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Cavernous carotid artery aneurysms on a single institution: An epidemiological study with 201 patients

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ABSTRACT

Objectives: Cavernous carotid aneurysms (CCA) represent 2–9% of all intracranial aneurysms. For long considered benign lesions, these entities are unique when it comes to clinical presentation and management. Usually asymptomatic, CCAs can grow and rupture causing different manifestations. The lack of a long-term assessment of both treated and untreated CCAs' natural history justifies why there is no consensus regarding what are the recommended therapeutic measures. While some advocate that an intervention is always necessary, others consider that patients deserve an individualized evaluation.

Patients and Methods: We describe our single-institution experience in diagnosis, follow-up, and management of 201 CCAs. In addition, we evaluate the association of giant CCAs with aneurysms in other locations using a Chi-square test.

Results: 201 patients had 245 CCAs. 92% of the patients were women. The mean age at diagnosis was 61 years. Concomitant aneurysms were observed in 53.2% of the patients, and the middle cerebral artery was the most affected artery. 66 (30.6%) CCAs were considered "giant", and the follow-up period ranged from 1 to 23 years.The presence of a giant CCA seemed to hinder other aneurysms' formation - RR 0.47 (IC 95% 0.31–0.67), $p < 0.0001$.

Conclusions: CCAs should be individually assessed. A conservative approach ought to be adopted for asymptomatic and oligosymptomatic lesions. Finally, a multidisciplinary team must evaluate the other situations, in order to define whether the microsurgical or the endovascular treatment is better option. Presence of a giant lesion within the cavernous sinus is associated with less occurrence of other aneurysms.

1. Introduction

Cavernous Carotid Aneurysms (CCAs) account for 2–9% of all intracranial aneurysms [1]. CCAs' etiology is variable, as they range from idiopathic to traumatic, infectious and iatrogenic lesions [2]. Whereas the first is often incidentally detected and merely needs continued follow-up, the others tend to require a more aggressive approach [2].

The clinical symptomatology associated with CCAs is majorly linked to their mass effect – due to their growth – and rupture [3]. Cranial nerve palsies, the most commonly observed manifestation, are primarily represented by the neuro-ophthalmologic system disruption [4]. However, trigeminal neuralgia and headache are also frequent. In addition, optic nerve impairment and subarachnoid hemorrhage can result from a CCA large transitional variant, which has an intradural component [5,6]. Caroticocavernous fistulas may follow intracavernous rupture [2]. On the other hand, fatal epistaxis is an unusual complication of aneurysmatical rupture into the sphenoid sinus [3].

When it comes to treatment modalities, the physicians may choose to assume an expectant approach or to intervein. When choosing the latter, either endovascular or microsurgical techniques can be performed. Hunterian ligation – with or without revascularization – is another possible option [7–9]. It is important to remember that spontaneous thrombosis of CCAs and ischemic strokes originating from intra-aneurysmal thrombus have already been reported [10,11].

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It is yet to be established whether or not it is necessary to treat all CCAs [4]. Currently, whereas some investigators recommend active management of both asymptomatic and symptomatic lesions, others defend an individualized evaluation of the different cases – therefore allowing isolated expectation. The scarce information about the natural history of CCAs, as well as the lack of a long-term assessment of the treated patients partially explain this divergence [12,13]. In addition, there are no studies in the literature that directly analyzed the concomitant occurrence of a giant intracavenous aneurysm and other aneurysms.

This article describes the major clinical series reported thus far. The authors discuss diagnosis, natural history, and management of the internal carotid artery's cavernous aneurysms, providing the reader with an overview of their single institution experience. In addition, we investigated whether the presence of a giant CCA is associated with smaller prevalence of aneurysms in other locations, as we hypothesized that the flow modifications caused by presence of a giant lesion may diminished the hemodynamic stress in distal arteries. This research is an update of a previously published work [14], with twice as many cases.

2. Patients and methods

The authors recorded the data of 201 patients, who arrived in the service through different routes, after inpatient and outpatient assessment by experienced neurologists, and after being referred by several emergency departments.

Only patients with idiopathic CCA arising from the C4 segment – that extends to the proximal dural ring and begins at the petrolingual ligament superior margin – were included. Conversely, infectious, traumatic, and partially intradural aneurysms were excluded. The symptoms – present at the moment of diagnosis and developed throughout the course of disease – were documented. Findings related to amaurosis, bleeding and compressive neuropathy were also evaluated.

A two-tailed Chi-square test for association between giant CCAs and concomitant supratentorial aneurysms was performed.

The patients were evaluated in the Division of Neurological Surgery of the University of São Paulo from June/2009 to March/2019.

