Case Report

Report of intradural aneurysm in the cavernous segment of the internal carotid artery presented with subarachnoid hemorrhage and oculomotor palsy

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INTRODUCTION

Subarachnoid hemorrhage (SAH) is a severe pathology with high morbidity and mortality and requires a fast and accurate therapeutic decision. We describe the case of a patient who presented at the emergency unit with acute SAH and left III cranial nerve palsy. Basic computed tomography (CT) was negative, but lumbar puncture was positive for SAH. Angio-CT showed an aneurysm located in the posterior genu of the cavernous segment. Cavernous segment aneurysms are classically extradural and present without SAH, raising the question in this case of the factors that could have led to such evolution. In a review of the literature, there is only one case reported with aneurysm located in the same topography described by Andaluz et al., 2003[2]. In this report, the patient presentation with sudden thunderstorm headache and the aneurysm was not ruptured.
CASE REPORT

A 55-year-old male patient with a medical history of systemic arterial hypertension was admitted to the neuroemergency department, after being transferred from another service, with a report of sudden, intense thunderstorm headache, associated with the left eyelid ptosis and diplopia. During the neurological examination, the patient was awake, lucid, and oriented. He had complete palsy of the left oculomotor nerve, with no other focal neurological deficits. Neck stiffness was present.

Cranial CT performed on the day following the headache showed no SAH, but showed an expansive process in the sella turcica associated with sellar enlargement [Figure 1]. SAH was then confirmed by lumbar puncture (Fisher I). A cranial angio-CT was made and revealed an intradural saccular aneurysm in the cavernous segment of the left internal carotid artery (ICA) [Figure 2].

The patient underwent cranial microsurgery for cerebral aneurysm clipping that confirmed the intradural location of the aneurysm, arising from a tortuous cavernous ICA [Figure 3].

He underwent control cerebral angiography on the 2nd postoperative day that demonstrated complete aneurysm occlusion [Figure 4], with no residual neck and no vasospasm and complemented the study with magnetic resonance imaging (MRI) of the sella turcica that was consistent of pituitary macroadenoma with apoplexy [Figure 5]. He was discharged on the 21st day after the hemorrhage, maintaining complete left oculomotor nerve dysfunction, but with no other complaints and no neurological deficits.

DISCUSSION

The cavernous segment of the ICA begins at the superior margin of the petrolingual ligament, just after the ICA emerges from the foramen lacerum. In general, the cavernous segment of the ICA includes the first vertical portion, the posterior genu, the anterior directed horizontal

Figure 1: Angio-CT scan. (a) Cavernous segment of the right internal carotid artery, below the line of projection of the planum sphenoidale (red dotted line). (b) Cavernous segment of the left internal carotid artery with its posterior genu above the line of projection of the planum sphenoidale (red dotted line). (c) Enlargement of the sella turcica and left carotid artery laterally displaced. (d) Superior displacement of the cavernous segment of the internal carotid artery and an aneurysm close to the communicating segment of the internal carotid artery (white arrow).

Figure 2: Three-dimensional reconstruction of cranial angiotomography showing saccular aneurysm (arrow) in the cavernous segment of the left internal carotid artery.

Figure 3: (a) Intraoperative image showing the carotid aneurysm (white arrow), compressing the left oculomotor nerve; (b) schematic representation of the aneurysm (white arrow), compressing the oculomotor nerve; (c) image after microsurgical clipping (black arrow). AC: Anterior clinoid; CN II: Optic cranial nerve; CN III: Oculomotor cranial nerve; FL: Frontal lobe; ICA: Internal carotid artery; R: Fixed retractor.

