Cavernous carotid aneurysms: to treat or not to treat?

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Most cavernous carotid aneurysms (CCAs) are considered benign lesions, most often asymptomatic, and to have a natural history with a low risk of life-threatening complications. However, several conditions exist in which treatment of these aneurysms must be considered, namely, large lesions, which confer increased risk of rupture according to the ISUIA data;¹⁰ symptomatic lesions that undergo acute thrombotic changes; lesions that have evidence of aneurysm growth; unruptured symptomatic lesions with mass effect; unruptured lesions causing significant local bone erosion; and/or ruptured lesions. The current modalities of CCA treatment include expectant management, luminal preservation strategies, and Hunterian strategies with or without revascularization procedures. In this article, we discuss the sometimes difficult decision regarding whether to treat CCAs. We consider the natural history of several types of CCAs, the clinical presentation, the current modalities of CCA management and their outcomes to aid in the management of this heterogeneous group of cerebral aneurysms. (DOI: 10.3171/2009.2.FOCUS0920)

Key Words • cavernous carotid aneurysm • vascular surgery • microsurgery • endovascular therapy

cerebral hemispheres. The nomenclature for classifying and numbering the segments of the ICA typically refers to the adjacent structures, of which there are 7 anatomically distinct segments, C1–7, proximal to distal. The ICAs are primarily derived from two embryological sources: The C-1, or cervical segment, which extends from its origin to the petrous bone, is derived from the fetal third aortic arches. All other ICA segments, namely C2–7, represent cranial extensions of the embryonic dorsal aortae.⁵⁹ The C-4 segment, namely the cavernous portion, begins at the superior margin of the petrolingual ligament and extends to the proximal dural ring (Fig. 1). The C-4 segment is further divided into 3 subsegments, namely the posterior ascending or vertical portion, a longer horizontal segment, and a short anterior vertical portion. The bends between these subsegments are referred to as the posterior and anterior genu. The C-4 segment is the most medial structure within the cavernous sinus proper. Anterolaterally, CNs III, IV, and VI course through the sinus, with CN VI being the only CN whose course is within the sinus proper. In the lateral dural wall of the cavernous sinus course CNs III and IV along with the ophthalmic and maxillary divisions of the trigeminal nerve. Several small but important arterial branches arise from the C-4 segment and include the meningoypophyseal seal (consisting of the inferior hypophyseal artery, the tentorial artery [Bernaconi-Cassinari], and clival branches), the inferolateral trunk (consisting of arterial branches that supply the CNs of the cavernous sinus as well as the gasserian ganglion and cavernous sinus dura), and finally the capsular arteries of McConnell, which supply the pituitary gland. Anomalous embryonic anastomoses can be found on the C-4 segment with the most common being a persistent trigeminal artery, which is found

Background

Anatomy

The ICAs supply the greatest amount of blood to the
CCAs is thought to be highly size dependent, according to the ISUIA data, with asymptomatic CCAs 13–24 mm in size harboring a 3% rupture risk within 5 years and asymptomatic CCAs > 25 mm having a 6.4% rupture risk within 5 years. However, the ISUIA data are not without limitations and flaws. The ISUIA does not consider, with specificity, the influence of medical factors such as genetic risk, comorbidities, and modifiable risks (for example, smoking). Therefore, risk assessment of CCA rupture is limited. Bilaterality has also been linked to increased rupture risk.

**Imaging**

Imaging of the CCA presents a unique challenge given the rich environment of surrounding neural structures and bone. Precise anatomical characterization of CCAs can make the difference between high- and low-risk lesions with respect to rupture potential as well as treatment strategies. One important anatomical feature that has traditionally been difficult to delineate is whether the lesion is projecting into the subarachnoid space. Lesions near the dural rings can seem to project into the subarachnoid space; however, some of these lesions have been found in surgery to involve expansion of the cavernous sinus roof without penetration into the subarachnoid space, possibly reducing the rupture risk. Furthermore, the angle of the dural rings with respect to the ICA may alter the treatment approach (a more tangential dural ring allows more lateral carotid artery wall exposure, which may render surgical clip placement more feasible). Unfortunately, current imaging limitations, due mainly to inadequate spatial resolution, do not permit the thicknesses of these cavernous structures to be determined, therefore making identification of exact anatomical relationships difficult at best.

