Unilateral Oculomotor Nerve Paresis Associated With Anterior Communicating Artery Aneurysm Rupture

—Two Case Reports—

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Abstract

Two cases of complete unilateral oculomotor nerve palsy occurred after subarachnoid hemorrhage (SAH) due to a ruptured anterior communicating artery aneurysm. A 61-year-old female suffered left oculomotor nerve paresis after mild SAH. This paresis was probably related to pre-existing oculomotor nerve stretching caused by abnormal positioning of the posterior cerebral and superior cerebellar arteries in the premesencephalic cistern. A 70-year-old female suffered right oculomotor nerve paresis after severe SAH. Elevated intracranial pressure might have caused this paresis, but the reason for the unilateral occurrence was undetermined. Both patients were treated by clipping of the aneurysm, and the signs of oculomotor nerve paresis gradually resolved. A pattern of pupil-sparing paresis was observed during the early recovery stage in both patients.

Key words: anterior communicating artery aneurysm, oculomotor nerve paresis, subarachnoid hemorrhage

Introduction

Unilateral oculomotor nerve paresis associated with subarachnoid hemorrhage (SAH) is usually caused by rupture of an ipsilateral internal carotid artery (ICA) aneurysm or, less frequently, of a basilar artery (BA) aneurysm. Aneurysms at other sites seldom cause oculomotor nerve paresis, except in association with cerebral herniation.

We report two cases of SAH caused by rupture of an anterior communicating artery (AcomA) aneurysm. In both cases, unilateral oculomotor nerve paresis was the initial neurological sign.

Case Reports

Case 1: A 61-year-old female was admitted to our hospital because of sudden onset of headache without consciousness loss. On admission, the patient was alert and had no neurological deficit except for left oculomotor nerve paresis. The left eye showed ptosis with complete absence of adduction. The left pupil was 5 mm in diameter and nonreactive to light; the right pupil was 3 mm in diameter. Computed tomography (CT) revealed only moderate

SAH (Fig. 1), and angiography showed an AcomA aneurysm. No aneurysm was detected on the ICA or BA. Additionally, the interpeduncular and crural portions of the left posterior cerebral artery (PCA)

Fig. 1 Case 1. Computed tomography scan on admission revealing moderate subarachnoid hemorrhage, predominantly on the right.

Fig. 2 Case 1. A: Right internal carotid angiograms showing an anterior communicating artery aneurysm, but no internal carotid artery (ICA) aneurysm. The posterior communicating artery runs downward after branching from the ICA. B: Left vertebral angiograms showing the premenencephalic portion of the duplicated left superior cerebellar artery (arrow) running above the posterior cerebral artery.

were displaced downward, and the corresponding portions of the duplicated left superior cerebellar artery (SCA) were displaced upward. Therefore, the positions of these two arteries were inverted in the premenencephalic cistern (Fig. 2).

On the day after admission (Day 1), the AcomA aneurysm was clipped via a right pterional approach, and rupture of this aneurysm was confirmed. The patient's pupils became isocoric on Day 3, ptosis improved on Day 16, and ocular adduction returned by Day 30. The patient returned home without evident neurological deficits, and no neurological problem was observed during the 6-year follow-up period. However, the patient refused follow-up magnetic resonance (MR) imaging.

Case 2: A 70-year-old female was admitted to our hospital because of severe headache and subsequent transient respiratory distress. She had a 5-year history of slowly progressive dementia, presumed to be Alzheimer's disease. On admission, the patient was confused and delirious. No paresis was observed in her face or extremities. The right eye showed complete ptosis. The right pupil was nonreactive to light and 5 mm in diameter; the left pupil was 2.5 mm in

Fig. 3 Case 2. Computed tomography scan on admission revealing severe subarachnoid hemorrhage.

Fig. 4 Case 2. Right internal carotid (left) and vertebral (right) digital subtraction angiograms revealing no aneurysms around the right internal carotid artery or basilar artery.

diameter. The ocular movements were difficult to evaluate because of the disturbed consciousness and pre-existing dementia, but absence of adduction in the right eye was suspected. CT revealed a dense clot in the basal cistern and brain atrophy (Fig. 3). Angiography showed an AcomA aneurysm but no other abnormality (Fig. 4).

