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Improvement in Visual Symptomatology after Endovascular Treatment of Cavernous Carotid Aneurysms:

A Multicenter Study

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Abstract

Introduction: Aneurysms arising from the cavernous internal carotid artery (CCAs) pose technical challenges for surgical management and such patients are frequently referred for endovascular treatment. These aneurysms often produce a variety of neurological deficits, primarily those related to oculoparesis. Our purpose was to determine the visual and neurological outcome of patients with treated CCAs.

Methods: We reviewed the medical records and angiograms for patients who underwent endovascular treatment for CCAs at three academic medical centers. The following outcomes were analyzed: angiographic assessment, visual improvement and outcome at 3 months using Glasgow Outcome Scale (GOS).

Results: Thirty-four patients (mean age 54.7 years) were treated for CCAs. The mean aneurysm size was 14.2 mm (range: 3-45 mm), and fourteen patients (41.2%) required stent assistance. Twenty-one aneurysms (61.8%) were completely occluded; nine aneurysms (26.6%) had near-complete occlusion; 4 aneurysms (11.8%) had partial occlusion. Seven patients (20.6%) required retreatment. Fifteen of the 34 patients (44.1%) presented with visual symptoms, while only eight patients had residual visual symptomatology at follow-up (44.1% vs. 23.5%; p=0.02). Patients that presented with visual symptoms (N=15) had a mean aneurysm size of 24.5 mm, while those without visual symptoms (N=19) had a size of 7.5 mm (p=0.001). Follow-up GOS was good (4-5) in 29 patients (90.6%). No thromboembolic complications were observed. One patient died (3.1%) of an unrelated cause.

Conclusions: Most patients in this multicenter series improved or remained stable after treatment. The results of this study indicate that endovascular treatment may improve the outcome of visual symptoms in patients with large cavernous aneurysms with low periprocedural morbidity.

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Table 1.

Demographic and clinical characteristics of cavernous carotid aneurysm patients.

Characteristics	N (%)
Total number of cases	34
Patient age in years	
mean (SD)	54.7 (16.1)
Female	29 (85.3)
Sympathology	
3rd N Palsy	3 (8.8)
6th N Palsy	3 (8.8)
Blurry Vision	1 (2.9)
Diplopia	2 (5.9)
HA	9 (26.5)
Incidental	2 (5.9)
Orbital Pain	3 (8.8)
Other	9 (26.5)
Vision Loss	1 (2.9)
Visual TIA	1 (2.9)
Glasgow Outcome Scale *Baseline: Follow-up*	
Good	30 (88.2): 29 (90.6)
Moderate	2 (5.9): 2 (6.3)
Severe	1 (2.9): 0 (0)
Died	1 (2.9): 1 (3.1)

Keywords

Cavernous Carotid Aneurysm (CCA); Endovascular treatment; Glasgow Outcome Scale (GOS); Raymond Royd (RR) Grade; Visual Sympathology

Introduction

The cavernous segment of the internal carotid artery (ICA) is a well known site of aneurysm formation with 2-12% of unruptured aneurysms found at this location.[1] Due to their extradural origin, subarachnoid hemorrhage is an uncommon presentation for these lesions; however, in large and giant aneurysms at this location, the 5 year risk of SAH is 3 and 6% respectively.[2-4] Rarely, an aneurysm arising from the cavernous segment will present with symptoms of a carotid-cavernous fistula or epistaxis after rupture. More common presentations include incidental diagnosis and mass effect on surrounding structures. Asymptomatic lesions are generally observed unless they appear to be expanding, causing extensive bony erosion, or have an intradural component[3]. Mass effect causing lesions most commonly present with headache and diplopia caused by compression of the oculomotor nerve (CN III), trochlear nerve (CN IV), or abducens nerve (CN VI). Lesions requiring treatment have been approached in a number of ways that fall into two broad categories: vessel preserving strategies (direct clipping, coiling, stent-assisted coiling, emoblization with liquid embolic agents, and flow-diverters) and vessel sacrifice (open surgical or endovascular parent vessel sacrifice with or without adjunctive bypass surgery). Older series have shown similar success rates between these two approaches, however, endovascular treatment has evolved at a rapid rate with greater dependence on stent-assisted coiling, use of liquid embolic agents, and most recently the use of flow-diverter technology.[3] These advances have increased the number of patients in whom vessel preservation is possible. This article reviews the experience at three institutions with modern endovascular techniques and their effect on patient outcome.