Noninvasive intracranial imaging is the most common mode of CCA detection. A standard imaging work-up for potential intracranial disorders often begins with a non–contrast-enhanced CT scan. Unruptured CCAs can often go unnoticed. However, ruptured CCAs can be detected due to the presence of acute hemorrhage, appearing as hyperdense signal emanating from within the cavernous sinus, in the surrounding subarachnoid space, or in surrounding areas such as the sphenoid sinuses. Computed tomography angiography has gained widespread popularity due to its speed of acquisition, its accuracy, its noninvasive nature, and the ability to create 3D reconstructions of the acquired images (Fig. 2). Critical anatomical information can be gleaned from CT angiography, including aneurysm dome dimensions, osmium size, osseous relationships with or without evidence of erosion, and other associated vascular lesions. Despite the advancement of 3D workstations for CT angiography imaging, the surrounding osseous structures of most CCAs make these reconstructions difficult at best to use for definitive imaging. Magnetic resonance angiography has also been used to image CCAs. The advantages of MR angiography are reduced imaging artifacts from surrounding osseous structures and the ability to reconstruct highly accurate 3D images. Advanced MR imaging techniques including dynamic and rotational viewing of CCAs allow delineation of the specific details of anatomical association—for
example, the dural rings and CNs. The current gold standard for intracranial vascular imaging remains cerebral DSA (Fig. 3). Advanced rotational cerebral angiography (3D-DSA) allows highly accurate image reconstructions of CCAs, which permit specific anatomical characterization. However, due to the temporal acquisition of 3D-DSA images, the anatomical accuracy of this modality is highly dependent on the quality of the equipment, contrast agent injection and concentration, and structural characteristics of the CCA itself (small-necked aneurysms with low inflow hemodynamics are not imaged as well as wide-necked lesions with higher inflow velocities).

**Clinical Presentation**

Most patients with CCAs are asymptomatic, and the lesions are most often discovered incidentally on intracranial imaging acquired during the work-up for unrelated conditions such as headache, closed-head trauma, or other nonspecific cranial, ocular, or facial complaints. However, of patients who do present with symptomatic CCAs, a significant majority are women, > 50 years old and Caucasian, who present with symptoms of mass effect.33,55,62 Progressive mass effect can be from the aneurysm dome on the cavernous sinus and its associated CNs, acute thrombosis, or spontaneous rupture, which are thought to be responsible in the majority of symptomatic cases.18,21,36,55 In the rare cases of traumatic or spontaneous CCA rupture, the origin of the mass effect is thought to be the result of increased intrasinus blood pressure resulting in compression of the cavernous sinus or complications due to venous reflux. Symptomatic acute thrombosis can result in either increasing mass effect on the local neural structures or disruption of the blood supply to those same neural structures.

The most common symptoms of mass effect from an unruptured CCA at presentation are diplopia and pain.55,62 Diplopia can result from isolated oculomotor nerve palsies—for example, CN III and VI pareses, independently or in combination, or the cavernous sinus syndrome, occurring in about 20% of patients, which involves CN III, IV, and VI, often leading to near-complete ophthalmoplegia. Pain has been reported to be of various forms, including, but not limited to, unilateral headaches, retro-orbital pain, and/or facial pain. Other oculofacial manifestations of symptomatic CCAs include ocular sympathetic paresis, compressive optic neuropathy, corneal hypesthesia, and trigeminal dysesthesias. The timing of the patient’s presentation normally reflects the etiology of CCA symptoms. More insidious presentations of diplopia and/or pain usually occur from enlarging CCAs resulting in progressive mass effect. More acute presentations of diplopia and/or pain are usually related to ruptured CCAs. However, although ruptured CCAs resulting in epistaxis are rare, the presentation of patients with these lesions is often delayed.9,29,35,36

