

Incomplete Oculomotor Nerve Palsy Caused by an Unruptured Internal Carotid-Anterior Choroidal Artery Aneurysm

—Case Report—

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Abstract

A 59-year-old woman visited our institute with the chief complaint of dizziness which persisted whenever she tried to focus on objects. She had not experienced apparent double vision and had no history of intracranial bleeding. Neurological examination revealed no abnormality except for exotropia at the mid-position and at upper gaze. Cerebral angiography revealed that the intracranial portion of the left internal carotid artery ran more horizontally and also identified an unruptured left internal carotid-anterior choroidal artery (IC-AChA) aneurysm of 3.0 mm diameter. The aneurysm at the origin of the AChA was confirmed during surgery. The proximal lateral wall of the aneurysm was in contact with the oculomotor nerve. This contact was released after complete obliteration of the aneurysm. The exotropia resolved 3 months later. Oculomotor nerve palsy usually indicates the presence of internal carotid-posterior communicating artery (IC-PcomA) aneurysm. Since sacrifice of the AChA will result in severe neurological deficits, accurate neuroimaging information is needed prior to the operation. Conventional angiography and/or three-dimensional computed tomography angiography should be performed to ascertain whether the aneurysm is an IC-PcomA or IC-AChA aneurysm, even if some neurosurgeons insist that conventional angiography is not always needed before surgery for an unruptured aneurysm.

Key words: anterior choroidal artery, cerebral aneurysm, internal carotid artery, oculomotor nerve, posterior communicating artery

Introduction

The most common cause of secondary oculomotor nerve palsy is ischemia of the peripheral nerve caused by a disease, such as diabetes mellitus.^{4,9)} Another common cause of secondary oculomotor nerve palsy is compression by an intracranial aneurysm, usually an internal carotid-posterior communicating artery (IC-PcomA) aneurysm.^{1,11)} Unilateral oculomotor nerve palsy is considered to be a warning sign of the presence and possible rupture of the IC-PcomA aneurysm. The anatomical relationship between the exit of the posterior communicating artery (PcomA) and the entry of the oculomotor nerve into the cavernous sinus usually results in superomedial compression of the nerve by

the aneurysm. Typical oculomotor nerve palsy in this situation may manifest as unilateral pupil dilation, ptosis, incomplete or partial extraocular palsies, and oculomotor synkinesis. The origin of the PcomA and the anterior choroidal artery (AChA) also has a close relationship, but no case of oculomotor nerve palsy associated with internal carotid-anterior choroidal artery (IC-AChA) aneurysm is known.

We describe a rare case of isolated oculomotor nerve palsy associated with a small, unruptured IC-AChA aneurysm.

Case Report

A 59-year-old woman visited our institute with the chief complaint of dizziness which persisted whenever she tried to focus on objects. She had visited our institute 1.5 years before, when a

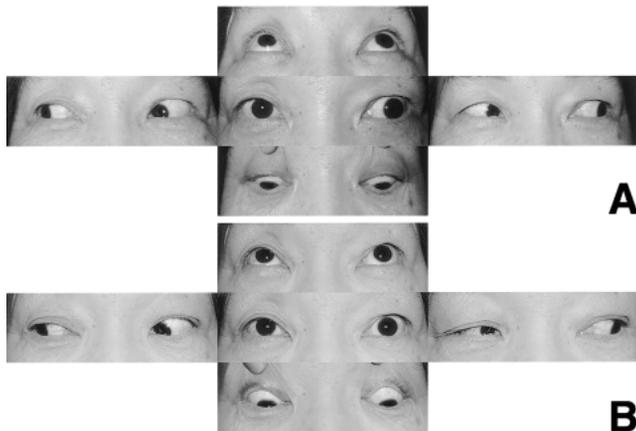


Fig. 1 Photographs showing the ocular position of the patient before (A) and 3 months after (B) the operation. Although abduction on the left was not apparently disturbed, the ocular position was exotropic in the neutral and upward positions, which resolved after the surgery.

cerebral aneurysm was suspected, but she had not returned for further examination. She had a past medical history of tuberculosis during childhood but had no episode suggesting intracranial bleeding. She also suffered from mild hyperlipidemia and had been a smoker for 22 years.