Methods

All patients with cavernous carotid aneurysms (CCAs) who underwent endovascular treatment between 2006 and 2009 were retrospective identified at three institutions: Cedars-Sinai Medical Center, Albany Medical Center, and Saint Louis University Hospital. CCAs was defined as an aneurysm originating between the petrocavernous junction and the proximal dural ring. Aneurysms originating from the petrous carotid, carotid cave, clinoidal or paraophthalmic segments were excluded.

The medical records and catheter angiograms were reviewed and data was collected on patient demographics, treatment indications, aneurysm characteristics, treatment modality selected, and one year clinical outcome. Clinical outcome was measured using the Glasgow Outcome Scale (GOS) and qualitative assessment of visual symptoms.

Statistical Methods

Analysis of the data collected from this study was primarily descriptive; simple descriptive statistics such as

Table 2.
Aneurysm Treatment Characterization.

Characteristics	*n* (%)
Aneurysm size, mm	
mean (SD)	14.2 (10.6)
[min, max]	[3, 45]
Aneurysm Location	
Cavernous	25 (73.5)
Cav-Paraclin	6 (17.6)
Petro-Cav	3 (8.8)
Cases	
ruptured	6 (17.6)
stented	14 (41.2)
rebled	1 (2.9)
retreated	7 (20.6)
Occlusion status	
complete	21 (61.8)
near complete	9 (26.5)
partial	4 (11.8)
Raymong & Royd Classification	
Class 1	21 (61.8)
Class 2	9 (26.5)
Class 3	4 (11.8)

mean, standard deviation, median, interquartile range (data mid-spread), and percents were used to summarize the data. The McNemar's test was use to compare visual improvement from baseline and follow-up post intervention. Non-parametric Kruskal Wallis test was used to assess continuous and categorical outcomes.

Results

Patient Characteristics

A summary of demographic and clinical characteristics in provided in Table 1. Thirty-four patients (mean age 54.7) were treated for CCAs; the majority of patients were females (85.3%). The initial GOS was good for 88.2% of patients, moderate for 5.9% and severe-death for approximately 2.9% of patients. At follow-up the proportion of patients that had good recovery increased to 90.6%.

Fifteen of the 34 patients presented with visual symptoms, while eight of those patients had residual visual symptomatology at follow-up (44.1% vs. 23.5%; p=0.05). Patients that presented with visual symptoms had a mean aneurysm size of 24.5mm, while those without visual symptoms had a size of 7.5mm (p=0.001). Follow-up GOS was good (4-5) in 29 patients (90.6%). No thromboembolic complications were observed. One patient died (2.9%) of an unrelated cause.

Aneurysm Characteristics

The average aneurysm size was 14.2 mm (range: 3-45mm) and cavernous (73.5%) was the most common location (Table 2). Among the 34 cases, 17.6% ruptured, 41.2% required stent assistance, 2.9% rebled, and 20.6% were retreated. Twenty-one aneurysms (61.8%) were completely occluded; nine aneurysms (26.5%) had near-complete occlusion; 4 aneurysms (11.8%) had partial-occlusion. Angiographic evidence of aneurysm dome or neck filling was assessed with the Raymond Roy classification system (Table 2). No filling of the aneurysm neck or dome was observed in 61.8% (Class 1), residual filling of the neck but not the dome was found in 26.5% (Class 2), and residual filling of the neck and dome (Class 3) was identified in 11.8% of the cases.

Patient 1 presented with a three month history of diplopia and an incomplete right 3rd CN palsy. Cerebral angiogram demonstrated a 16mm x 12mm right cavernous carotid aneurysm with a 12mm neck (Figure 1A,B). Patient underwent Neuroform (Stryker, Natick MA) stent assisted coiling of the aneurysm with near-complete occlusion and without any complications (Figure 1C,D).

At one week follow-up the diplopia had began to improve but follow up angiogram at three months showed significant recurrence of the aneurysm secondary to coil compaction. Patient underwent repeat stent assisted coiling of the aneurysm using an Enterprise (Codman Neurovascular, Raynham, MA) stent with only a small amount of residual filling at the neck (Figure 1 E, F). At six month follow-up the diplopia had resolved and the right 3rd CN palsy had improved to only a mild ptosis. Angiogram at that time showed no recurrence of the aneurysm.