3. Results

Two hundred and one patients had 245 CCAs. 185 of the patients were women (92%), and 16, men (8%). The mean age at diagnosis was 61 years – ranging from 21 to 88 years. At this moment, 135 patients were asymptomatic (67.2%), whereas 55 reported compressive neuropathies (27.4%). Of these 55 symptomatic patients, 50 reported diplopia (91%), 9 trigeminal pain (16.4%), and 1 amaurosis (1.8%). Furthermore, 15 of all patients experienced headache (7.5%).

In 113 (56.2%) cases neurological conditions such as headache, diplopia, and trigeminal pain justified the evaluation that led to the diagnosis. In 50 (24.9%) patients the CCAs were discovered during subarachnoid hemorrhage assessment. In 27 patients (13.4%) the diagnosis was made after stroke. In 10 cases (5%), the physicians reached the diagnosis because of other reasons, including seizures, brain tumor investigation, jugular vein thrombosis, myasthenia and dural fistula.

Besides the CCA, 107 (53.2%) patients had other 160 aneurysms in intradural sites. These were located as follow (Table 1): 20.6% in the internal carotid artery (communicating posterior segment); 23.1% in the internal carotid artery (ophthalmic segment); 11 paraclinoid (6.9%), 38 (23.8%) middle cerebral artery, 4 (2.5%) anterior cerebral artery, and 10 (6.3%) anterior communicating artery aneurysms were found. Moreover, 2 (1.3%) anterior choroidal artery, 5 (3%) internal carotid artery bifurcation and 5 (3%) pericallosal artery aneurysms were observed.

Regarding the posterior circulation, we noticed 10 (6.3%) basilar bifurcation, 1 vertebral artery (0.6%), 2 posterior cerebral artery and 2 posterior–inferior cerebellar artery (1.3% each) aneurysms.

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)	33	20.6
Internal carotid artery (ophtalmic segment)	37	23.1
Paraclinoid aneurysm	11	6.9
Internal carotid artery bifurcation	5	3.0
Middle cerebral artery	38	23.8
Anterior cerebral artery	4	2.5
Anterior communicating artery	10	6.3
Pericallosal artery	5	3.0
Basilar bifurcation	10	6.3
Posterior cerebral artery	2	1.3
Vertebral artery	1	0.6
Posterior–inferior cerebellar artery	2	1.3
Choroidal artery	2	1.3
TOTAL	160	100.0

In 34 patients (17%), the CCA was bilateral. Of the 16 men, 10 (62.5%) had left CCA and 7 (43.8%), right CCA. Among the 185 women studied, 116 (62.7%) had right CCA and 102 (55.1%), left. Moreover, when we consider the total amount of CCAs and the whole population, we find 128 right CCAs – a prevalence of 63.7% - and 117 left CCAs (prevalence of 58.2%). Of 66 patients with giant carotid cavernous aneurysms, 20 had other concomitant aneurysms (30.3%). Contrarily, 87 patients from the group that did not have giant aneurysms (64.4%) presented other aneurysms in the supratentorial compartment. A relative risk of 047 (IC 95% 0.31 – 0.67) was obtained, indicating the protective effect of giant aneurysms ($p < 0.0001$).

The CCAs were classified according to size (Table 2). We found 58 (26.9%) aneurysms < 4 mm ("baby aneurysms"), 65 (30%) between 5 and 9 mm, 27 (12.5%) ranging from 10 to 24 mm, and 66 (30.6%) > 25 mm ("giant aneurysms").The follow-up period after diagnosis, demonstrated in Fig. 1, ranged from 1 to 23 years.

4. Discussion

To our knowledge, this the largest single-institution series reported to date. In agreement with the available literature, we observed that CCAs have a low morbimortality rate. CCAs' management, nonetheless, remains a field of dilemma. The risk of compressive cranial neuropathies, persistent – and progressive – headache, sphenoid sinus erosion, and rupture – which can generate caroticocavernous fistula – are a few of the reasons why some researchers advise prompt intervention despite the patients' symptomology at the time of diagnosis [15,16].

Notwithstanding, ISUA data on CCAs revealed that the risk of rupture of oligosymptomatic lesions ≤12 mm is about 0% in 5 years. For those aneurysms that range from 13 to 24, the cumulative risk is 3% [17]. This benign course justifies with others choose to be more selective towards surgical treatment. CCAs grow enclosed in the cavernous sinus. This fact explains why most of these lesions not only tend to be incidentally found but also tend to become large or giant before any clinical manifestation occurs. It is valid to remember that as for now

Table 2
Distribution of aneurysms regarding to size.