Neurological examination revealed that she was alert and had no motor weakness or sensory disturbance. No cranial nerve deficit was observed except that: external ocular movement was apparently intact by the confrontation test, but the eye position displayed exotropia at the mid-position and at upper gaze (Fig. 1A). She had noticed recently that the appearance around her eyes had somehow become different. However, she had not experienced or noticed apparent double vision. Anisocoria was not constantly observed, but the size of the left pupil was the same or larger under bright light in a few specific situations. These findings suggested left incomplete oculomotor nerve palsy confined to extraocular movement.

Magnetic resonance (MR) imaging revealed no abnormalities indicating subarachnoid hemorrhage or ischemic lesion of the brain parenchyma including the brain stem. However, MR angiography showed an abnormal shadow appearing from the left internal carotid artery (ICA) and protruding laterally, suggesting the presence of cerebral aneurysm. Cerebral angiography showed that the intracranial portion of the left ICA ran more horizontally rather than vertically and clearly revealed a left IC-AChA aneurysm (Fig. 2A, B). The left PcomA was well de-

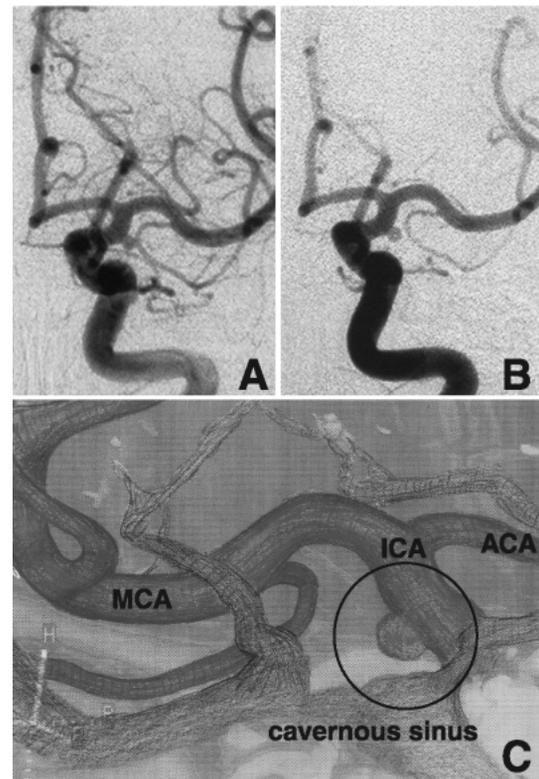


Fig. 2 Routine (A) and caudal (B) projections of the anteroposterior view of the left carotid angiograms showing that the aneurysmal shadow projected laterally. The intracranial portion of the internal carotid artery ran horizontally rather than vertically. The thick posterior communicating artery ran medially from its origin toward the periphery. Three-dimensional computed tomography angiogram (C) viewed from the left posterolateral direction (120 degrees from the anterior sagittal plane) more clearly revealing the relationship between the aneurysm (circled) and the cavernous sinus. The aneurysm projected laterally (95 degrees) toward the cavernous sinus. ACA: anterior cerebral artery, ICA: internal carotid artery, MCA: middle cerebral artery.

veloped. Three-dimensional computed tomography (3D-CT) angiography more clearly revealed the aneurysm protruding at 95 degrees from the anterior sagittal plane (Fig. 2C). The aneurysm was 3.0 mm in diameter.