When traumatic or spontaneous CCA rupture does occur, the formation of a CCF can occur, and the resulting lesion is usually classified as a Type A, direct, high-flow CCF.16,18,55,64–65 Common clinical manifestations of the presence of a CCF are chemosis, pulsatile exophthalmos, and the presence of an orbital bruit thought to originate from venous reflux into the ophthalmic veins.29 Other associated symptoms related to hemorrhagic mass effect can include, but are not limited to, oculomotor palsy,
ocular pain, and visual impairment. Very rarely, intraparenchymal hemorrhage or ocular ischemia can occur due to steal phenomena occurring as a result of high-flow arteriovenous shunting.\textsuperscript{51,61} Other consequences of CCA rupture can be subarachnoid hemorrhage (2%), thought to occur when the aneurysm, most often large (10–24 mm) or giant (> 25 mm), is projecting intradurally at the time of rupture, or very rarely, epistaxis, which is often a delayed manifestation and is thought to result from progressive osseous erosion of the sphenoid sinus by the aneurysm dome which ruptures directly into the sinus.\textsuperscript{5,9,12,35,56,61,62}

**To Treat or Not to Treat—That is the Question**

The decision to treat CCAs always presents a challenge because treatment implies that the benefits of therapy outweigh the risks of intervention. According to the ISUIA data on CCAs, the rupture risk of asymptomatic lesions < 13 mm in size is ~ 0% rupture risk over 5 years.\textsuperscript{66} Despite these estimates, the ISUIA data do not address symptomatic lesions or CCAs with characteristics that predispose them to increased risk. As such, the decision to treat certain CCAs has to depend on the lesion characteristics and/or the severity of the symptoms. As a result, the strategy of CCA management differs significantly from that of other intracranial aneurysms.

Small (< 12 mm) asymptomatic CCAs do not necessitate immediate therapy, generally being associated with a rather benign natural history and a low risk of life-threatening complications.\textsuperscript{66} However, other characteristics of small asymptomatic CCAs may render these lesions unstable or qualify them for treatment. For example, small CCAs have been reported to increase in size, so periodic monitoring is important.\textsuperscript{32,52} Furthermore, thromboembolic events from developing thrombus and acute thrombosis within small CCAs have been reported in up to 2% of asymptomatic, untreated CCAs.\textsuperscript{5,9,12,35,56,61,62}

Some parameters for treatment of small CCAs may be size independent. Anatomical extension into the subarachnoid space or evidence of significant osseous erosion may render patients at risk for subarachnoid hemorrhage or potentially fatal epistaxis (Fig. 4).\textsuperscript{5,9,12,35,56} Furthermore, CCAs that show evidence of growth, regardless of size, should be considered for treatment, because growth has been reported to increase the risk of rupture.\textsuperscript{32,52} Acute thrombotic and ruptured CCAs are considered unstable lesions and are usually treated expediently, as the symptoms that patients present with are acute and progressive.

However, it is the management of the unruptured CCA, either symptomatic or with threatening anatomical characteristics, that is often debated. Clinical presentation, quality of symptoms, and temporal course of symptoms often dictate whether unruptured CCAs are managed conservatively or invasively. All therapies share the common goal of durable elimination of rupture risk and relief of mass effect symptoms. Common indications for treatment of CCAs are projection of the aneurysm into the subarachnoid space, acute thrombosis, worsening ophthalmoparesis or ophthalmoplegia, intractable or intolerable ocular or retroorbital pain, possibly coagulopathy, and/or increasing aneurysmal enlargement with or without osseous erosion into the surrounding sinuses.

**Treatment and Outcomes**

Treatment paradigms for CCAs include constructive and deconstructive strategies with or without revascularization. Constructive strategies include direct microsurgical clip placement; endovascular coil embolization, with or without the use of intracranial stents or endoluminal balloons; liquid embolization; and parent artery reconstruction without embolization of the aneurysm itself. Deconstructive strategies include Hunterian strategies via parent artery occlusion by means of microsurgical clip placement or endovascular parent artery occlusion with or without revascularization.