Figure 4: Postoperative left carotid angiography (a: Frontal; b: Oblique; and c: Lateral) showing the clip adjacent to the cavernous segment of the left internal carotid artery, with complete aneurysm occlusion.
segment, and the anterior genu exiting the cavernous sinus at the proximal dural ring, which is composed of the junction of the medial and inferior periosteum of the anterior clinoid process. The cavernous segment of the carotid is surrounded by a venous plexus known as the cavernous sinus. Classical teaching is that this segment of the carotid artery is all extradural. Aneurysm rupture in this location causes arteriovenous fistulas and rarely results in SAH. Proximal ICA aneurysms that result in SAH are usually located between the proximal and distal dural ring and are known as transactional or paraclinoid aneurysms when located in the dural cavity and therefore with some intradural projection. A review of the literature identified only one similar case report of posterior genu aneurysm with intradural projection without passing through the clinoid space. The origin of the cavernous segment is normally surrounded by the dura of the cavernous sinus, the bones of the sphenoid sinus, the sella, and the base of the skull, which provide support for the aneurysm wall and restrict expansion. This may be the reason why extravascular rupture occurs infrequently. As long as the rupture occurs within the cavernous sinus, the blood remains intravascular within the venous system, creating a carotid-cavernous fistula (CCF). The signs and symptoms of a CCF are well known, principally causing cranial neuropathy and orbital congestion.

There are, however, some case reports of SAH from cavernous ICA aneurysms. In one report, spontaneous SAH due to rupture of a cavernous ICA aneurysm occurred in a patient whose large prolactinoma had shrunk following cabergoline treatment. Dural erosion from giant intracavernous aneurysms has been reported during autopsy and craniotomy. Although intracavernous aneurysms smaller than 1.5 cm have been associated with SAH, dural erosion into the subarachnoid space is more likely to occur in cases of giant aneurysms and aneurysms arising from the anterior genu of the carotid siphon and those eroding into the sella turcica. In a case of SAH from a cavernous aneurysm without evidence of dural erosion, a loose dural ring was proposed as the mechanism of the SAH. Our patient had a pituitary macroadenoma diagnosed incidentally on the initial CT scan. The patient had no previous history of cabergoline treatment, but further investigation with MRI revealed findings suggestive of the previous pituitary apoplexy, which could explain the further shrinkage of the adenoma.

As shown in our patient's angiographic study, the aneurysm was located at the posterior genu of the cavernous segment that should be inside the cavernous sinus. By definition, the cavernous segment is completely extradural, except for those that become partially intradural by traversing the clinoidal space (between the proximal and distal dural rings); in these cases, they constitute transitional (or clinoidal) aneurysms. During surgery, it was observed that this aneurysm was fully exposed in the intradural compartment. The etiological hypothesis for this finding would be that initially a large adenoma may have eroded the dural boundary of the cavernous sinus. Subsequently, shrinkage of this adenoma following apoplexy could have left a residual dural defect allowing communication between the cavernous sinus and the subarachnoid space and further exposure of the posterior knee of the cavernous segment of the ICA. One of the possible locations for the dural failure might be the virtual ring where the oculomotor nerve enters the cavernous sinus and that had been loosened after the tumor shrank, forming a route for the aneurysm to extend intradurally and compress the third cranial nerve, resulting in the dysfunction of the third nerve, present in our patient, as shown in Figure 6.

**CONCLUSION**

This case report confirms that anatomical variations produced by disease process can lead to intradural exposure of the proximal cavernous segment of the carotid artery. A detailed analysis of image studies and critical surgical planning is required to avoid surprises or intraoperative

![Figure 5: Sella turcica magnetic resonance imaging consistent with pituitary macroadenoma with apoplexia (a: Axial fluid-attenuated inversion recovery; b: Coronal T1 with gadolinium; and c: Sagittal T1 with gadolinium).](image)

![Figure 6: Cranial magnetic resonance imaging (sagittal T1 with gadolinium) showing the presence of an expansive sellar tumor (pituitary macroadenoma) involving the internal carotid arteries. Left: The cavernous segment of the right internal carotid artery (ICA) maintains its usual curvature, being restricted to the cavernous sinus (arrow). Right: The left ICA is elongated and its curvature exceeds the limits of the cavernous sinus (arrow).](image)
technical difficulties and determine the best therapeutic strategy.

**Declaration of patient consent**

Patient consent not required as patients identity is not disclosed or compromised.

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**Conflicts of interest**

There are no conflicts of interest.

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