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Resolution of Symptoms after Parent Artery Occlusion Treatment for Giant Cavernous Carotid Artery Aneurysms

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Abstract

Background and Objective: Giant cavernous carotid artery aneurysms (CCAAs) often produce a variety of neurological deficits, primarily those related to ophthalmoplegia/paresis and headache. This study was designed to evaluate the resolution of symptoms after parent artery occlusion (PAO) treatment for giant CCAAs. Methods: We retrospectively reviewed a series of 17 consecutive giant CCAAs treated with PAO treatment. All patients were evaluated by balloon occlusion test (BOT) before treatment. Patients who could tolerate BOT were treated by PAO. The following outcomes were analyzed: angiographic assessment, evolution of symptoms and outcome at clinical follow-up using modified Rankin Scale (mRS). Results: A total number of 17 giant CCAAs were treated by PAO. The initial post-procedure and follow-up angiogram revealed complete occlusion in all patients, no new lesion was detected. Periprocedural infarcts occurred in 1 patient (5.9%). Procedure-related mortality and morbidity were 0% and 5.9%, respectively. At mean 31.8 months clinical follow-up, symptoms had disappeared in 7 (41.2%) of the patients, partially improved in 5 (29.4%), remained unchanged in 4 (23.5%) and worsened in 1 (5.9%) of cases. Sixteen (94.1%) patients presented a good clinical outcome (mRS 0 - 1). Conclusion: Most patients in our series improved or remained stable after PAO. The results of this study indicate that PAO can improve the outcome of those symptomatic giant CCAAs if BOT can be tolerated.

Keywords

Giant, Cavernous Carotid Artery, Aneurysms, Parent Artery Occlusion

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1. Introduction

Giant CCAAs are defined as larger than 25 mm and they most often produce mass effects [1]. Such effects are likely due to compression of the adjacent third to sixth cranial nerves which can result in symptoms such as headache, ophthalmoplegia or facial pain. Surgical treatment of these aneurysms is challenging due to their location often involving the cavernous sinus region and the dysplastic characteristic of the parent artery in case of dissecting lesions [2]. Despite endovascular remodeling techniques, Stent/coil endovascular embolization is usually ineffective, with a high recanalization rate. Flow-diverter stents appear to be a promising tool; however, the risks and the outcomes of these new treatments are not well known [3] [4]. PAO is a well-established technique for the treatment of giant CCAAs if BOT can be tolerated [5] [6], however, its efficacy concerning resolution of symptoms has not yet been adequately assessed, which to our knowledge, is likely due to the limited number of patients included in studies that have been reported in the literature. The purpose of our study was to evaluate the evolution of symptoms after PAO treatment for giant CCAAs in this series of patients.

2. Material and Methods

2.1. Ethics Statement

Approval of this study was granted by the Institutional Review Board at our Hospital. All patients gave written informed consent to participate and the privacy of patients was strictly protected.

2.2. Patients

Seventeen patients with giant CCAAs who underwent PAO treatment between January 2007 and September 2012 were enrolled in our center. CCAA 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. Clinical and radiological records were, retrospectively, reviewed by two neuroradiologists in our hospital. The medical records and catheter angiograms were reviewed and data was collected on patient demographics, evolution of symptoms and clinical follow-up. Clinical outcome was measured using the modified Rankin Scale (mRS).

2.3. Balloon Occlusion Test (BOT)

The decision to undertake PAO for the treatment of giant CCAAs was assessed by BOT before treatment. Before BOT, all patients had a complete neurological examination, including oculomotor testing, performed by the neurosurgeons or neurologists and the interventional neuroradiologist. Balloon occlusion test was performed under neuroleptic anesthesia to allow clinical neurologic examination. Under fluoroscopy, the HyperGlide balloon (ev3, Micro Therapeeutics. Inc, USA) was introduced into the target carotid artery through a 6 French Envoy (Cordis Neurovascular, Inc.) guiding catheter via right percutaneous femoral artery. Temporary complete occlusion of the internal carotid artery was achieved during thirty minutes with continuous monitoring of the clinical neurological function, including language, movement, sensation and consciousness. During the temporary internal carotid occlusion, contralateral carotid artery and bilateral vertebral artery angiography were performed during the balloon occlusion test with a 5F diagnostic catheter via left femoral artery. If any neurological deficit came out, the test was immediately stopped and the balloon deflated. The cross filling of the arteries were also examined and special attention was paid to the simultaneous or delayed filling of the veins in the occluded side. In all our cases, the anterior communicating artery and the biolateral posterior communicating arteries were open, and the BOT showed sufficient countercurrent flow to the target artery through the posterior communicating artery and anterior communicating artery, so parent artery occlusion with endovascular treatment was feasible without the need for high flow by-pass.