The aneurysm was clipped via a right pterional approach on the day after admission, and we observed that the right oculomotor nerve appeared intact in the internal carotid cistern. The patient's pupils became isocoric on Day 7, the ptosis disappeared, and adduction returned by Day 60. MR imaging on Day 45 showed no abnormal lesion in the midbrain or basal cistern (Fig. 5). The patient

Fig. 5 Case 2. T₂-weighted magnetic resonance images revealing no abnormal intensity in the midbrain or basal cistern except for the artifact caused by the aneurysm clip.

remains bed-ridden mainly because of the dementia.

Discussion

There are several possible pathological causes of oculomotor nerve paresis, including midbrain bleeding or ischemia, ischemia of the nerve itself, tumors, trauma, neuritis, meningitis, and intracranial aneurysm.¹⁾ A reported series of 1130 patients with oculomotor nerve paresis at the Mayo Clinic included 179 cases (15.8%) caused by aneurysms, but the cause was undetermined in 270 cases.¹⁴⁾

Another series of 24 patients with oculomotor nerve paresis due to intracranial aneurysms included 23 patients with ICA aneurysm and one with a BA-SCA aneurysm.⁴⁾ Another large series of 534 SAH patients included 31 patients with oculomotor nerve paresis associated with a ruptured ICA aneurysm on the ICA ($n = 17$), BA aneurysm ($n = 5$), middle cerebral artery (MCA) aneurysm ($n = 7$), posterior inferior cerebellar artery aneurysm ($n = 1$), and distal anterior cerebral artery aneurysm ($n = 1$).⁹⁾ The number of patients with MCA aneurysm leads us to suspect that some of the cases were associated with cerebral herniation.

The present cases of oculomotor nerve paresis caused by AcomA aneurysm are very unusual. Clinically, our patients presented with complete oculomotor nerve palsy. Pupil response was the first sign of recovery. Both patients showed a pattern of pupil-sparing oculomotor nerve paresis during the early recovery stage, which is not common with ICA aneurysms or traumatic damage.^{7,8,14,16)} Pupil-sparing is most likely to result from extraaxial microvascular ischemia, which mainly occurs in diabetic patients, because pupillary fibers are located peripherally in the oculomotor nerve.¹⁾ However, our patients had no history of diabetes and did not report eye pain which often accompanies oculomotor nerve ischemia. Acute subdural hematoma,⁶⁾

compressive cavernous sinus lesions,¹²⁾ cerebrovascular accidents or tumors in the midbrain,^{3,15,17)} and insult to the nerve root exit zone by an aneurysm or tumor^{10,12,18)} can also cause pupil-sparing paresis, but our patients showed no radiological evidence of these conditions.

Our Case 1 had PCA and SCA inversion in the premenencephalic cistern, an anomaly that has not been reported in previous anatomical studies.^{2,5,11,13)} The oculomotor nerve may pass under the SCA in the case of branching from the PCA, but there is no case of the nerve passing above the PCA.^{2,11,13)} The nerve in Case 1 was probably chronically stretched downward by the PCA and upward by the SCA. We speculate that this unique situation weakened the nerve at the premenencephalic cistern, which resulted in the onset of paresis after the relatively mild SAH. This would also explain the occurrence of pupil-sparing, which results from the insult to the nerve at the root exit zone.¹⁸⁾ Pupil-sparing in this case could also have occurred because the small-caliber unmyelinated parasympathetic pupillomotor fibers are more resistant to chronic stretching than other fibers in the oculomotor nerve.¹²⁾

Our Case 2 suffered severe SAH with elevated intracranial pressure, which may have caused the oculomotor nerve paresis. An alternative explanation is that the dense clot in the basal cistern stretched and damaged the nerve. However, these speculations do not support the very unusual onset or the pupil-sparing observed in this patient. Because no particular pathology was detected around the nerve, the origin of unilateral oculomotor nerve paresis in this patient is undetermined.

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