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Case Report
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Unilateral Sixth Nerve Palsy due to Spontaneous Subarachnoid Hemorrhage

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Abstract

Context: Unilateral Abducens Nerve Palsy (ANP) caused by ruptured Anterior Communicating Artery (ACoA) aneurysm has been rarely reported. Most of the ANP reports due to spontaneous subarachnoid hemorrhage (SAH) are bilateral paralysis. Facilitate Cerebrospinal Circulation (CSF) through microsurgery could help to solve this neurological deficit after SAH.

Case Report: A patient was admitted on emergency department with sudden onset of headache, vomiting and left unilateral sixth nerve palsy. Computed tomography angiography revealed cisternal SAH and a 4 mm saccular aneurysm on anterior communicating artery. The patient underwent surgical aneurysm clipping with concomitant lamina terminalis and Lilliequist's membrane fenestration. Few days after the procedure she evolved with complete ANP regression and received discharge without neurological deficits.

Conclusions: Microsurgical lamina terminalis and Lilliequist's membrane fenestration performed concomitant to the surgery for aneurysm clipping may be a factor, which contributes to more rapid clinical improvement in patients who develop ANP after SAH.

Keywords: Sixth nerve palsy; Subarachnoid hemorrhage; Lilliequist's membrane; Lamina terminalis

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Introduction

Unilateral Abducens Nerve Palsy (ANP) caused by ruptured anterior communicating artery (ACoA) aneurysm has been rarely reported [1]. In the literature, most of the ANP reports due to spontaneous subarachnoid hemorrhage (SAH) are bilateral paralysis. There are some causes that may explain the unilateral ANP, as the following listed: increased intracranial pressure induced by brain swelling or parenchyma hemorrhage; direct aneurysm mass effect on abducens nerve, compression by the localization of cerebrospinal fluid (CSF) or hematoma in the basal cistern space and vasospastic pontine branch of basilar artery supplying to abducens nuclei [2].

The sixth cranial nerve has a long intracranial course and therefore is affected in several pathologies. Non-traumatic isolated ANP has an undetermined prevalence and its diagnosis is a challenge in daily routine demanding a detailed investigation. ANP could greatly affect quality of life especially when cause diplopia.

In this article, the authors describe a case of a patient who developed unilateral ANP after spontaneous SAH due to a ruptured ACoA aneurysm. Microsurgical clipping of the aneurysm with concomitant fenestration of Lilliequist's membrane and lamina terminalis was performed. A brief literature reviews also performed.

Case Report

A 40 year-old female patient was admitted on emergency department after sudden onset of severe headache and vomiting. Recent history of trauma was denied. On admission, the patient was alert and did not present any history of comorbidity. On examination, she was conscious with mild neck stiffness. There was no oculomotor and trochlear nerve palsy but it was noticed a left abducens nerve palsy (Figure 1). The initial brain Computed Tomography (CT) revealed thick cisternal SAH mainly on inter-hemispheric fissure but also present in pre-pontine cistern and Sylvian fissure bilaterally (Figure 2A and 2B). Computed Tomography Angiography (CTA) was performed and revealed a 4 mm saccular aneurysm on anterior communicating artery (Figure 2C and Figure 2D).

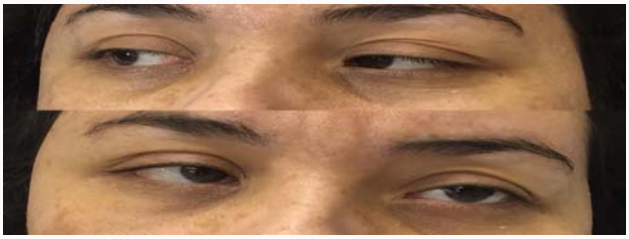

Figure 1: Left unilateral sixth nerve palsy.

