Endosaccular treatment of 113 cavernous carotid artery aneurysms

Armen Choulakian, Doniel Drazin, Michael J Alexander

ABSTRACT
Objective Cavernous carotid aneurysms (CCAs) can present with visual symptoms or with subarachnoid hemorrhage (SAH). As surgical treatment of these aneurysms can be technically challenging, endovascular management has emerged as the preferred treatment modality.

Methods A retrospective review was conducted of 113 patients who underwent endosaccular treatment for CCAs. Presenting symptoms, aneurysm size, use of stent assistance, rate of thromboembolic complications, presence of SAH and angiographic follow-up were reviewed.

Results 29 patients (26%) with CCAs presented with diplopia due to cranial nerve palsies. Mean aneurysm size in this group was 17 mm. Three patients (2.6%) presented with SAH with a mean aneurysm size of 15.3 mm. Mean length of stay for ruptured versus non-ruptured aneurysms was 11.7 and 1.7 days, respectively. Clinically significant thromboembolic complications occurred in four cases (3.5%). Stent assistance was required in 53 cases (47%). Of the 86 patients (76%) returning for follow-up angiography (mean 6.2 months), 58 (75%) had no residual aneurysm and 14 (12%) showed regrowth. Thirteen patients (11.5%) underwent repeat endovascular treatment.

Conclusions CCAs commonly produce diplopia and cranial nerve palsies when a critical size is reached (mean 17 mm in our series). Aneurysm obliteration with internal carotid artery preservation is the preferred treatment modality and can be accomplished with coil embolization with or without stent assistance. Although recurrence and retreatment can occur, the thromboembolic risk of endovascular treatment is low. Consideration should be given to treatment of asymptomatic CCAs 15 mm or larger due to potential risks of cranial neuropathy and SAH.

INTRODUCTION
Cavernous carotid aneurysms (CCAs) account for 2–9% of all intracranial aneurysms.1 The majority of these aneurysms are discovered incidentally with non-invasive imaging during work-up for unrelated intracranial disorders. Occasionally CCAs can become symptomatic either from a mass effect on adjacent structures in the cavernous sinus or from rupture resulting in subarachnoid hemorrhage (SAH) or carotid–cavernous fistula (CCF) formation. Treatment is necessary for symptomatic lesions to prevent potentially permanent cranial neuropathies or re-rupture. Most asymptomatic lesions do not require treatment as they pose a very small risk of severe complications. Larger CCAs, those which demonstrate growth on serial imaging studies, or those which have extended into the maxillary sinus or subarachnoid space, may be considered for treatment before they become symptomatic.2 The minimum size of asymptomatic CCAs for which treatment is recommended has not yet been established. Due to the potential morbidity and technical challenges from surgical approaches to the cavernous sinus, endovascular treatment has become the primary treatment modality for CCAs. Aneurysm obliteration with internal carotid artery (ICA) preservation has emerged as the preferred strategy with the advent of detachable coils and intracranial stents.

METHODS AND MATERIALS
A retrospective review was conducted of 115 patients who underwent endosaccular treatment for CCAs from 2001 to 2010. Patients with deliberate parent artery occlusion were excluded from the analysis. Presenting symptoms, aneurysm size, use of stent assistance, rate of thromboembolic complications, presence of SAH and angiographic follow-up were reviewed.

RESULTS
Twenty-nine patients (26%) with CCAs presented with diplopia due to cranial nerve palsies. Mean aneurysm size in this group was 17 mm. Three patients (2.6%) presented with SAH with a mean aneurysm size of 15.3 mm. Mean length of stay for ruptured versus non-ruptured aneurysms was 11.7 and 1.7 days, respectively. Clinically significant thromboembolic complications occurred in four cases (3.5%). Stent assistance was required in 53 cases (47%). Of the 86 patients (76%) returning for follow-up angiography (mean 6.2 months), 58 (75%) had no residual aneurysm and 14 (12%) showed regrowth. Thirteen patients (11.5%) underwent repeat endovascular treatment for either significant residual aneurysm or aneurysm regrowth. Representative cases are shown in figures 1–3.

DISCUSSION
CCAs can be found incidentally on neuroimaging or can become symptomatic in a variety of ways, including rupture. Although rare, CCAs can present with rupture into either the cavernous sinus or subarachnoid space. Direct CCFs can result if a CCA ruptures into the cavernous sinus. In one study, the incidence of CCF by a ruptured CCA was 1.5%.3 If a CCA erodes through the sphenoid bone into the maxillary sinus, rupture may result in catastrophic epistaxis. Extension of a CCA into the subarachnoid space, occasionally seen as ‘dural waisting’ on cerebral angiography, can predispose to SAH.4 The risk of SAH from CCAs is relative to
size, with aneurysms <12 mm having a 0% rupture risk over 5 years while those >25 mm have a 6.4% rupture risk over the same time period. CCAs 13–24 mm in size have a 5 year rupture risk of 3.0%. In our series, three patients with CCAs presented with SAH and had no other aneurysms identified on digital subtraction angiography. In all three cases the aneurysm neck began in the cavernous carotid artery but the aneurysm was transitional into the subarachnoid space.