2.4. Parent Artery Occlusion (PAO)

The performance of PAO was based on clinical (no neurologic deficits) and angiographic (delay in cortical venous drainage less than 3 seconds between occluded and contralateral hemispheres) followed by BOT [7]. PAO was conducted with the patient under general anesthesia. Activated clotting time was maintained between 250 and 300 seconds by systemic anticoagulation with heparin. PAO was performed by coil embolization alone

in 8/17 cases (40.1%) and using both detachable balloons (Goldbal; Balt Extrusion, Montmorency, France) and coils [GDC coils (Boston Scientific, Natick, Massachusetts, USA), Trufill DCS coils (Cordis, Miami Lakes, Florida, USA), MicroPlex coils (MicroVention, Aliso Viejo, California, USA), and HydroCoils (MicroVention)] in 2/17 cases (11.8%). Occlusion was performed with detachable balloons alone in 5/17 cases (29.4%), coils with additional glue (Onyx-18 [MTI-EV3, Irvine, CA, USA]) was used in 2/17 (11.8%) cases. For permanent ICA occlusion using detachable balloons, a first balloon was detached in close proximity to the aneurysm, followed by a second, more proximally situated "safety" balloon. Systolic blood pressure was maintained above 140 mmHg during the two days after the treatment.

2.5. Clinical and Angiographic Follow-Up

After the treatment by PAO, clinical and imaging follow-up were available in all patients. The mean length of clinical and angiographic follow-up period was 31.8 months (with a range of 8 to 57 months). The clinical follow-up was classified by Modified Rankin Scale (mRS) scores. Clinical FU included neurological examination with oculomotor testing and/or visual field exams. The degree of the aneurysm occlusion was classified as: complete obliteration; neck remnant; incomplete occlusion.

3. Results

3.1. Patient Characteristics

A summary of demographic and clinical characteristics in provided in **Table 1**. Seventeen aneurysms were treated by PAO (8 with coils, 5 with balloons, 2 with balloon and coils, 2 with coils and onyx) were included in this study. There was a female predominance with 13 patients (76.5%); 4 patients were male (23.5%). The median age was 44 years (range, 20 - 61 years). Twelve patients presented with cranial neuropathies, 3 patients presented with headache, and 2 patients were asymptomatic (detected incidentally during neuroradiological imaging because of an unrelated medical condition). The median aneurysm size was 32.3 mm, ranging from 25 to 45 mm.

Table 1. Patient demographics and aneurysm characteristics.

Patient	Age	Sex	Size (mm)	Presentation	Treatment summary	Final symptoms	mRS	Follow up (month)
1	55	F	25 × 22	Headache, ophthalmoparesis, diplopia	Coil	Headache	1	57
2	52	F	36 × 28	Diplopia, headache, ophthalmoparesis	Coil	Headache	1	57
3	54	F	27 × 19	Headache, diplopia, ophthalmoparesis	Coil	None	0	53
4	51	F	30 × 26	Diplopia	Balloon	Diplopia	1	47
5	20	F	35 × 35	Diplopia, headache, visual blurring	Balloon	None	0	45
6	53	F	30 × 20	Diplopia	Balloon	Diplopia	1	35
7	25	M	28 × 28	Asymptomatic	Coil	None	0	35
8	61	F	34 × 24	Headache, diplopia	Coil /balloon	hemiparesis	2	33
9	38	F	38 × 31	Headache	Coil/Onyx	Headache	1	33
10	36	F	29 × 23	Headache	Coil/Onyx	None	0	28
11	38	F	45 × 45	Asymptomatic	Coil	None	0	27
12	36	F	34 × 28	Headache	Coil	None	0	24
13	24	M	27 × 22	Visual blurring	Coil	Headache	1	21
14	58	F	32 × 29	Diplopia, headache	Coil/ balloon	Headache	1	17
15	58	F	30 × 20	Diplopia	Coil	None	0	13
16	46	M	25 × 13	Diplopia	Balloon	Diplopia	1	8
17	43	M	44 × 25	Headache, diplopia	Balloon	None	0	8

