\[18](#page-13-14)]. In one study, 70% of the patients who received a second course of radiotherapy developed a pseudoaneurysm [[19\]](#page-13-15). Whether this justifies surveillance with MRA or another modality merits study.

Presentation

ECCA aneurysms are rare, but can present in patients of any age. Rupture and thrombotic events are similarly rare. Due to an increase in neurovascular imaging and improvements in imaging techniques, a greater number of ECCA aneurysms are being reported. In one study, half of the ECCA aneurysms were asymptomatic and discovered incidentally [\[9](#page-13-5)]. The common modes of presentation are outlined below.

Stroke

ECCA aneurysms can present as a transient ischemic attack (TIA) or strokes which can occur in almost half of patients. An asymptomatic, pulsatile mass is the second most common form of presentation found in nearly one third of patients [\[10](#page-13-6)].

Compression

Another important mode of symptomatic presentation is compressive symptoms due to pressure on surrounding structures. Local compression can cause pain in the neck and retro-orbital areas along with headaches [[12\]](#page-13-8). It is rare to have a ruptured aneurysm compress the pharyngeal muscles resulting in dysphagia [\[9](#page-13-5)]. Compression of the glossopharyngeal nerve can cause angular pain dysphagia and pharyngeal dysfunction. If compression involves the sympathetic chain, a Horner's Syndrome can result. Vagal compressions can result in hoarseness and hypoglossal compres-sion can cause tongue deviation and decrease in function [[6\]](#page-13-2).

Rupture

However rare rupture of ECCA aneurysms are, one cannot minimize its risks, as fatal airway compressions may occur. Infected aneurysms are more likely to rupture, compared to non-infected aneurysms [\[13](#page-13-9)]. Pseudoaneurysms following radiation treatment are also known to rupture and must be kept under surveillance [\[19](#page-13-15)].

Inflammation

ECCA aneurysms that are infected can present as an expanding, pulsating cervical mass associated with local pain, tenderness fever, dysphonia and dysphagia [[14\]](#page-13-10).

Diagnosis and Imaging

A pulsatile mass in the neck is found in nearly 90% of the patients [[20,](#page-13-16) [21\]](#page-13-17). Ultrasound is the initial imaging modality for evaluation of neck masses. But further imaging with computer tomographic angiography (CTA) or magnetic resonance angiography (MRA) is usually required to help define the aneurysm. CT or MR angiograms give a better view of the aneurysm and its location, diameter, and/or the presence of a thrombus. It can also provide information about both intracranial and collateral circulation. Catheter arteriography is highly sensitive and can play a role in both diagnosis and collateral assessment. Imaging of other areas is recommended to document associated aneurysms, especially in patients with connective tissue disorders and in patients with a strong family history of aneurysms. In a 48-patient study, 24% of patients with true aneurysms had an associated aneurysm. The most common place was the abdominal aorta [\[22](#page-13-18)].

Treatment

Given the rarity of ECCA aneurysms, the optimal management protocol for these lesions is less clearly defined. But broadly, the management options include observation, open surgical repair, and endovascular treatment. The primary goal of treatment is to prevent thromboembolic complications and alleviate local compression. The current evidence on management of ECCA aneurysms is based primarily on observational studies. A randomized control trial is largely impractical because of low prevalence rates of the disease. Therefore, most of the cases are usually managed at the discretion of the physician based on individual patient factors. Conservative management for large and symptomatic lesions may not be advisable. Older literature suggested a high incidence of mortality from observed symptomatic lesions (as high as 71% described in older literature [[23\]](#page-14-0)) in untreated patients. This has not be substantiated in more modern series which include a much higher number of more benign incidentally discovered lesions. An expansion of treatment options including endovascular management has lowered the threshold for treatment in select cases. A summary of treatment options is outlined in Table [12.2](#page-6-0). A general guideline has been developed from the current evidences and is depicted in Fig. [12.2.](#page-8-0)

Conservative Treatment

Conservative treatment is usually advocated in asymptomatic and incidentally detected aneurysms. Medical management focuses on prevention of progression of atherosclerosis using statins and prevention of thromboembolic episodes using antiplatelets (aspirin with or without clopidogrel) or anticoagulation (warfarin or apixaban). Anticoagulation may be preferred if an acute dissection is suspected, particularly if a thrombus is present. In select low risk patients, observation and follow up with serial imaging can be judiciously used.

