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Case Report

Ruptured aneurysm in the posterior communicating segment of carotid artery presenting with contralateral oculomotor nerve palsy

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ABSTRACT

Background: Brain aneurysms are mostly discovered during the investigation of subarachnoid hemorrhage (SAH). Some patients present neurological signs that may suggest the aneurysm's topography, and the oculomotor nerve palsy (ONP) of the same side of the aneurysm is the most common sign. Only one case report of contralateral palsy was previously described in the medical literature.

Case Description: Authors describe a patient who presented a classic manifestation of SAH associated with complete ONP, whose vascular investigation demonstrated a brain aneurysm located in the contralateral intracranial carotid. The patient was surgically treated with great neurologic outcome, and late angiography did not evidence other vascular abnormalities.

Conclusion: The ipsilateral ONP is a common sign found in posterior communicating artery aneurysms; however, such aneurysm can have different presentations due to the elevation of intracranial pressure, and, in rarer cases, the ONP cannot be operated as a localizing sign.

Keywords: Brain aneurysm, Cranial mononeuropathy, Oculomotor nerve disease, Ruptured intracranial aneurysm, Subarachnoid hemorrhage

INTRODUCTION

Subarachnoid hemorrhage (SAH) is a condition characterized by the presence of blood in the subarachnoid space, whose origin may be secondary to arterial dissection, trauma, and venous or aneurysm rupture.[18] Among the nontraumatic SAH, aneurysm rupture is responsible for 85% of the cases and represents the most life-threatening etiology of SAH whose 30-day mortality can reach 50%, and 30% of the survivors have moderate-to-severe disability.[1,5,11] Mortality occurs secondary to complications of the primary bleeding, such as increased intracranial pressure (ICP), hydrocephalus, and late brain ischemia; or due to a rebleeding, if the aneurysm is not urgently treated.

Oculomotor nerve palsy (ONP) is a classic localizing sign of aneurysms situated along its course, being the posterior communicant artery (PComA) or communicant segment of internal carotid artery (ICA), the main locations of aneurysms associated with ONP at the same side of the aneurysm.[4,8,19] The third nerve dysfunction usually results from a direct compression of the nerve by the aneurysm growth or rupture.[18] However, contralateral presentation represents a very rare clinical finding of this pathology[5,18]

This case report describes the third case of ONP associated with contralateral ICA-PComA junction aneurysm,[15,17] but the second whose palsy was manifested at the acute scenario. The patient consented to publication of his image.

CASE REPORT

A 60-year-old male, with no previous medical comorbidity, woke up during the night with a severe sudden headache. The patient tried to get up from the bed when he lost consciousness. The patient could not determine how long he persisted unconscious, but when he recovered mental functions, he called for medical support (patient lives alone). According to the patient, since he recovered consciousness, he persisted with a painful headache noticing that he could not open his left eyelid. The urgent medical assistance team promptly forwarded the patient to a tertiary hospital. At emergency admission, the patient was conscious, interacting adequately with the physicians, mobilizing all members without motor or sensitive deficits, but presented a marked neck stiffness and positive Brudzinski's sign. The patient presented a complete left eyelid ptosis, with divergent strabismus due to a lateral rotation of the left eyeball and anisocoria with the left pupil mydriasis. Ocular extrinsic movement of the right eyeball was preserved; however, the superior, inferior, and medial movement of the left eyeball was compromised [Figure 1], and the left pupil constriction due to light stimuli was absent (for both photomotor and consensual tests). The patient was immediately submitted to a computed tomography (CT) examination without contrast infusion, which showed SAH in the interpeduncular and ambient cisterns, interhemispheric and lateral fissures, and also in the fourth ventricle [Figure 2]. Once the diagnose of spontaneous SAH was established, the patient was submitted to arteriography of the brain vessels that showed an aneurysm of PComA segment of the right ICA, measuring 9 mm of length, 5 mm wide, and with a neck of 4 mm. No other aneurysms were identified. Therefore, authors concluded that they were facing a patient with a ruptured aneurysm of the ICA-PComA junction, classified as Hunt-Hess 2 and CT Fisher scale of 4. The patient was submitted to surgical clipping of the aneurysm. The procedure was conducted without aneurysm bleeding or other surgical complications, and the patient returned to the intensive care unit (ICU) where he was managed according to the current protocols for the treatment of acute SAH. The patient presented a satisfactory clinical and neurologic recovery, without late ischemia, vasospasm, infections, or other systemic complications. The patient was released from the hospital with an excellent clinical condition harboring only the oculomotor palsy that he had partially recovered by the time of 6 months of follow-up.