SIDE	No. of aneurysms	%
Up to 4 mm	58	26.9
5–9 mm	65	30.0
10–24 mm	27	12.5
> 25 mm	66	30.6
TOTAL	216	100.0

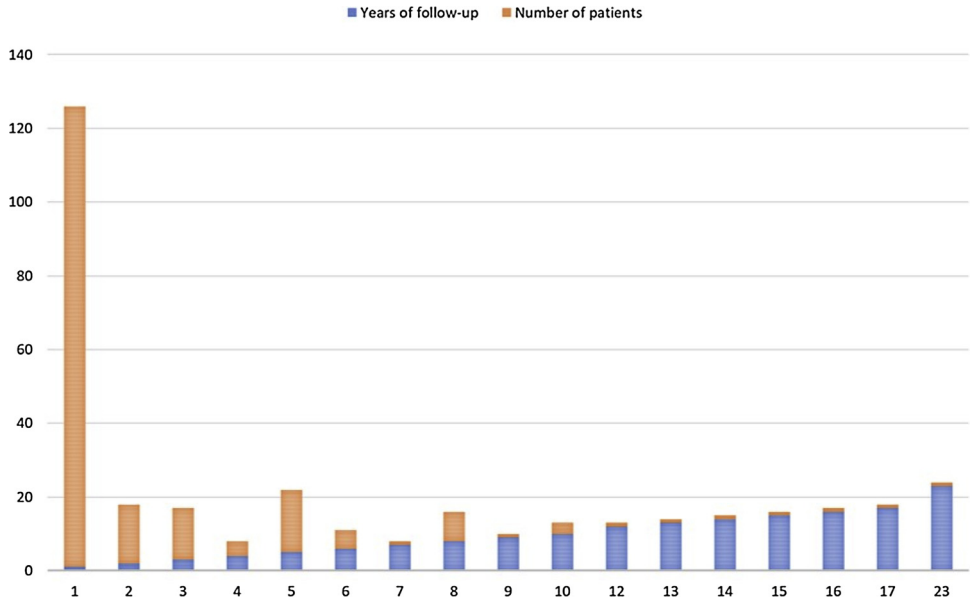

Fig. 1. Carotid cavernous aneurysms. Follow-up years.

these aneurysms differ from the others; common predictors – for example, age, gender, and aneurysm size – do not strongly apply for CCAs [3].

Overall, despite the controversies, physicians agree that bleeding CCAs, as well as those associated with unbearable pain, thromboembolism and visual loss require direct approach [3]. Finally, CCAs with an intradural component and expanding aneurysms with a relevant risk of sphenoid bone erosion are other indications of immediate treatment, regardless of any symptom [3]. Although there are formal recommendations towards the management of some specific cases, CCAs that do not pose as an impending threat, for instance, those related to less morbid manifestations, land on a grey area – where an individualized – and often conservative – therapeutic plan must be traced.

Historically, Hunterian parent artery ligation after balloon test occlusion, either surgically or through endovascular methods, must be mentioned as a treatment alternative [18–22]. When surgery is necessary, and its benefits outweigh the intervention risks, the aim is to obliterate the CCA, without jeopardizing the flow in the associated artery [4]. These aneurysms' treatment usually requires ipsilateral internal carotid artery occlusion, which can result in blindness and stroke.[8] An endovascular approach is linked to fewer ischemic complications, but up to this moment, neither technique has proven to be statistically superior [8,23]. Our management protocol includes surgical, endovascular and conservative options. For asymptomatic lesions, we favor conservative management. Giant symptomatic aneurysms, aneurysms that present bony erosions or are transitional should be treated, either by intracranial-extracranial anastomosis or stenting. However, this series is representative of the natural history of these lesions, since the great majority of the cases have not been treated.

CCAs can arise from any cavernous carotid artery segment. However, they commonly appear in the horizontal section, which is related to the meningohipofisary artery, the inferolateral trunk and the McConnell's capsular artery [14]. The hemodynamic stress observed in this segment allows a better understanding of CCAs genesis. The aneurysms project laterally and forwardly, below the anterior clinoid process [14]. We noticed a significant association between the presence of giant CCAs and the absence of other concomitant supratentorial aneurysms ($p < 0.0001$), demonstrating giant CCAs "protective role". Meng et al. [24] have previously demonstrated the impact of an accelerated flow in its adjacent regions; according to this study, the phenomenon leads to an internal elastic lamina disruption. Fibronectin and medial muscle cells are likewise impaired, resulting in a strong aneurysm formation predisposition, due to high wall shear stress.

Whether or not the aforenamed stress can promote aneurysms rupture – or progression – is yet to be answered. Nonetheless, some researchers have suggested that while a high wall shear stress may trigger small aneurysms growth and rupture, a low stress may have the same effect on large, atherosclerotic aneurysms due to an inflammatory reaction [25,26].