Open Surgical Management

Open surgical management for extracranial carotid artery aneurysms dates back to 1805 when Sir Astley Cooper performed ligation of the common carotid artery to treat an aneurysm [[24\]](#page-14-1). Unfortunately the patient died 48 hours later. However, this opened the door to technical advances and subsequent reports of successful ligation were reported. Till the mid twentieth century, carotid ligation was the preferred method of treatment of such aneurysms, although with a risk of perioperative stroke of 30% [\[25](#page-14-2)]. Surgical treatment options can include resection of the aneurysm and end to end anastomosis, resection followed by dacron or saphenous graft anastomosis, and/or aneurysmorrhaphy.

The type of surgical intervention depends upon the size and location of the aneurysm, the etiology, and patient factors like age, associated comorbidity, and/ or type of presentation. In the series by Radak et al., most of the Type 1 aneurysms were managed with resection and end-to-end anastomosis, Type 2 aneurysms by resection, shortening, and ICA re-implantation, and Type 3 and 4 aneurysms by resection with tubular graft insertion [[26\]](#page-14-3). Although atherosclerotic aneurysms are relatively easier to repair, pseudoaneurysms especially from a previously operated artery, prior infection, or radiation are challenging. Meticulous dissection, removal of the diseased segment including the suture line, and/or use of an appropriately sized graft are basic nuances for successful repair. The most described complication after surgical repair are thromboembolic complications and postoperative cranial nerve dysfunction. Proper use of carotid clamping tech-

(continued)

(continued)

Fig. 12.2 A general guideline for treatment options, developed from the current evidence

niques and intraoperative anticoagulation are important to prevent potential clot migration. Similarly, careful dissection during manipulation of the posterior wall is essential to prevent postoperative cranial nerve dysfunction. The rates of postoperative stroke in the literature range from 11% to 22% with a permanent deficit of 3% to 13% [\[21,](#page-13-17) [27](#page-14-4)[–31\]](#page-14-5). Some of the common surgical procedures are discussed below.

Carotid Ligation

Carotid ligation is a simple and relatively straightforward procedure. In an early series of 22 patients reported by Schievink et al. in 1994, five patients underwent carotid ligation without any ischemic or other neurological complications with a long term follow up of 8.8 years [[32\]](#page-14-6). The most obvious requirement to determine candidacy for ligation is documentation of adequate collateral flow by a preoperative balloon occlusion test with possible inclusion of a perfusion study such as xenon-133 cerebral blood flow monitoring or single photon emission computed tomography (SPECT) [\[33](#page-14-7)[–36](#page-14-8)]. Despite good cross circulation during testing, there is still a risk of ischemia in the ipsilateral circulation. The newer trend of surgical management is towards anatomic reconstruction of the affected segment rather than ligation. In the review by Welleweerd et al., carotid ligation consisted of only 5% of all open procedures in 1102 surgically treated aneurysms [[10\]](#page-13-6).

Resection of the Aneurysm and Reconstruction

Resection of the aneurysm is now the most commonly performed open surgical procedure. Options for restoration of flow after resection include direct anastomosis, interposition graft, partial resection followed by patch graft, and aneurysmorrhaphy. Aneurysms above the carotid bulb with short segment involvement of the ICA can be resected with primary end-to end anastomosis. However, carotid bulb involvement or long segment disease usually requires the use of an interposition graft. Other important considerations in this regard include the health of the vessel wall, tension on the ends, and the history of radiation or infection of the surrounding tissues. Fusiform dilation of the carotid bulb or the carotid bifurcation can be suitably managed with partial resection of the aneurysm wall followed by reconstruction with primary closure. Extensive circumferential involvement by false aneurysms may need a venous patch graft or an artificial dacron patch graft for reconstruction of the lumen.