DISCUSSION

Aneurysmal SAH usually presents unspecific symptoms such as severe headache, vomiting, and photophobia. These symptoms, despite its importance to suspicion of SAH, do not allow the topographic diagnosis of the aneurysm. However, the presence of certain infrequent specific signs can suggest the aneurysm position, operating as localizing signs.[2,3] Examples of these signs are partial blindness by ICA-ophthalmic artery junction aneurysm,[14] ataxia and nystagmus by posterior circulation aneurysms,[3] and complete ONP by PComA aneurysms.[2,10]

The oculomotor nerve emerges from the interpeduncular fossa in the midbrain, medially to the cerebral peduncles. At its point of emergence of the brainstem, it is closely related to the posterior cerebral artery (PCA) superiorly and superior cerebellar artery (SCA) inferiorly. Furthermore, the nerve

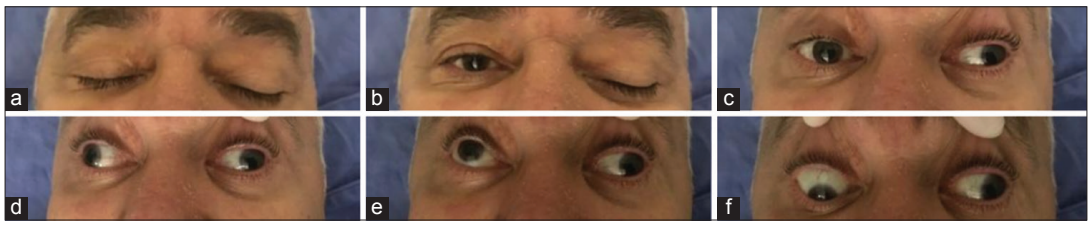

Figure 1: Ocular extrinsic examination at hospital admission demonstrating complete left oculomotor nerve palsy. (a) Both eyelids closed. (b) Complete ptosis of the left eyelid. (c) The patient looking forward, demonstrating a left rotation of the left eyeball due to abducens nerve function not balanced by the oculomotor nerve stimuli over the medial rectus muscle. (d) The patient looking to the right, without medial movement of the left eyeball. (e) The patient looking upward without superior vertical movement of the left eyeball. (f) The patient looking downward without inferior vertical movement of the left eyeball.

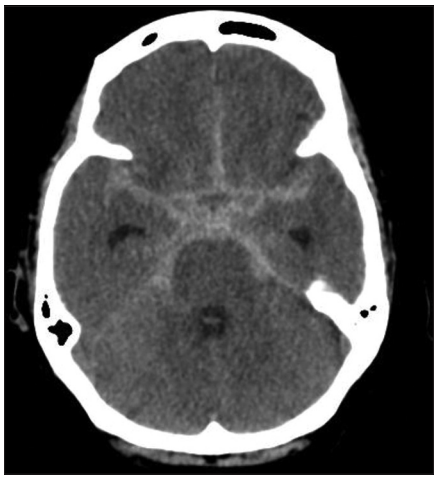

Figure 2: Computed tomography at hospital admission demonstrating subarachnoid hemorrhage on the interpeduncular and ambient cisterns, interhemispheric and lateral fissures, and also in the fourth ventricle.

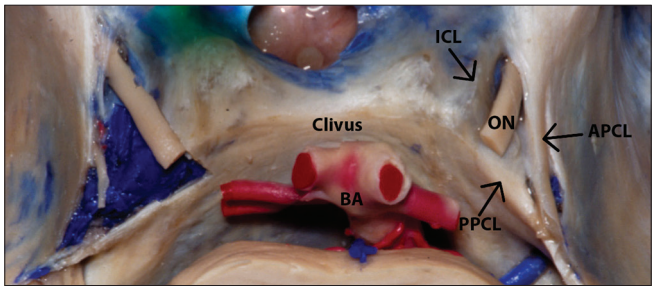

Figure 3: Entry of the oculomotor nerve into the cavernous sinus through the oculomotor triangle, which is a potential compression point. Structures of the oculomotor triangle are shown. ON - Oculomotor nerve, BA - Basilar artery, APCL - Anterior petroclinoid ligament, PPCL - Posterior petroclinoid ligament, ICL - Interclinoid ligament. Courtesy of the Rhoton Collection, American Association of Neurological Surgeons/Neurosurgical Research and Education Foundation.

gets its vascular supply from the basilar system at this point. It passes inferomedial to the uncus and inferolateral to the PComA to enter the roof of the cavernous sinus (CS) through the oculomotor trigone. Up to this point, the nerve can be affected by several pathological processes such as tumors, clots in the interpeduncular cistern, and direct compression by aneurysms of the underlying arteries.[12]