More commonly, CCAs present with either pain or visual symptoms, especially when they reach a giant size. Pain is usually facial or retro-orbital in location but generalized headache can also be reported. Diplopia is the most common visual symptom and results from paresis of the third, fourth or sixth cranial nerves. Involvement of any of the divisions of the fifth cranial nerve can result in analgesia or hypalgesia in that territory. Any combination of cranial nerves in the cavernous sinus can become affected and the degree of symptoms is highly variable.

Symptomatic CCAs, specifically those which have ruptured or are causing cranial neuropathies, have traditionally undergone treatment to prevent rebleeding or permanent oculoparesis and vision loss. Multiple studies have found better outcomes in improvement or stabilization of pain symptoms and neuro-ophthalmologic deficits in patients with CCAs who receive treatment versus those who do not. One study however found that treatment had a significant effect only on patients presenting with pain but not on patients presenting with diplopia. Follow-up visual analysis was not included in our study as only 86 patients (76%) had descriptions of their visual symptoms after treatment at angiographic follow-up. Inclusion of data from this select group of patients would not be an accurate reflection of the 113 aneurysms treated in this series, as it is perceived that patients with recurrent or residual symptoms are more likely to follow-up, presenting a selection bias.

Traditionally, asymptomatic lesions, especially when small, have not been treated as they are of low risk to the patient. However, consideration can be given to treatment of large asymptomatic CCAs in the hope of preventing future symptoms, because as these aneurysms enlarge they can produce a mass effect on structures of the cavernous sinus and

**Figure 1** Patient No 1 presented with headaches. (A) Pre-procedure right internal carotid artery (ICA) angiogram demonstrated a 14 mm cavernous carotid artery aneurysm. The patient underwent coil embolization without stent assistance. (B) Post-procedure right ICA angiogram demonstrated complete occlusion of the aneurysm.

**Figure 2** Patient No 2 presented with a 2 month history of right third and sixth nerve palsies. (A) Pre-procedure right internal carotid artery (ICA) angiogram demonstrated a 20 mm cavernous carotid artery aneurysm. The patient underwent coil embolization with Neuroform (Boston Scientific, Natick, Massachusetts, USA) stent assistance. (B) Post-procedure right ICA angiogram demonstrated a good result.
Endovascular sacriﬁce is at times necessary, aneurysm obliteration with ICA preservation is the preferred treatment modality and can be accomplished with coil occlusion of the ICA followed by embolization of the aneurysm. This is currently our preferred method of treatment for large wide necked CCAs with nearly half of the cases in our series requiring stent assistance (47%). Emerging endovascular treatment options for these lesions include easily navigateable covered stent grafts, stand alone flow diverters like the Pipeline Embolization Device (ev3, Irvine, California, USA), and liquid embolic agents such as Onyx (ev3).

The rates of incomplete treatment, recurrence and retreatment are high when treating large CCAs via the endovascular route with ICA preservation. Speciﬁcally, we had a 25% rate of residual aneurysm on follow-up imaging with 12% of patients showing regrowth and 11.5% being signiﬁcant enough to require retreatment. However, the risk of endovascular treatment of CCAs is low, with only 3.5% clinically signiﬁcant thromboembolic events occurring in our series.

**CONCLUSION**

CCAs commonly produce diplopia and cranial nerve palsies when a critical size is reached (mean 17 mm in our series). Aneurysm obliteration with ICA preservation is the preferred treatment modality and can be accomplished with coil occlusion of the ICA followed by embolization of the aneurysm. This is currently our preferred method of treatment for large wide necked CCAs with nearly half of the cases in our series requiring stent assistance (47%). Emerging endovascular treatment options for these lesions include easily navigateable covered stent grafts, stand alone flow diverters like the Pipeline Embolization Device (ev3, Irvine, California, USA), and liquid embolic agents such as Onyx (ev3).

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embolization with or without stent assistance. Although recurrence and retreatment can occur, the thromboembolic risk of endovascular treatment is low. Consideration should be given to treatment of asymptomatic CCAs 15 mm or larger, or those that demonstrate progressive rapid growth due to potential risks of cranial neuropathy and SAH.

Competing interests MJA is a consultant for Boston Scientific (Natick, Massachusetts, USA) and Codman Neurovascular (Raynham, Massachusetts, USA). AC is the recipient of Boston Scientific and Cordis Endovascular (Bridgewater, New Jersey, USA) fellowship training grants.

Patient consent Detail has been removed from this case description to ensure anonymity. The editors and reviewers have seen the detailed information available and are satisfied that the information backs up the case the authors are making.

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