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Cavernous carotid artery aneurysms: Epidemiology, natural history, diagnostic and treatment. An experience of a single institution

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ABSTRACT

Background: Cavernous carotid aneurysms (CCA) account for 2–9% of all intracranial aneurysms. They have been considered benign lesions, most often asymptomatic, and to have a natural history with a low risk of life-threatening complications. These aneurysms are unique, their rupture can present in many different forms, they can suffer spontaneous thrombotic changes and the symptomatology related to the mass effect involves the neuro-ophthalmologic system. In this scenario the natural history and clinical presentation are largely different from other intracranial aneurysms. Some investigators advocate treatment of both symptomatic and asymptomatic CCAs, others recommend no treatment. The reason for this controversy relates to a lack of information on the long term natural history of these aneurysms, as well as on the long term results of treatment.

Methods: In this article the authors discuss their single institution experience in diagnosis, natural history and management of 123 asymptomatic and oligosympotomatic aneurysms located in the cavernous portion of internal carotid artery.

Conclusions: According to our results asymptomatic or olygosymptomatic (pain) CCAs should be conservatively managed with serial images while the others presentations should be analyzed by a multidisciplinary team, involving the neuroendovascular and microsurgical services.

1. Introduction

Cavernous carotid aneurysms (CCA) account for 2–9% of all intracranial aneurysms [1] and 15% of those originated in the internal carotid artery [2]. They have been considered benign lesions, most often asymptomatic, and to have a natural history with a low risk of life-threatening complications [3–5]. The etiology of CCAs can be traumatic, infectious or idiopathic. These aneurysms are unique, their rupture can present in many different forms, they can suffer spontaneous thrombotic changes and the symptomatology related to the mass effect involves the neuro-ophthalmologic system [6]. In this scenario the natural history and clinical presentation are largely different from other intracranial aneurysms. The current modalities of treatment include endovascular strategies, microsurgical approaches, Hunterian ligation with or without revascularization and expectant management [3–6]. Thus, although some investigators advocate treatment of both symptomatic and asymptomatic CCAs, others recommend no treatment [6]. The reason for this controversy relates to a lack of information on the long term natural history of these aneurysms, as well as on the long term results of treatment [7,8].

In this article the authors discuss their single institution experience in diagnosis, natural history and management of the asymptomatic and oligosympotomatic aneurysms located in the cavernous portion of internal carotid artery.

2. Method

The authors recorded the data of 100 patients that were followed in the Division of Neurological Surgery in the University of Sao Paulo between June/2009 until January/2014. Patients were referred by a variety of routes, including numerous Sao Paulo emergency departments and after inpatient and outpatient evaluations by outside neurologists. Only patients with spontaneous CCA arising from C4 segment, which begins at the superior margin of the petrolingual ligament and extends to the proximal dural ring, were included. Aneurysms that were partially intradural, traumatic and mycotic etiologies were excluded. Those patients who presented

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Table 1
Distribution of concomitant aneurysms outside carotid cavernous artery.

Aneurysm site (outside carotid cavernous segment)	No. of aneurysms	%
Internal carotid artery (communicating posterior segment)	25	26
Internal carotid artery (ophtalmic segment)	14	14.6
Paraclinoid aneurysm	8	8.3
Internal carotid artery bifurcation	4	4.2
Middle cerebral artery	25	26
Anterior cerebral artery	4	4.2
Anterior communicating artery	3	3.2
Pericallosal artery	2	2.1
Basilar bifurcation	7	7.3
Posterior cerebral artery	2	2.1
Vertebral artery	1	1
Posterior–inferior cerebelar artery	1	1
Total	96	100

with carotid cavernous fistula which treatment was required were excluded from analysis.

The symptoms at presentation and follow up evaluations were documented. Olygosymptomatics were classified those patients whose pain were the only complain. Findings related to bleeding, compressive neuropathy, and amaurosis were also evaluated.

3. Results

One hundred patients had 123 CCAs. Of the 100 patients, 92 (92%) were women and 8 (8%) were men. The mean age at diagnosis was 60 years (range: 21–85 y). At presentation, compressive neuropathy were documented in 30 patients (30%), of this diplopia was observed in 27 patients (27%), trigeminal pain in 8 patients (8%) and amaurosis in 1 patient (1%).

In 32 (32%) patients the CCAs were incidentally discovered during diagnostic evaluation of subarachnoid hemorrhage from other site. In 25 patients (25%) other neurological conditions justified the evaluation. In 13 (13%) cases the diagnosis was made after stroke, and in 12 (12%) for other reasons like post traumatic headache, seizure, lupus, glaucoma, jugular vein thrombosis and dural fistula.