The diagnosis was left IC-AChA aneurysm with some effect on the oculomotor nerve. Therefore, an operation was performed to eliminate the aneurysm and to allow closer examination of the relationship between the left IC-AChA aneurysm and the oculo-

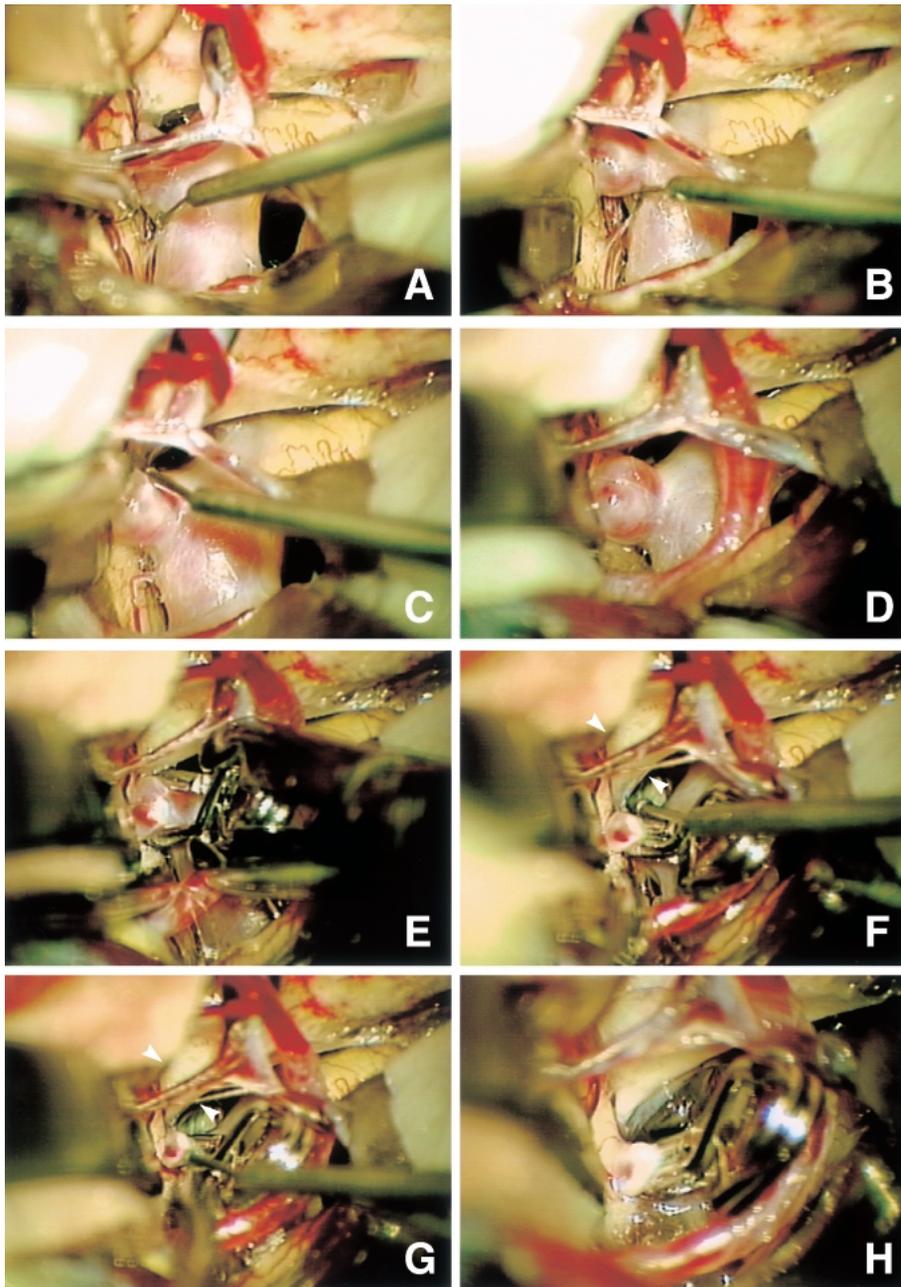


Fig. 3 Intraoperative photographs after the proximal part of the internal carotid artery was secured, showing the probing of the carotid artery toward the periphery to dissect the aneurysm. Part of the aneurysm body was found to be embedded in the temporal lobe (A). The aneurysm was confirmed to originate at the origin of the anterior choroidal artery (AChA) (B). The proximal wall of the body was also dissected and probably contacted the cavernous sinus or the oculomotor nerve (C). Following gentle removal of the aneurysm body from the temporal lobe, the whole aneurysm could be observed with the reddish wall (D). A Yaşargil straight mini-clip was applied to obliterate the aneurysm (E) and to avoid occlusion of the origin of the AChA. After complete obliteration of the aneurysm, the aneurysm wall adhering to the arachnoid around the cavernous sinus was probed and dissected (F). A part of the wall was in contact with the oculomotor nerve (G). The oculomotor nerve was completely freed from the aneurysm wall (H). Arrowheads: oculomotor nerve.

motor nerve.

A left frontotemporal craniotomy was performed, and the proximal part of the sylvian fissure was widely dissected. The proximal part of the left ICA was secured, then the distal part of the intracranial ICA was dissected and observed. The aneurysm was found protruding laterally with almost half of the body embedded in the temporal lobe. The wall of the aneurysm was reddish. The major branches of the AChA, which ran medially toward the periphery, were observed right behind the ICA. The aneurysm was confirmed to arise at the origin of the AChA. The proximal lateral wall of the aneurysm was in contact with the oculomotor nerve. This contact was dissected and released after complete clipping of the aneurysm with a Yaşargil mini-clip (FE720K; Aesculap AG & Co. KG, Tuttlingen, Germany) (Fig. 3).

The postoperative course was uneventful. The left oculomotor nerve palsy showed no aggravation such as anisocoria and double vision after the operation. The exotropia improved gradually and resolved 3 months later (Fig. 1B). She did not complain of dizziness anymore.