It could be thought that when someone has a giant CCA, the diminished wall shear stress could act as a protective factor, hindering aneurysm formation in other sites of the supratentorial compartment. Although this finding's reproducibility corroborates the speculation, further research is necessary in order to adequately address this query. Worldwide studies state that trigeminal pain and neuro-ophthalmological manifestations are still the most common complaints among CCAs patients, leading them to seek particular treatments, as there is no spontaneous improvement reported [27]. The use of analgesics in CCA-related pain was assessed by Stiebel-Kalish et al [8]. Researchers found that up to 56% of patients improved with this medication. Adversely, they reported persistence of pain after successful endovascular treatment, in another group of patients. Conservative approach, however, was associated with a relapsing–remitting pain course in another study.[20] In our series, all the symptomatic patients improved with analgesic therapy.

Because there are several reports in which an active treatment did not modify the patient's diplopia, this symptom was not considered a strong reason to indicate treatment in our research. It is important to highlight that 44% of untreated CCAs have no complaints in spite of ocular misalignment – a fact easily understood due to second image central suppression or to adaptative head positioning [8].

Approximately 2% of patients with untreated and asymptomatic CCAs evolve with thromboembolic events [8,28]. In our study, cerebral infarcts happened in 26 patients (13%). There were no deaths, and the follow-up of these subjects included aspirin as a secondary prevention measure. Bavinzski et al. study showed a mortality of 0% among the 11-years followed-up patients with untreated CCAs 28].

Current data reveals that surgical complications occur in up to 37% of patients, whereas the endovascular approach complicates in 5–9% of the cases [4,8]. Rupture and perforation of aneurysms, thromboembolic events, coil rupture and migration, and vasospasm are some the possible complications [4,8]. When different aneurysms are taken into consideration, thromboembolic events after embolization can occur in up to 61% of the patients, as stated by Soeda et al [29]. As thrombotic

modifications are usually observed in CCAs, this number may be higher when only these entities are considered. Furthermore, the majority of the microembolization lesions are asymptomatic, and their impact on cognitive functions is unknown.

The incidence of cerebral infarct seems to be greater in treated CCAs (4%), than in untreated ones (2%) [8]. Vasconcellos et al. [20] reported brain infarctions in 3 of the 19 patients who underwent interventionist treatment. Similarly, in Schaaf et al.'s [30] study, 1.6% of patients suffered ischaemic lesions in the first 24 h, while 3,4% experienced late complications.

Visual loss as a manifestation of optic nerve compression is a formal indication of either endovascular or microsurgical treatment. Among the 201 analyzed patients, only one experienced this symptom. She successfully underwent a bypass procedure. Multicenter series affirm that about 5% of the CCAs are associated with this complication [8].

Nowadays, refined techniques and equipment, as well as an optimized pretreatment screening, which includes temporary balloon occlusion alongside SPECT scanning and cerebral angiography, have led to a diminished CCAs morbimortality rate. Lastly, most investigators advocate against an internal carotid artery occlusion, for the increased blood flow in the opposite side can trigger aneurysms development [4,14,21]. This procedure was not performed in our patients.

4.1. Limitations of the study

Due to its retrospective characteristic, this research has inherent limitations. The lack of a validated questionnaire is another setback. Our series, nonetheless, reinforces the benign features of CCAs, as none of the evaluated patients worsened throughout outpatient follow-up. Thus, the tendency toward interventional therapy does not apply for CCAs, which ought to be individually assessed. As formerly stated, this is the most extensive series of CCAs. Cavernous carotid aneurysms are unique entities confined inside bone structures that differ from other supratentorial aneurysms due to their low rupture rate. The natural history of CCAs is not fully understood, but its benign course allows a conservative approach in the majority of the cases. Physicians must remember to individually assess the patients, for there are associated risks of osseous erosion and persistent growth, which can ultimately cause death or long-term disability. Presence of a giant CCA is associated with smaller prevalence of aneurysms in other location, which may imply in a protective role due to alterations in flow and in hemodynamic stress. We emphasize the need for more prominent studies in order to further characterize these lesions course and therapeutic implications.

5. Conclusions

CCAs should be individually assessed. This series with the largest number of cases described thus far recommends a conservative approach for asymptomatic and oligosymptomatic lesions. A multidisciplinary team must evaluate the other situations, in order to define whether the microsurgical or the endovascular treatment is better option. Presence of a giant lesion within the cavernous sinus is associated with less occurrence of other aneurysms.

Author contributions

Jefferson Rosi Junior: study concept and design, acquisition of data.
Louise Makarem Oliveira: study concept and design.
Antonio Luiz Boechat: study concept and design, study supervision.
Manoel Jacobsen Teixeira: study supervision.
Eberval Gadelha Figueiredo: study concept, acquisition of data, study supervision.

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