Patient 2 had a history of hypertension, coronary artery disease, and hypercholesterolemia who presented with approximately one year of worsening double vision. Additionally, patient described a band of decreased sensation and tingling involving the forehead that had also

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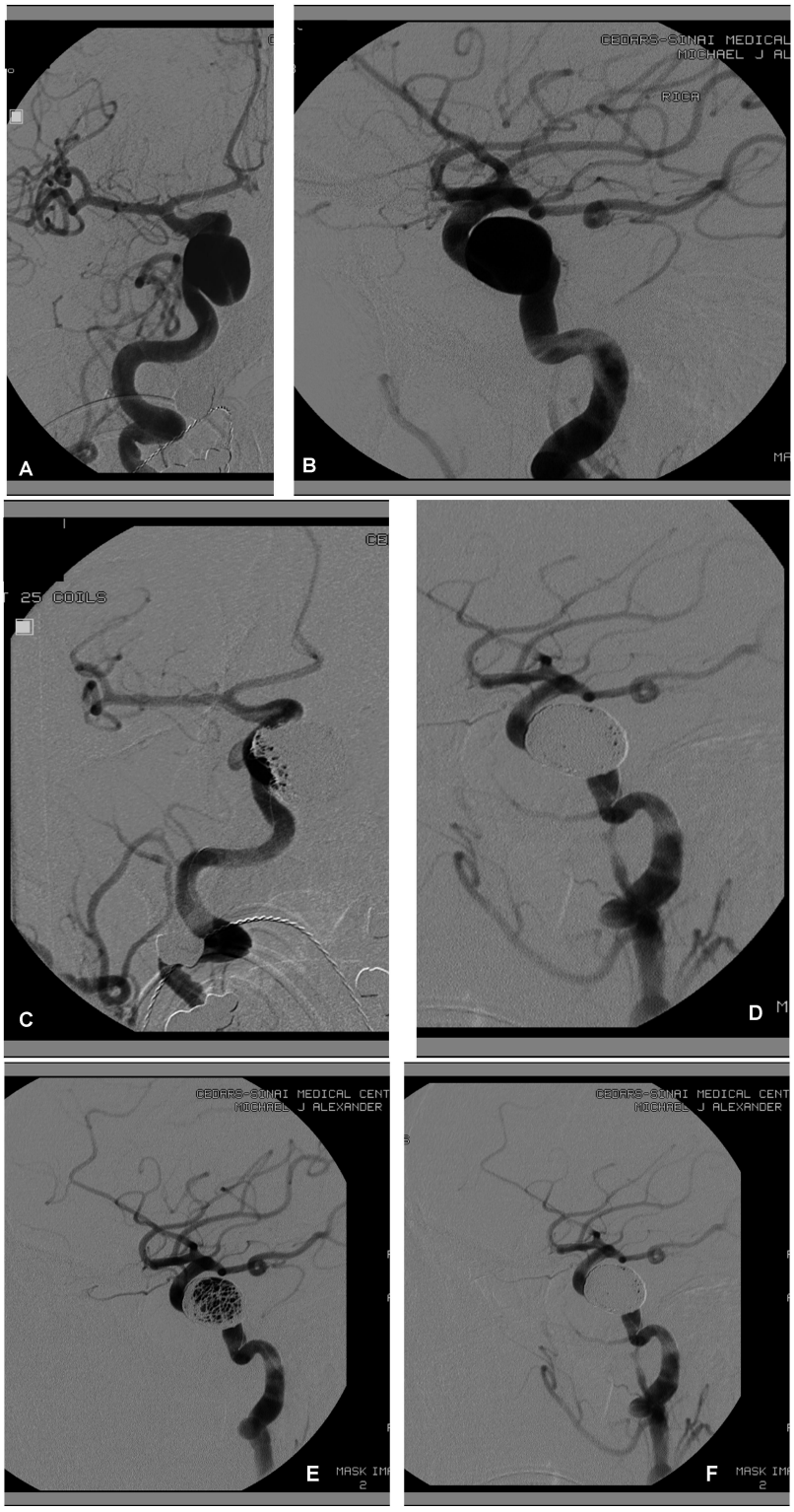

Figure 1. (A) AP and (B) lateral pre-treatment right ICA angiogram demonstrating a 16mm x 12mm right CCA with a 12mm neck. (C) AP and (D) lateral post-treatment right ICA angiogram demonstrating near complete occlusion of the aneurysm after Neuroform stent assisted coiling. (E) Lateral right ICA follow-up angiogram at three months showing significant recurrence of the aneurysm secondary to coil compaction. (F) Lateral right ICA angiogram after repeat treatment using Enterprise sent assisted coiling. Only a small amount of residual neck filling remains.