An important concern regarding aneurysm resection is the exposure of distal involved segments of the ICA. Different approaches for high cervical exposures have been described in the literature. In most cases, this can be achieved by mobilization of the parotid gland with elevation of the facial nerve. The occipital artery may need division along its course. A higher exposure can be obtained with fracture and removal of the styloid process, along with division of the posterior belly of the diagastric muscle along its origin. The most distal segment of the extracranial ICA is accessed by drilling of the mastoid tip with exposure of the stylomastoid foramen. A more extreme form of the skull base approach has been described by Fisch et al., which can expose the proximal portion of the petrous segment [[37\]](#page-14-9). This surgery involves mobilization of the facial nerve proximal to the geniculate ganglion by drilling its canal in the mastoid bone and middle ear with anterior transposition of the nerve. This safely exposes the bony canal of the proximal petrous carotid segment. Drilling of the thin bone of the petrous bone in the tympanic and mastoid portion, along with division of the fibrous ring around the ICA at its entrance into the canal, enables mobilization of the artery. This procedure requires extensive middle ear manipulation with the sacrifice of the ossicles and necessarily comes with the cost of hearing loss.

The most common complication associated with cervical carotid exposure and manipulation is cranial nerve damage. The most common nerve involved in the exposure is the vagus nerve and its branches including the pharyngeal and superior laryngeal nerves. This usually leads to dysphagia and dysphonia in the immediate post-operative period. Damage to the spinal accessory nerve is also possible during mobilization of the sternocleidomastoid muscle. The facial nerve is the other cranial nerve which can be damaged, especially with high cervical exposures which is at risk during parotid manipulation or manipulation around the stylomastoid foramen. Other complications related to anastomosis or reconstruction include anastomotic leak or dehiscence leading to a neck hematoma, thromboembolism, or complete graft thrombosis. Chronic complications include graft stenosis at the anastomosis site and/or formation of pseudoaneurysms.

Extracranial (Cervical) to Intracranial Bypass

An extracranial to intracranial bypass procedure may be needed when the proximal extent of the aneurysm is too high for a safe resection, with complex aneurysms, or with a history of prior infection or radiation with extensive scarring of the neck. An important consideration during the decision making process for a bypass procedure is the type of bypass needed. A balloon test occlusion (BTO) provides objective evidence to determine the degree of tolerance to vessel occlusion and the adequacy of cross flow from the contralateral and posterior circulations. This test can help in determining the type of bypass, high flow or low flow, needed to sustain the circulation. The BTO usually consists of four components which are performed during the temporary occlusion of the ipsilateral vessel [\[36](#page-14-8)]. It includes clinical assessment in which the patient's neurological status is assessed while awake; hemodynamic assessment in which an angiogram may be performed from the contralateral vessel; neurophysiological assessment in which the EEG waveforms may be monitored; and finally a provocative assessment in which the mean arterial pressure is decreased using pharmacologic agents and clinical and neurophysiological tests are repeated.

Surdell et al. have described three broad groups based on the BTO findings [[35\]](#page-14-10). The patients who pass all four modalities have adequate cross circulation and, therefore, should tolerate a permanent vessel occlusion alone. Patients who only pass in their clinical assessment have moderate cross circulation and may need at least a low flow bypass (typically superficial temporal artery to middle cerebral artery bypass) in addition to vessel occlusion. Patients who fail the clinical assessment have poor cross circulation and may require a high flow bypass to sustain the circulation. Pedicled arterial grafts including the superficial temporal artery (STA) or occipital artery (OA) are used for a low flow bypass. The free arterial (radial artery) or venous grafts (saphenous vein) are used for high flow grafts. High flow bypasses are technically more challenging, but are associated with high patency rates if successful. The saphenous vein graft proximal connection can be to an external carotid artery branch, including the superficial temporal artery proximal to its bifurcation. The size of the parent arteries and the location of the aneurysm help define the best anatomic solution. Distal anastomosis can be to the supraclinoid carotid artery or to a large branch of the middle cerebral artery after its first bifurcation. In any of the bypass procedures, the major risk is failure of the anastomosis with subsequent ischemic stroke. Vasospasm can affect outcomes with radial artery bypass. This can be mitigated with postoperative infusion of a vasodilator and by keeping the graft integrated with its venous circulation [[38\]](#page-14-11).

Endovascular Management

With increasing technical advancements, endovascular management has grown in feasibility and efficacy. Challenging Type 1 and low Type 4 lesions, aneurysms associated with scarring, and aneurysms with other unfavorable local conditions may be better treated with endovascular reconstruction with stents. Endovascular treatment may have a more effective long-term outcome in pseudoaneurysms as compared to true aneurysms, possibly due to the self-limiting nature of pseudoaneurysm etiology and the lower incidence of intraluminal thrombus in pseudoaneurysms [\[9](#page-13-5)]. The rationale of using endovascular stenting as a treatment in presence of compressive symptoms by the aneurysm is controversial. Theoretically, compression symptoms should preclude endovascular procedures, as it does not decompress or remove the aneurysm. However, proponents of endovascular treatment argue that the thrombosis of the aneurysm with subsequent remodeling reverses the compression of the surrounding structures. In the series by Leng Ni et al., two patients presented with compressive symptoms who underwent endovascular stenting [\[39](#page-14-12)]. One patient who had swallowing difficulty improved. However, the other patient had persistent hoarseness after stenting.