In 54 (54%) patients 96 other aneurysms in intradural sites were identified and located as follow: 26% in middle cerebral artery, 26% in posterior communicating segment of internal carotid artery (ICA), in 14.6% in ophtalmic segment of ICA. Seven patients presented with aneurysms at basilar artery bifurcation. In 23 patients the CCAs were bilateral. Other compromised sites are depicted in Table 1.

Of 35 patients with giant carotid cavernous aneurysms others aneuryms in supratentorial compartment were observed in 10 patients. On the other hand of 65 patients with non-giant carotid cavernous aneuryms others concomitant aneurysms were observed in 44 paients ($p = 0.04$).

According to the size, CCAs were classified as "baby aneurysms"(<4 mm) in 29 (23.5%) of the carotid cavernous aneurysms, between 5 and 9 mm in 44 (35.7%) (Fig. 1), between 10 and 24 mm in 10 (8.1%) and giants in 40 (32.5%) (Table 2).

Table 2
Distribution of the aneurysms regarding to size.

Size (in milimiters)	No. of aneurysms	%
Up to 4 mm	29	23.6
5–9 mm	44	35.8
10–24 mm	10	8.1
>25 mm	40	32.5
Total	123	100

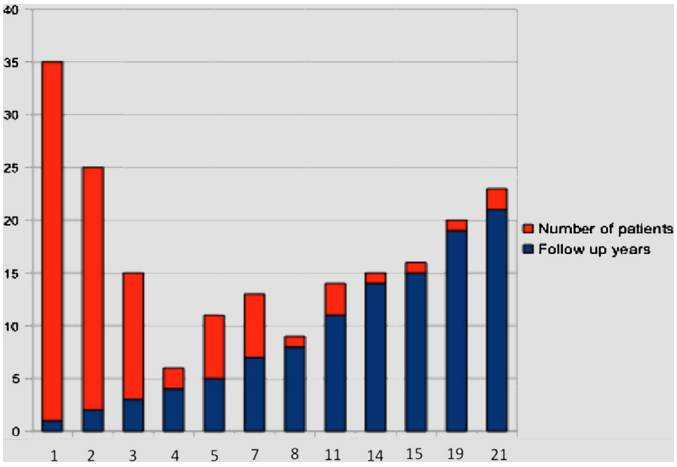

Fig. 1. Carotid cavernous aneurysms. Follow-up years.

Thrombotic changes were observed in 6 (6%) at presentation. Erosion of sphenoid sinus was observed in 6 (6%) patients.

The follow-up period after diagnosis is demonstrated in Fig. 1. It comprised 966 patients-year with an average of 3.7 years per patient (range 1–21 years).

During the follow up evaluation, 71 (71%) maintained asymptomatic and 29 (29%) had complaints like trigeminal pain and diplopia. No patient presented bleeding of the aneurysm. Pain was treated conservatively with antidepressants, anticonvulsivants and analgesic opioids. Diplopia was managed conservatively, with adaptative measures.

4. Discussion

From our experience, which is the largest single institution series reported to date, we observed that these lesions have a low propensity for life threatening sequelae. Classically, the risk of rupture with the creation of a carotid cavernous fistula, compressive cranial neuropathy, progressively worsening headache and erosion of the sphenoid sinus are the several compelling reasons to treat this entity [6,9,10]. CCAs requiring treatment tend to be large or giant [6]. According to ISUIA data on CCAs, the rupture risk of asymptomatic lesions <13 mm in size is approximately 0% over 5-years [11]. Although most intradural aneurysms can be treated surgically or with endovascular techniques that isolate them from parent vessel without occluding that vessel, CCAs usually could not be treated in these ways [6,12–15].

Generally, the goal of intracranial aneurysm surgery is to obliterate the aneurysm while the flow in the vessels associated with the aneurysm is maintained [6]. On the other hand, for CCAs other strategies are employed [6–9]. Techniques of historical interest include hunterian parent artery ligation, after balloon test occlusion, either surgically or through endovascular methods [13–17]. However, in cases of pain, asymptomatic patients and simple cranial neuropathy the conservative management can be proposed [4,18]. In general, the treatment of CCAs requires occlusion of the ipsilateral ICA with the risk of stroke, blindness or both [4]. Endovascular occlusion of the ipsilateral ICA is said to have lower risks of subsequent rupture and cerebral ischemic complications than carotid ligation, but this issue remains controversial [4]. Thus, although some investigators advocate treatment of both symptomatic and asymptomatic CCAs, others recommend no treatment [6]. The reason for this controversy relates to a lack of information on the long-term natural history of these aneurysms, as well as on the long-term results of treatment [7,8].