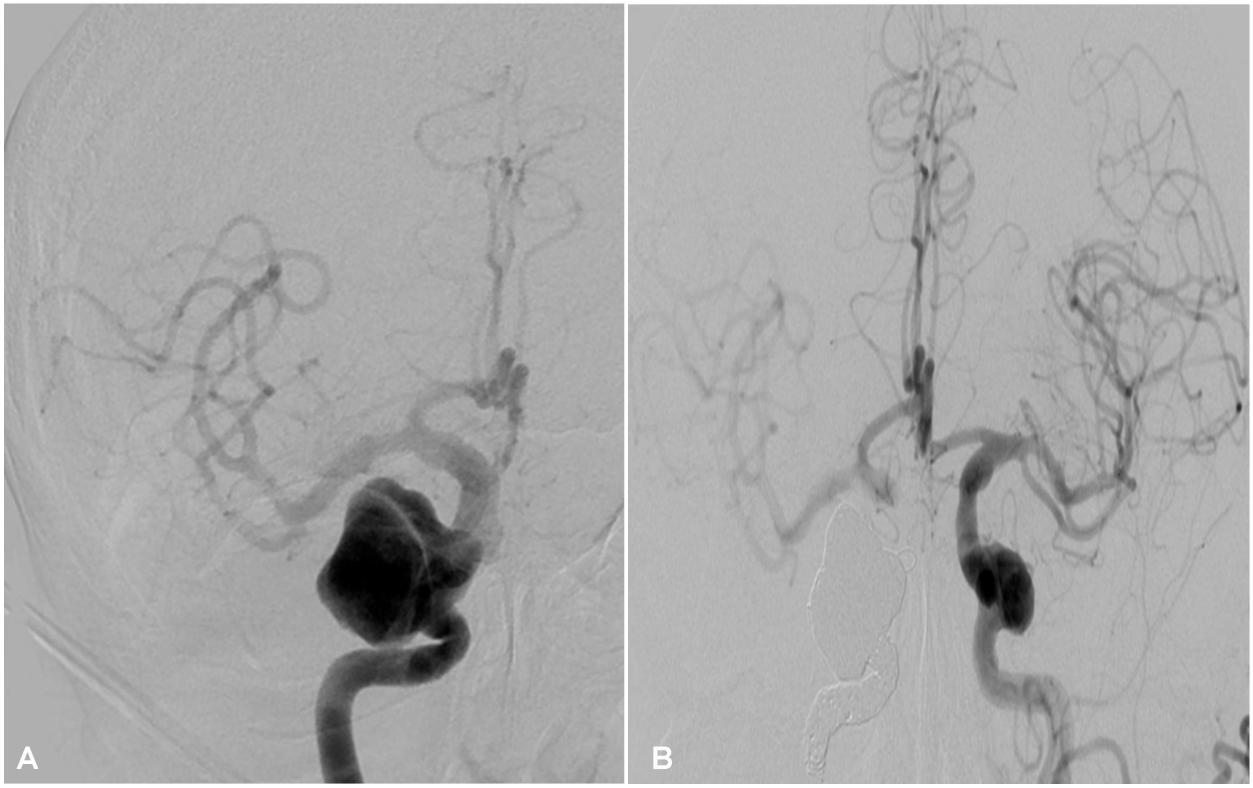

Figure 2. (A) AP pre-treatment right ICA angiogram demonstrating a 25mm CCA. (B) AP post-treatment left ICA angiogram showing filling of the contralateral ACA and MCA distributions after endovascular coil sacrifice of the right ICA and aneurysm.

become progressively worse over the prior year. Finally, the patient noted tinnitus which involved both ears and was constant. Examination revealed impairment of extraocular movements on the right with partial ptosis. In addition there was a band of decreased sensation the V1 distribution. Cerebral angiogram demonstrated a 25mm right CCA (Figure 2A).

Patient initially underwent diagnostic angiography along with temporary balloon test occlusion (BTO). A hypotensive challenge confirmed the adequacy of his collateral supply to the right hemisphere. The aneurysm and parent vessel were them occluded using coiling embolization (Figure 2B). Symptoms improved over the next 24 months with cessation of the tinnitus and a return to near normal vision.

Discussion

CCAs can be found incidentally on neuroimaging or can become symptomatic in a variety of ways. Although rare, CCAs can present with rupture into one of three spaces. Rupture into the cavernous sinus can lead to the formation of direct carotid-cavernous fistulae, rupture through eroded sphenoid bone into the sphenoid sinus can cause severe epistaxis, and rupture from intradural extension can cause SAH [3]. The risk of SAH from CCAs is relative to size with aneurysms < 12mm having a 0% rupture risk over five years while those > 25mm have a 6.4% rupture risk over the same time period. CCAs 13-24mm in size have a five year rupture risk of 3.0%.[4]

More commonly, these aneurysms present with either pain or visual symptoms, especially when they reach a giant size. Pain is usually facial or retroorbital in location, however 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.[3] The symptoms most likely to improve were mild sixth nerve palsies or third nerve palsies that had been present for a relatively short duration (i.e. diplopia 2 months or less).