Currently, the options for endovascular treatment include either covered or bare metal stents, stent placement with coil embolization, or vessel occlusion. Covered stents may be more effective as they exclude the aneurysm from the circulation and also prevent endoleak and recanalization [[18\]](#page-13-14). Li et al. in their systematic review on endovascular procedures demonstrated a significantly lower incidence of endoleak, re-intervention, and late complications with covered stents [\[40](#page-14-13)]. However, the major disadvantage of covered stents, as compared to bare metal stents, is their stiff construct which precludes their use in tortuous anatomy. Another important limitation of covered stents is the discrepancy in parent artery diameter when aneurysms involve the ICA bifurcation. This scenario is associated with in increased risk of endoleak when covered stents are used. Nonetheless, a number of authors have reported promising results in carefully selected patients. Leng Ni et al. in their series, successfully used PTFE covered stents, such as Viabahn endoprosthesis, which has significant flexibility and conformability to the vessel configuration and neck movement [[39](#page-14-12)]. Welleweerd in their series of 7 patients described successful use of bare metal stents [\[41\]](#page-14-14). Although, the metal coverage of the stents used were less than 10%, an 86% exclusion rate in their series was demonstrated.

With the advent of flow diverting stents, there is a paradigm change in the treatment of extracranial aneurysms, especially pseudoaneurysms. Flow diverting stents (which have been designed and approved for select intracranial aneurysms) provide a more flexible construct that can easily be navigated through tortuous segments. Initially, the major concern was stent migration due to the increased caliber of the cervical ICA, increased mobility of the artery, and tendency of the cervical vessel to change with neck movements [\[42](#page-14-15)]. In the early description by Rahal et al., a second, non-flow diverting stent was used as an anchoring support to mitigate the potential

for foreshortening and migration of the flow diverting stent [[43\]](#page-14-16). Chen et al., in 2016, described their successful use of multilayer stents by covering multiple bare stents. They achieved a 100% occlusion rate in their 8 patients [\[44](#page-15-0)]. Subsequently, there have been several published reports describing successful use of flow diverting stents in cervical pseudoaneurysms [\[45](#page-15-1)[–47](#page-15-2)]. The continued evolution of stent configurations may make endovascular treatment a more feasible and safer option for these ECCA aneurysms.

The common complications described after endovascular treatment include post procedure strokes, stent restenosis, and occlusion. Adequate anti-thrombosis in the perioperative and post-operative period is essential. In their review, Li et al. demonstrated a 92.8% procedure success rate and 93.2% stent patency rate with a very low complication rate. Another review specifically addressing carotid artery dissection associated pseudoaneurysms demonstrated a 98.3% occlusion rate with use of an endovascular stent with or without coiling [\[48](#page-15-3)]. The restenosis rate after stenting is reportedly around 6%, which usually occurs adjacent to a bent or kinked carotid artery [\[39](#page-14-12)].

Conclusion

ECCA aneurysms are rare lesions with limited validated recommendations for their management. Although a significant number of patients are asymptomatic, these lesions can still give rise to thromboembolic complications. Given the rarity of ECCA aneurysms, a definite recommendation regarding their management is still not available. Several factors play a role in determining the optimal treatment modality, including aneurysm type, location, and cause. Similarly, other important factors include a prior history of radiation or neck surgery, associated comorbidities, age, and functional status. Open surgery with resection of the aneurysm and primary anastomosis or graft reconstruction is favored for true aneurysms and large aneurysms with compressive symptoms. Endovascular treatment with covered or bare metal stents is another emerging treatment with comparable results. Pseudoaneurysms, especially with prior infection or radiation may be better treated by endovascular means. Balloon test occlusions can help define extracranial to intracranial bypass options or support parent artery occlusion as a treatment option. Observation and follow up is an option, especially for smaller asymptomatic aneurysms or in patients who are otherwise not candidates for intervention.

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