**Constructive Strategies**

*Microsurgical Strategies.* The main goal of constructive approaches to the treatment of CCAs is exclusion of the lesion while maintaining patency of the parent artery. One additional indication for open surgical treatment of CCAs is failed endovascular therapy. As with other intracranial aneurysms, direct surgical treatment with clip reconstruction of the parent artery without injury to surrounding vascular structures offers the most direct and
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Fig. 5. Computed tomography (A and B) and digital subtraction (C–E) angiograms showing a CCA before (A–D) and after (E–F) coil embolization. Coronal (A) and axial (B) CT angiograms show a left-sided CCA with projection into the subarachnoid space with significant erosion of the anterior clinoid process (arrows). Left lateral (C and E) and left anterior oblique (D and F) digital subtraction angiographic views of an ICA injection before (C and D) and after (E and F) coil embolization without stent or balloon assistance show complete occlusion.

Endovascular Strategies

Coil Embolization and Intracranial Stent Placement. To avoid the potential complications of microsurgical constructive strategies, constructive endovascular therapies have been developed, with improved deployment and design of intracranial stents, detachable coils, and liquid embolics. Early reported complete CCA occlusion rates ranged from 40 to 60% for lesions treated with coil embolization alone (Fig. 5). Initial poor occlusion rates were reportedly due to insufficient coil packing density and coil compaction as a result of either the preprocedural presence of aneurysmal thrombus or high-inflow impact upon the coil mass. In order to increase coil packing density, balloon-remodeling techniques were developed; in these techniques an intravascular balloon was inflated in the parent artery, covering the ostium of the CCA, while an additional catheter was placed in the aneurysm ostium to deploy coils into the aneurysm with a reduced incidence of coil mass herniation into the parent artery. However, inflation of balloons in the parent artery carries a risk of vessel injury, potentially leading to thromboembolic complications, especially in cases of wide-neck CCAs in which the neck of the aneurysm cannot always be occluded prior to coil embolization. Furthermore, deflation of the balloon can still lead to coil mass herniation due to the absence of a physical barrier.
TABLE 1: Algorithm for determining revascularization procedures in cases in which parent artery occlusion is required*

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Selection Criteria</th>
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<tbody>
<tr>
<td>PAO w/out bypass</td>
<td>BTO*: no evidence of failure to tolerate occlusion; SPECT: no perfusion abnormality</td>
</tr>
<tr>
<td>PAO w/ low-flow bypass (EC-IC)</td>
<td>BTO: no evidence of failure to tolerate occlusion on angiography or clinically during normotensive condition; failure to tolerate occlusion on clinical testing in hypotensive state, w/ or w/ out abnormal EEG changes</td>
</tr>
<tr>
<td>PAO w/ high-flow bypass (vein or radial artery)</td>
<td>BTO: failure to tolerate occlusion in all tests; SPECT: asymmetrical perfusion</td>
</tr>
</tbody>
</table>

* EC-IC = extracranial-intracranial; EEG = electroencephalographic; PAO = parent artery occlusion.
† The following tests were performed during BTO: clinical testing (task performance) with and without hypotension; electroencephalography; angiography. Asymmetry in task performance or nonperformance of requested tasks, asymmetry on electroencephalographic recording, and asymmetry in angiograms were regarded as evidence of failure to tolerate occlusion.

between the coil mass and the parent artery. Despite the findings of a recent meta-analysis of the literature on balloon-assisted coil embolization of CCAs, which demonstrated near-complete occlusion of CCAs in >90% of lesions, with neurological complications occurring in <1% of cases, a recent study on balloon-assisted coil embolization of aneurysms found that balloon assistance does not increase the coil packing density and has a higher rate of complications than coil embolization alone.7