3.2. Clinical and Angiographic Results

Of those that were endovascularly treated, a group of 17 giant CCAAs, the initial post-procedure angiograms revealed complete occlusion. Periprocedural infarcts occurred in 1 patient as a result of hemodynamic insufficiency after PAO. She is a 61-year-old nonhypertensive, diabetic woman, developed an ipsilateral cerebral infarct associated with a right hemiparesis and expressive dysphasia after the procedure. MRI performed immediately after the procedure showed a small ischemic lesion in the left cerebral cortex. Three months after the treatment, neurological improvement was observed, despite a slight hemiparesis was retained (mRS = 2). At 3 - 36 months follow-up, angiograms revealed complete occlusion in all 17 patients. No *de novo* flow-related aneurysm due to PAO was observed in our series. No stenosis/occlusion of the remaining cervical vessels was observed on imaging follow-up. Of the 17 giant CCAAs, symptoms were resolved in 7 (41.2%), improved in 5 (29.4%), remained unchanged in 4 (23.5%), worsened in 1 (5.9%) at 31.8 months clinical follow up. Patients presented with unchanged symptoms were all treated more than 1 month when symptoms onset. A good clinical outcome (mRS 0 - 1) was observed in 16 patients (94.1%), while a patient who was performed with coils and balloon, was observed in slight hemiparesis (mRS 2). Procedure-related morbidity and mortality was 5.9% and none, respectively.

4. Discussion

CCAAs account for 2% to 9% of all intracranial aneurysms and form a distinct group [8]. They are considered benign lesions, often asymptomatic, and to have a natural history with a low risk of life-threatening complications. When they reach a giant size, however, these aneurysms more commonly present with either ocular symptoms or headache. The pathophysiological mechanisms of giant CCAAs development remain a matter of speculation. A rupture risk of giant aneurysms was 6.4% in the cavernous segment of the carotid artery over a 5-year period, documented by The International Study of Unruptured Intracranial Aneurysms [9]. Several mechanisms have been described concerning the pathophysiology of cranial nerve palsy, such as pulsatility exhibited by aneurysmal sac, direct compression by an enlarged aneurysmal sac within the arachnoid cisterns, Compression of the pain sensory afferent fibers [10]. Giant aneurysms of the cavernous sinus may lead to localized oculomotor or abducent nerve dysfunction and Horner syndrome [11].

Management of these lesions is a daunting task for the physician. Lv *et al*. reported eight giant CCAAs managed conservatively (6 - 65 months, median 33.6 months), symptoms were worsened in four patients (50%), remained unchanged in three, improved in one [12]. Linskey *et al*. observed 20 CCAAs without intervention (5 months - 13 years, median 2.4 years): symptoms were worsened in seven (35%), unchanged in nine (45%), and improved in four (20%) [13]. Lye and Jha also reported ten CCAAs managed conservatively (mean 6.9 years). Three (30%) improved, six (60%) were unchanged and one (10%) died following intracranial hemorrhage [14]. Goldenberg *et al*. reported ten CCAAs without treatment, three (30%) remained stable, and seven (70%) worsened [15]. Common indications for treatment of CCAAs are projection of the aneurysm into the subarachnoid space, acute thrombosis, giant size, 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. In our series, all the CCAAs are giant and/or symptomatic, so an invasive management was needed.

Surgical approaches for direct repair of CCAAs are technically challenging, especially for giant aneurysms, and carry with them a high risk of cranial nerve morbidity. For these reasons endovascular approaches have become the primary treatment modality for CCAAs. Endovascular obliteration of CCAAs with preservation of the parent artery is the most desirable outcome. Current advancements in self-expandable neurovasular stents and flow-diverting devices are expected to broaden the application of the endovascular option in the management of CCAAs. Hauck *et al.* [16] reported 15 patients with very large and giant unruptured ophthalmic and cavernous aneurysms treated with stent/coil, five patients improved during follow-up and 10 patients remained unchanged, however, twelve patients required retreatment. Robert *et al.* [17] also reported a 35.3% reopening of stented aneurysms (mean size: 10.6 ± 6.8 mm), which may leave a bleeding risk, despite 52.6% patients had an improved symptom with favorable follow-up outcome. Recently, the use of flow-diverting devices has been rapidly popularized with increasing reports of their remarkable ability to reconstruct the neck of large, complex intracranial aneurysms. Some initial published results indicate a higher rate of complete occlusion at follow-up with flow diverters than conventional techniques, with less complications leading to permanent neurologic deficits or death [18] [19].