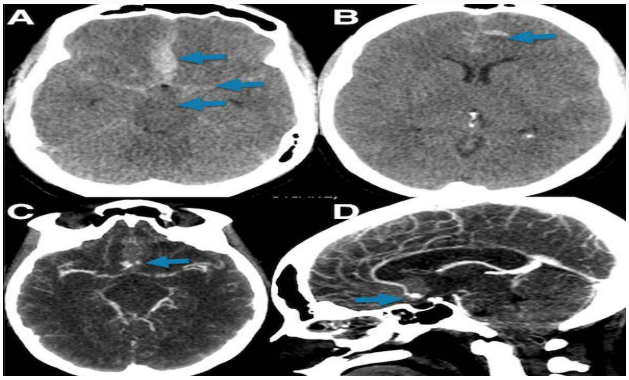

Figure 2: Brain computed Tomography (CT) revealed SAH and Computed tomography angiography (CTA) revealed a saccular aneurysm:
A: Blue arrows - SAH on interhemispheric fissure, pre-pontine cistern and Sylvian fissure bilaterally.
B: Blue arrow - SAH present on interhemispheric fissure.
C: Blue arrow - 4 mm saccular aneurysm on anterior communicating artery (axial view).
D: Blue arrow - 4 mm saccular aneurysm on anterior communicating artery (sagittal view).

After five hours of headache onset, the patient underwent to microsurgery through a classical right pterional approach with a transylvian route to reach the aneurysm and after its clipping, lamina terminalis and Lilliequist's membrane fenestration was performed. Surgery occurred without complications. It was performed a head CT and transcranial Doppler that showed no abnormalities. The patient was monitored daily through neurologic examination and showed improvement of symptoms after 7 days. The patient was discharged after 15 days of hospitalization without changes in neurologic examination.

Discussion

ANP is the most common isolated cranial nerve palsy due to its long peripheral course. Several causes are related and could have non-traumatic or traumatic etiologies [3]. The abducens nucleus is located in the caudal pons at the level of the facial colliculus. In the vicinity of the abducens nerve nucleus are facial nerve loop and pontine gaze center. Abducens nerve exits the brainstem at the border of the pons and medullary pyramids. Upon exiting the brainstem it climbs superiorly along the ventral surface of the pons, through the pre-pontine cistern. On its way to the cavernous sinus it passes at the apex of the petrous portion of the temporal bone penetrating the dura mater through the Dorello'scanal [1,4]. In the cavernous sinus it passes below and lateral to the internal carotid artery and laterally to oculomotor, trochlear and first and second branch of the trigeminal nerve to enter the orbit through the superior orbital fissure [5].

Most cases of ANP after SAH are bilateral and it is not found in the literature the incidence of ANP associated with the specific location of cerebral aneurysm. There are also few reports that describe the deficit start time. As previously reported, some possible causes of unilateral and isolated ANP: increased intracranial pressure, direct aneurysmal mass effect, compression by the localization of Cerebrospinal Fluid (CSF) or hematoma in the basal cistern space, and vasospastic pontine branch of basilar artery [6,7].

Considering the size, location and direction of the ACoA aneurysm, the chance of direct aneurysmal compression on abducens nerve could be ruled out in this patient. Absence of marked swelling, acute neither hydrocephalus nor parenchyma hematoma was seen at initial and postoperative brain CT, making the possibility of those etiologies low. The abducens nerve courses through the pre-pontine cistern so a direct mass effect by Cerebrospinal Fluid (CSF) entrapment or clot can cause isolated unilateral ANP after ruptured ACoA aneurysm. We may speculate local CSF entrapment as the most probable mechanism for isolated unilateral ANP in this patient. Opening of lamina terminalis and Lilliequist's membrane may be effective for clinical improvement [2]. Routinely in our service these techniques are performed on surgical clipping of ruptured aneurysms.

The recovery period of isolated abducens nerve palsy related to ruptured ACoA aneurysm has reportedly varied. It is described recovery reports from 3 days to 3 months after aneurysm clipping [8,9]. In the case described it is believed that the quick symptom improvement is due to microsurgical fenestration of Lilliequist's membrane and lamina terminalis that allows the releasing of possible Cerebrospinal Fluid (CSF) or clot entrapped present on the infratentorial cisterns [10].

Conclusions

Several neurological manifestations can occur after a spontaneous subarachnoid hemorrhage due to aneurysm rupture. Neurosurgeons may be aware to the aneurysm localization and the nearby structures for performing a proper evaluation and know what can be expected or no after microsurgery clipping and what could be done to improve neurological deficits.

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