Another constructive endovascular approach to CCA treatment has been the use of intracranial stents. Newer-generation stents have been designed for specific intracranial use, allowing improved navigation through tortuous arterial segments such as the cavernous carotid artery; these stents are self-expandable with a low intrinsic radial force, thus minimizing vascular injury and decreasing the risk of thrombembolic complications.25,43,64 Intracranial stent design has also expanded to include open-cell and closed-cell designs, with potential for certain closed cell designs to be used without coil embolization and in areas with minimal risk of occlusion of important perforating vessels or other vascular structures. Initial results with closed and covered intracranial stents have yielded encouraging results, but further studies are needed. When used in combination with coil embolization, intracranial stents have been shown to facilitate increased coil packing density without the risk of compromising the parent vessel lumen through coil herniation.5 Further, stents have been shown to alter aneurysm hemodynamics by not only diverting flow streams but also reducing the free surface area of the aneurysm neck, which has been shown to reduce intraaneurysm blood flow and thus increase the likelihood of future thrombosis.2,31,39,62 Finally, the mesh of the stent may serve as a growth matrix for neointimal proliferation leading to permanent exclusion of the aneurysm from parent artery flow. Reported risks associated with stent placement include acute in-stent thrombosis, dislodgement or misplacement of the stent, and in-stent stenosis thought to be due to endothelialization. Premedication with antithrombotic agents significantly reduces thrombosis and stenosis. However, concerns remain for CCAs located in the subarachnoid space or within the sphenoid sinuses, where the risk of hemorrhage is significant without occlusion of the lesion proper. Complication rates associated with intracranial stents have been reported to be <10%, with minimal permanent morbidity.25,36,37,43

Although stent-assisted coil embolization of CCAs offers improved coil density at the aneurysm neck/vessel interface, recanalization of aneurysmal lesions due to the attenuation of coil density still frequently occurs. Fortunately, technological advancement in coil design has produced newer hybrid coils that take advantage of the physiological response to different chemical pretreatments in order to increase complete coil occlusion. One approach has been to enhance bioactivity of the coil, via induction of an inflammatory reaction that facilitates accelerated smooth muscle cell migration leading to enhanced thrombus organization and scar retention. Another approach has been to increase the density of coil packing of the aneurysm, via an expandable hydrogel material that results in delayed progressive volumetric expansion of the coils themselves. However, there are no current published studies examining hybrid coil embolization of CCAs.

**Liquid Embolic Agents.** Another development in the endovascular treatment of CCAs has been the increased use of liquid embolic agents. The advantage of using liquid embolics is, theoretically, that the agent completely fills and conforms to the unique geometry of the CCA cavity resulting in complete obliteration.41,65 Further, once polymerized, the mass left behind does not undergo compression or compaction. The difficulty with using such an agent is the great amount of experience that is necessary to appropriately apply liquid embolic agents into aneurysms given the highly fluid nature of the material upon delivery. Balloon-assisted delivery of liquid embolics is associated with the same periprocedural complications as balloon-assisted coil embolization. The rate of total occlusion of CCAs using liquid embolic agents has been reported to be up to 91%, with no reported permanent severe morbidity and no mortality at an average follow-up of 13 months posttreatment.41,65

Unfortunately, there are several disadvantages to endovascular treatment of CCAs. If symptoms were initially caused by mass effect of the CCA, occlusion of the lesion, with either coil embolization or liquid embolic agents,
Cavernous carotid aneurysms will not immediately reduce the mass effect, although some report symptomatic relief thought to be due to the reduction in arterial pulse pressure within the aneurysm. Further, coil and liquid embolization can elicit a localized inflammatory reaction, which can result in acute or delayed worsening of symptoms. Careful patient and lesion selection should always be undertaken to determine which patients are more likely to benefit from endovascular therapy of CCAs.

Other Endovascular Strategies. Challenging anatomical characteristics of CCAs, such as a very acute angle between the aneurysm neck and the parent artery, often make them difficult to treat by means of coil or liquid embolization. Another emerging technique is parent artery reconstruction, which involves the use of a low porosity or 2 or more high porosity intracranial stents placed over the aneurysm neck without embolization of the aneurysm itself. Reduction of intraaneurysm inflow by the struts of the stent (diverting blood flow) increases the incidence of aneurysm thrombosis. Overlapping of multiple stents can only increase the number of flow-diverting structures, thus further decreasing the inflow. Possible complications include increased risk of dissection, movement of previously placed stents, mechanical injury to the stent leading to possible vascular injury, and increased risk of acute or delayed in-stent stenosis. Outcomes of the double-stent technique for CCA treatment have not been widely reported.