However, van Rooij considered flow diverters is a dangerous therapy for unruptured aneurysms, especially in patients who cannot tolerate ICA occlusion, due to the 10% combined morbidity and mortality together with the risk of delayed aneurysm rupture and delayed parent vessel occlusion had reported [20]. These findings indicate that further prospective studies are still required in order to evaluate the exact causes of complications and the long term effectiveness of this treatment method.

Previously, patients with giant CCAAs were treated with balloon occlusion both with and without bypass. There is a risk of cerebral infarction in 25% of patients when adequate collateral blood flow is not assessed [21]. Hence, only a patient successfully passed a BTO, can the ICA be endovascularly sacrificed proximal and distal to the aneurysm to exclude it from the circulation. In our study, 17 patients underwent PAO for the treatment of giant CCAAs presenting with mass effects (n = 15) or discovered incidentally (n = 2). Obliteration of the aneurysm was achieved in all patients. Our results concerning PAO treatment are very encouraging: In a mean follow-up of 31.8 months (with a range of 8 to 57 months), complete resolution or improvement of ocular motor nerve (third, fourth, and sixth) paresis occurred in 70.6% of our patients, while 29.4% showed no improvement. Previous statistical studies stress that the degree and duration of preoperative cranial nerve palsy is an important factor in determining eventual recovery [22]. Our results agree with this, as the recovery of symptoms are significantly higher in patients who undertook treatment less than 1 month when symptoms onset. A recent systematic review of endovascular PAO for carotid cavernous aneurysms confirmed low complication and high aneurysm obliteration rates [1] [23] [24]. It seems probable that coiling eliminates the pulsatility inside the aneurysm sac and, therefore, the "hammer effect" on the nerve. Nassir *et al.* [22] also reported the degree of aneurysmal occlusion did not correlate with the resolution of symptoms, due to the reduction of aneurysmal pulsatility despite incomplete occlusion. Shrinkage of approximately 57% of initial volume after 18 months of endovascular coiling has been reported [25]. Clarencon *et al*. [1] observed 26 giant/large aneurysms of the ICA treated by PAO with or without intra-aneurismal occlusion, clinical symptoms had disappeared in 75% of the patients, partially regressed in 10%, and remained unchanged in 15%. No patient presented worsening of clinical symptoms or intracranial bleeding. Niiro *et al.* [26] analyzed the results of the long-term follow up of 11 patients with a giant or large cavernous sinus aneurysm treated by only proximal occlusion between 1975 and 1989. Eight of the 11 patients (72.7%) showed improvement of cranial nerves paresis or headache. In our series, symptoms were resolved or improved in 70.6%. In a series by Mansour *et al.* [22], the complete resolution rate was 71.4% for aneurysms <15 mm and 50% for aneurysms >15 mm. However, in the largest published cohort of carotid cavernous aneurysms, Stiebel-Kalish *et al.* [27] reported that endovascular PAO did not alter the patient's final diplopia compared with observation alone, but only that it reduced the incidence and severity of facial pain.

Complications of PAO include early or late stroke and "*de novo*" aneurysm formation at a distant site because of hemodynamic changes in the circle of Willis [26] [28] [29]. In our patient group, of the seventeen aneurysms treated by PAO, one (5.9%) developed post-occlusion ischemic infarction. Although the detailed mechanisms of this ischemic complication presented here have not yet been clarified, it may results from thrombosis and embolism which caused by either embolism related to intraoperative angiography or manipulation around the thrombosed aneurysm rather than hypoperfusion because of insufficient revascularization. In other series, the permanent morbidity ranges from 0% to 8.3%, and the mortality ranges from 0% to 6%, respectively [1]. The overall low mortality/morbidity in our series is noteworthy because the aneurysms were selected for the PAO because of their complex architecture with associated higher risk. No new aneurysms were found in our patients. However, longer term follow-up data will be needed to draw definitive conclusions regarding new aneurysm formation.

5. Conclusion

Most patients in this series improved or remained stable after PAO treatment. The results of this study indicated that PAO is an effective and safe method in patients with giant symptomatic CCAAs with low periprocedural morbidity if BOT can be tolerated. Larger multicenter series are needed to better determine the long term effectiveness of this treatment method.

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