Ruptured Posterior Communicating Artery Aneurysm Causing Bilateral Abducens Nerve Paralyses
- Case Report -

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= Abstract =

A rare case of bilateral abducens nerve paralyses after rupture of a left posterior communicating artery (PcomA) aneurysm with multiple unruptured aneurysms in a 46-year-old female is presented. Sudden left abducens nerve paralysis followed by progressive right abducens nerve paralysis were present without additional neuro-ophthalmological signs. Postoperatively, bilateral abducens nerve paralyses gradually recovered and disappeared in 2 weeks. The authors reviewed and discussed the possible mechanisms involved in this uncommon neuro-ophthalmological manifestation.

KEY WORDS Bilateral abducens nerve paralyses, Ruptured posterior communicating artery aneurysm, Unruptured PcomA aneurysms.

Introduction

Neuro-ophthalmological abnormalities frequently follow the rupture of intracranial aneurysm. They are generally classified into visual field defects, disturbances of ocular movement, and ptosis\textsuperscript{10}. In most cases presenting with disturbances of ocular movement, the location and direction of the aneurysm to the 3rd and 6th cranial nerves (CN) is closely related. However, aneurysms located far from the oculomotor systems may sometimes cause disturbance of ocular movement. In such cases, the manifestations are unilateral or bilateral and appear to be a false localizing sign secondarily to increased intracranial pressure\textsuperscript{9}.

The authors report a case of bilateral abducens nerve paralyses developing immediately after the rupture of a left posterior communicating artery (PcomA) aneurysm with unruptured left anterior choroidal artery (AchA) and right PcomA aneurysms. Possible pathogenesis are reviewed and discussed.

Case Report

A 46-year-old female patient suffered from sudden severe headache and double vision on left lateral gaze without disturbance of consciousness for two days before admission. She visited local clinic and took the computed tomographic (CT) scan, which showed no specific abnormalities. Even treating with some medications, her symptoms were persisted and followed by new double vision on right lateral gaze.

On admission, she was somewhat drowsy but oriented, complained of severe headache. Bilateral abducens nerve paralyses without other cranial nerve palsies were noted. Bilateral extensor plantar responses and nuchal rigidity were also apparent. Brain CT scan revealed slight oblitera-
tion of the left sylvian fissure and mild ventricular enlargement (Fig. 1). Diagnostic lumbar puncture revealed bloody cerebrospinal fluid. Left AchA, PcomA, and right PcomA aneurysms were found on the four-vessel cerebral angiography. Left PcomA aneurysm was elongated and projecting to postero-infero-laterally. And other aneurysms were small, ovoid, and projecting dorsolaterally (Fig. 2, 3).

Because sudden left abducens nerve palsy developed first, it strongly suggested that left PcomA aneurysm was one that had ruptured. A left frontotemporal craniotomy was performed and successful clippings of the ruptured left PcomA aneurysm and unruptured left AchA aneurysm were done. Blood clots were in the perimesencephalic cistern. Postoperative course was uneventful and bilateral abducens nerve paralyses gradually improved and disappeared by the 14th postoperative day. The second operation for the unruptured right PcomA aneurysm was performed with successful clipping 1 month later (Fig. 4).

**Discussion**

Cranial nerve palsies accompanying intracranial aneurysms are not uncommon, but there appear to have been few cases with bilateral abducens nerve paralyses. Disturbances of ocular movements, caused by unilateral oculomotor nerve paralysis, are observed most frequently with ICA and basilar artery (BA) aneurysms. BA or vertebral artery aneurysms may also cause unilateral abducens nerve paralysis.

Pathologic mechanisms causing these paralysis are 1) direct contact to the nerve of the aneurysmal sac, 2) secondary effect of hemorrhage from the aneurysmal rupture, 3) remote effect due to increased intracranial pressure, and...
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4) vasospasm of the basilar artery pontine branches. Most direct compression of the nerve is ascribed to unruptured aneurysm and usually involving the oculomotor nerve. Neuro-ophthalmological abnormalities related to ocular movements are seldom reported after the rupture of intracranial aneurysms. However, the incidence of direct cranial nerve involvement by the aneurysm is nearly equal to that of indirect involvement secondary to hemorrhage or intracranial hypertension.

There are clinical and pathological reports on the directly compressed cranial nerves by the aneurysm itself only in optic, oculomotor, and abducens nerves. The location of the aneurysm was usually the anterior part of the circle of Willis, most commonly in relation to the internal carotid artery. However, few authors reported that over three fourths had definite evidence of small amount of blood extravasation, suggesting leakage of blood. Clinically this evidence indicated that functional impairment of the nerve by an adjacent aneurysm occurs most frequently in conjunction with hemorrhage. While there is no doubt that gradual compression by dilatation of a pulsatile aneurysm sac will damage nerve tissue, this nerve is evidently capable of functional adjustment for a considerable time. An abrupt dilatation without rupture could impair nerve function by mechanical stretching or it might lead to edema or hemorrhage in the involved nerve through venous obstruction, which would account for a sudden onset of the nerve paralysis.

Increased intracranial pressure readily causes abducens nerve paralysis. This is generally considered as a false-localizing sign. The abducens nerve has a long intracranial course at the base of the brain, and is consequently vulnerable to injury, especially where it crosses the petrous apex and traverses the sharp edge of the petrous temporal bone. However, such damage generally results in unilateral abducens nerve paralysis.

Bilateral abducens nerve paralyses in neurosurgical patients is rare and may occur secondary to subarachnoid hemorrhage or increased intracranial pressure). A few ruptured AcomA aneurysm causing bilateral abducens nerve paralyses were reported. It probably results from the symmetrical filling of the basal cisterns with blood and followed by stretching of the structures lying between the brain and skull in the subarachnoid space. The acute hydrocephalus and brain swelling that accompany subarachnoid hemorrhage may also causes the paralyses.

Several authors reported that the mechanism might also be due to vasospasm of the basilar artery pontine branches affecting the abducens motor nucleus. However, other accompanying signs have to be observed in these cases. Bilateral abducens nerve paralyses of the nuclear type is usually associated with gaze abnormality and/or bilateral facial paresis due to the close anatomical relationship between the abducens nuclei, the medial longitudinal fasciculus, and the facial nuclei.

Our patient had multiple aneurysms including ruptured left PcomA aneurysm. With the review of the clinical history, we could conclude that the left abducens nerve paralysis was secondary to hemorrhage and the right abducens nerve paralysis was due to increased intracranial pressure. Therefore, unilateral or bilateral abducens nerve paralyses may occur after ruptured or unruptured aneurysms. Precise history taking and more meticulous neuro-ophthalmologic or neuroradiologic examination should be done to evaluate cranial nerve paralyses.

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