Deconstructive Strategies

If preservation of the parent artery is not possible with constructive strategies, deconstructive strategies must be used, namely Hunterian strategies involving occlusion of the ICA.15,26,62 In addition, if inadequate collateral cerebral circulation exists, as determined by BTO, or the hemodynamic burden of ICA occlusion places other cerebral circulation exists, as determined by BTO, or the hemodynamic burden of ICA occlusion places other lesions at risk (for example, contralateral cavernous aneurysms or previous dissections) revascularization may be needed before definitive CCA therapy (Table 1).46 Therefore, 3 types of Hunterian strategies exist: 1) occlusion alone, 2) occlusion with a low-flow bypass (extracranial artery), and 3) occlusion with a high-flow bypass (saphenous vein or radial artery). The determination of the need for revascularization is currently established with a BTO of the ipsilateral ICA. Balloon test occlusion can be used to determine not only the need for revascularization in cases of necessary ICA occlusion, but also the type of bypass needed. The parameters examined during BTO are clinical (age, neurological examination findings, presence of other vascular lesions), angiographic (presence of adequate collateral circulation whether through natural communicating arteries or through anastomotic connections through the extracranial circulation), and hemodynamic (cerebral blood flow, venous transit times). The safety profile of revascularization procedures (for example, extracranial-intracranial bypasses with and without the ELANA [excimer laser-assisted nonocclusive anastomosis] procedure18,27,57) has improved such that Hunterian strategies with revascularization have become an often preferred technique of treatment for CCAs not amenable to direct clip reconstruction. In cases in which microsurgical carotid artery occlusion was performed for CCA treatment, morbidity and mortality rates ranged from 9 to 22%.15,23,28 Endovascular Hunterian strategies to treat CCAs involve parent artery occlusion with detachable balloons or coils. Endovascular occlusion enables shortening of the ICA segment that is occluded and thus reduces the risk of thromboembolic and other associated complications. Furthermore, in cases in which high-flow revascularization is necessary, endovascular ICA occlusion can be performed at the time of the revascularization procedure. Although recanalization of endovascularly occluded ICAs can theoretically occur, it is extremely rare with modern techniques. In large series of CCAs treated with endovascular occlusion, mortality rates of 0–1.7% and permanent morbidity rates of 2.7–6.6% have been reported, significantly lower rates than those reported for open surgical treatment of these lesions.17,18,44,47,49,50,60,67

Expectant Management of Symptomatic CCAs and Outcomes

Despite promising results of CCA treatment, controversy still arises from the fact that the clinical manifestations of symptomatic CCA lesions improve in a very inconsistent fashion, with or without therapy, thus complicating the decision whether to treat symptomatic CCAs. In a large series of CCA patients with significant diplopia and pain, 56% of the ocular symptoms either improved or became unnoticed without invasive therapy.21 However, ocular alignment was a symptom often unnoticed in untreated patients (44%). Therefore, complete assessment of ocular function should precede management so that postprocedural changes can be documented. In addition, of patients that presented with ocular or retroorbital pain whose only treatment was with analgesic agents, 56% experienced improvement without invasive intervention. However, 5% of the patients in the untreated group had significant visual loss due to progressive optic nerve compression, which is an indication for therapy. On the other hand, of patients who were treated for symptomatic CCAs, while 96% reported improved pain control or absence of pain, only 63% reported improvement in ocular symptoms. Cerebral infarcts affected both populations of patients with unruptured CCAs, 4% of treated patients and 2% of untreated patients.21

To Treat or Not To Treat—What Is the Answer?