The size and orientation of CCAs are important factors concerning symptomatology. As the aneurysms grow in

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size, they can compress structures of the cavernous sinus or erode through structures surrounding the sinus. Erosion medially into the sphenoid sinus can lead to fatal epistaxis if the aneurysm ruptures. Extension superiorly through the dural confines of the cavernous sinus puts the aneurysm at risk for causing SAH. The sixth cranial nerve, located in the center of the cavernous sinus, and the third, fourth, and fifth cranial nerves, located in the lateral wall of the sinus, can become compressed when CCAs reach a certain size.[1,3] In this multicenter series the mean aneurysm size was 14.2mm and the mean size of aneurysms presenting with visual symptoms was 24.5mm compared to 7.5mm for those without visual symptoms.

Treatment of CCAs is reserved for symptomatic lesions; either those which have ruptured or those which are causing pain or visual symptoms. Treatment of large asymptomatic lesions or smaller growing lesions is less common but may be taken into consideration to prevent symptoms from occurring.[5] The treatment of small stable lesions is not recommended as these aneurysms are unlikely to become symptomatic.[6]

Surgical approaches for direct repair of CCAs are technically challenging, especially for larger aneurysms, and carry with them a high risk of cranial nerve morbidity. For these reasons endovascular approaches have become the primary treatment modality for CCAs. If a patient successfully passes a BTO, the ICA can be endovascularly sacrificed proximal and distal to the aneurysm to exclude it from the circulation. Initially detachable balloons were used for this purpose but have now been replaced by detachable coils. If a patient fails a BTO, then extracranial-intracranial bypass followed by either surgical or endovascular carotid trapping can be performed to treat the aneurysm and maintain blood flow to the hemisphere.[7,8]

Endovascular obliteration of CCAs with preservation of the ICA is the most desirable outcome. This can be accomplished in smaller aneurysms with narrow necks with detachable coils alone.[9] Unfortunately many of the aneurysms that require treatment are large and can have extremely wide necks; sometimes even encompassing the entire ICA. Balloon remodeling of the aneurysm neck can be used in the treatment of some wide necked CCAs but is of limited use when the aneurysm is fusiform or has no discernable neck. With the addition of neurovascular stents to the neurointerventional armamentarium, even these extremely challenging lesions can be treated with ICA preservation. Stent assisted coiling, either with the open cell Neuroform or closed cell Enterprise stent, allows reconstruction of the ICA wall 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 (41.2%). Emerging endovascular treatment options for these lesions include easily navigateable covered stent grafts, stand alone flow diverters like the Pipeline Embolization Device (ev3, Irvine, CA), and liquid embolic agents such as Onyx (ev3, Irvine, CA).

Symptoms, either pain or diplopia (with or without ophthalmoplegia) from large CCAs can resolve, incompletely improve, or at least have their progression haulted following endovascular treatment.[8-10] With coil embolization the volume of the aneurysm is filled with coils and thrombus, but the compression of the cavernous sinus structures remains. Despite the presence of this compressing mass, symptoms can improve because the mass is no longer pulsatile. In our series 15 patients presented with visual symptoms. Seven patients had resolution of their symptoms after treatment while the remaining eight patients showed improvement but with some residual symptoms. No patients had progression or worsening of their symptoms after treatment. Historically, giant aneurysms may have transient worsening of their pre-existing cranial neuropathy. However, we believe that the use of peri-procedural steroids has reduced the incidence of transient CN palsy worsening.

Even with stent assisted coiling, complete occlusion of large CCAs is not always possible. We were able to achieve complete occlusion in 61.8% of our cases with near-complete occlusion in another 26.5%. Seven (20.6%) patients required retreatment on follow-up due to significant aneurysm recurrence. No procedure related complications were encountered and all but one patient, who died from an unrelated cause, had favorable outcomes (GOS 4-5) on follow-up. Given the improvement in patients with visual symptoms and the low periprocedural morbidity, endovascular embolization of CCAs is a valid treatment option despite the potential for incomplete occlusion and high retreatment rates.

Conclusions

Most patients in this multicenter series improved or remained stable after treatment. The results of this study indicated that endovascular treatment may improve the outcome of visual symptoms in patients with large CCAs with low periprocedural morbidity. Larger multicenter series are needed to better determine the long term effectiveness of endovascular treatment of CCAs.

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