The cavernous segment of the oculomotor nerve runs through the lateral wall of the CS superiorly. The nerve enters on the CS passing through the oculomotor triangle, which is delimited by the anterior petroclinoid ligament, the posterior petroclinoid ligament, and the interclinoid ligament [Figure 3]. In addition, there is an entry point known as oculomotor porus and represents the main fixation point of the nerve. These areas of attachment and narrowing of the nerve pathway could explain the high vulnerability during pathological conditions, which allow contusions against the dural (ligaments) and bone structures (petrous apex).[12]

The ICA-PComA junction is the most common location of aneurysm that courses with ipsilateral ONP, with a prevalence of 34%–56% among ruptured cases.[16] However, due to the proximity with other arterial structures, the palsy can also be associated with aneurysm located at the ICA, SCA, PCA, basilar artery apex, and anterior choroidal artery. [4,8,19]

In the aneurysm scenario, the main mechanism of ipsilateral ONP is related to the compression of the nerve fibers by the aneurysm itself, increased ICP with or without brain herniation, compression within the perimesencephalic cisterns by focal blood clot, or chemical lesion by the blood metabolites in subarachnoid space.[18] In the few reports of contralateral palsy in the acute SAH scenario, some theories to explain the remote contralateral deficit were proposed: one hypothesis consists in compression of nerve due to the increased ICP. All the cranial nerves can be affected by increased ICP with or without herniation syndromes,[13] and it cannot be excluded as a possible cause of the deficit in our patient. Other possibilities are oculomotor nerve compression by a subarachnoid blood clot within the perimesencephalic cistern and the lesion by the jet stream of blood from the aneurysm. In this scenario, the osmotic tonicity of the surrounding blood, the toxic effects of the concentrated blood breakdown products, and the ischemia by compression of the small feeding arterioles of the nerve can contribute.[6] Moreover, vasospasm of feeding arteries of the nerve is also a possible cause. However, the early onset of the palsy in our patient suggests that vasospasm, which typically occurs 4 days after SAH,[7,9] is less likely to be the etiology.

Especially in this case, authors hypothesize that elevation of ICP may be more strongly pointed as the etiological factor of the contralateral ONP due to the establishment of the deficit in the ictus of the event. A sudden elevation of ICP may compress the oculomotor nerve at its vulnerable points into the CS roof or in the oculomotor trigone.[12] Compression of the nerve by clots can also not be ruled out as hypothesis since there was a presence of hyperdense images in the perimesencephalic cistern in the CT [Figure 2].

Somagawa *et al*. reported a similar case of an acute SAH of the ICA-PComA junction with contralateral ONP.[15] In this case, the patient was affected with neurogenic pulmonary edema due to spontaneous SAH. After 1 day on the ICU, the patient presented with rebleeding and the left ONP. Subsequent angiography revealed a right ICA-PComA aneurysm. The clinical finding was associated with a massive perimesencephalic hemorrhage on CT, which suggests that

the compression by the clot or the jet stream could have been the cause of the contralateral nerve lesion. The properly treatment of the aneurysm with coiling did not improve the palsy deficit.[15]

Another case reported was a patient of Sun *et al*., who presented a PComA-ICA junction aneurysm that was treated with clipping.[17] Preoperatively, a lumbar catheter was placed after anesthesia induction to facilitate intraoperative brain relaxation and postoperative drainage of bloody cerebrospinal fluid (CSF). On postoperative day 3, the patient developed a complete left ONP that was associated with CSF overdrainage by the lumbar catheter: a head CT showed collapsed cerebral cisterns and shifted midline structures. This could collectively push the oculomotor nerve to the tentorial notch, causing the palsy. In addition, the clipping of the catheter resulted in the total recovering from the ONP palsy 6 h later. Although it occurred in the acute SAH scenario, the contralateral ONP was associated with CSF overdrainage and not directly to the pathophysiological consequences of the presence of blood in the subarachnoid space.

In our case, the definitive treatment of the aneurysm with clipping was not associated with early improvement; however, at 6 months of follow-up, he had partially recovered the nerve's functions. The recovery from nerve palsy depends on the time between the onset of symptoms and surgery. The earlier the operation is done, the higher is the chance of nerve function recovery.[2,4,19] In addition, such recovery is less likely if the palsy is present for more than 10 days before surgery or if signs of recovery have not started within a few weeks after operation.[16,19]

This case may demonstrate that ONP may behave as a nontopographic sign in cerebral aneurysmal disease, and advanced neuroimaging is required to determine the location of aneurysms in patients with SAH.

CONCLUSION

Contralateral ONP is an extremely rare symptom of ICA-PComA aneurysm with only two cases reported, including ours. The pathophysiology of this phenomenon may depend on several factors such as intracranial hypertension, the presence of clots in the subarachnoid space, or the toxic effect of blood metabolites. In cases of late contralateral ONP, other events such as vasospasm and rebleeding may also be associated.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that his names and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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