Aneurysms, by nature, are not static entities, but rather dynamic structures that are likely to change in some respects with time. Despite the CCA’s most often being thought of as a benign and static lesion as well as its being confined to a bony enclosure, which is thought to reduce its rupture risk, there have been many examples of these lesions rupturing, growing, and even eroding the surrounding osseous structures, rendering them aneurysmal lesions that can pose significant risk to the patient. As a result, we evaluate CCAs initially as any other aneurysmal lesion, using a multidisciplinary evaluation, involving other services such as the neuroendovascular service and the neuroradiology service (Fig. 6). The ISUIA data showed that CCAs < 13 mm have a 0% annual risk of

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rupture; however, aneurysm size is not the only indication for treatment. At our institution, other risk factors, such as progressive symptomatology (either psychological or physical), individual history of SAH, significant family history of SAH, aberrant morphological factors (significant bone erosion into the sphenoid sinus or projection into the subarachnoid space), and progressive growth (usually > 1 mm in a 12-month period) may lead to treatment of a CCA that may not otherwise be recommended for therapy according to the literature. When treating CCAs without significant mass effect, our preference is to preserve the parent artery, most preferably by use of endovascular therapy.

The endovascular strategies most often used at our institution are direct coil embolization with or without stents. For lesions that are difficult or unsafe to catheterize for coil embolization, the use of one or more intracranial stents across the aneurysm neck has been shown to decrease the inflow hemodynamics, thus at least reducing the hemodynamic load to the aneurysm. However, in cases of significant aneurysmal mass effect, in which a coil mass may continue to contribute to the mass effect, Hunterian strategies are preferred. Patients undergo BTO in all cases in which direct endovascular strategies are not thought possible and parent artery occlusion may be necessary, be it during microsurgical clipping or with any primary Hunterian strategy. Microsurgical strategies are normally reserved for lesions that project into the subarachnoid space or cases in which endovascular therapy has failed. If patients require a bypass, the type of bypass is determined (see Table 1) and the revascularization procedure is completed. If the patient has other vascular lesions or other anatomical anomalies that render acute parent artery occlusion at high risk of thromboembolic or ischemic complications, we usually wait 2–3 days to perform the parent artery occlusion (normally coil occlusion). Improvement of surgical techniques and safety profiles makes this approach very safe and effective for patients with CCAs not amenable to direct endovascular or microsurgical therapy. Patients who are not treated usually undergo repeat imaging in 6 to 12 months. Any progression of growth, bone erosion, or development of symptoms warrants reevaluation and possible treatment at that time.

**Conclusions**

On the basis of their natural history, most CCAs may be considered benign lesions, presenting a very low risk of rupture and/or life-threatening complications. As a result, asymptomatic CCAs are often conservatively managed with intermittent imaging, unless the anatomical characteristics render the patient at risk for SAH or epistaxis. However, in rare instances, CCAs can be symptomatic, either due to rupture, traumatic or spontaneous, or progressive mass effect. Ruptured lesions should be treated on an emergency basis. Surgical therapy is typically reserved for experienced neurosurgeons, anatomically approachable lesions, and endovascular treatment failures despite being the most durable modality. Endovascular therapy is currently the mainstream of therapy with balloon-assisted and stent-assisted coil embolization being the most championed of modalities. However, improvement in the safety profile of bypass procedures makes Hunterian surgical strategies more attractive due to the durability of parent artery occlusion. Because of the variability of treatment outcomes, treatment of CCAs should be highly individualized. Indications for definitive therapy should be debilitating pain, visual loss from compression or diplopia in primary gaze, risk factors for major complications (such as progressive symptomatology (either psychological or physical), individual history of SAH, significant family history of SAH, aberrant morphological factors (significant bone erosion into the sphenoid sinus or projection into the subarachnoid space), and progressive growth (usually > 1 mm in a 12-month period) may lead to treatment of a CCA that may not otherwise be recommended for therapy according to the literature. When treating CCAs without significant mass effect, our preference is to preserve the parent artery, most preferably by use of endovascular therapy.

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as preexisting coagulopathy), sphenoid sinus erosion, projection into the intradural or sphenoid sinus spaces, aneurysmal growth, acute thrombosis, and acute rupture. Patients with less severe symptoms often experience improvement with conservative therapy, which consists of pain control and physical therapy. Due to the normally benign nature of CCAs, any modality of therapy that is used should have a lower risk profile than the natural history of the condition; however, it is the natural history of CCAs, which present with other, under-represented characteristics that render them at higher risk of rupture, that make the decision for treatment often difficult.

Disclaimer

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