Discussion

The clinical features of oculomotor nerve palsy caused by aneurysm compression are well documented.^{4-7,10,13} Orbital pain or headache sometimes proceed to oculomotor nerve palsy which frequently involves the pupil. The external ocular movement is partly disturbed or the eye position becomes exotropic resulting in double vision. Ocular synkinesis is also a specific feature. Considering such characteristics, the diagnosis of cerebral aneurysm at this location is easier to establish. However, the manifestations of oculomotor nerve palsy vary widely.^{1,3,12} The oculomotor nerve palsy may cause only anisocoria, ptosis with or without pupil involvement, or total ophthalmoplegia. Such variation is probably due to the differences in duration, severity, or location of the compression with or without associated arachnoiditis or minor bleeding. Even so, the palsy seems to result from the anatomical relationship between the PcomA and the oculomotor nerve at the entrance of the cavernous sinus. Quite a few cases of oculomotor nerve palsy caused by other types of aneurysms have been reported, such as ICA posterior wall aneurysm¹² or pure PcomA aneurysm.⁸ However, these aneurysms were fairly large space-occupying lesions.

The present patient had slight incomplete oculomotor nerve palsy, that is, exotropia. The patient experienced dizziness when she tried focusing on

objects rather than apparent diplopia, and the pupil and upper eyelid movement were not constantly involved. Angiography revealed that the patient harbored a cerebral aneurysm, but an IC-AChA aneurysm projecting laterally, not the more common IC-PcomA aneurysm. Contact or compression of the oculomotor nerve at the entrance into the cavernous sinus was confirmed during the operation. Moreover, the exotropia gradually resolved after the operation. This small IC-AChA aneurysm could compress the oculomotor nerve because the intracranial part of the ICA ran horizontally rather than vertically along the cranial base, then turned abruptly through a right angle. Additionally, the aneurysm originated at the proximal portion of the AChA and projected laterally rather than posteriorly, so the body of the aneurysm was directly facing the cavernous sinus. This rare coincidence allowed this fairly small IC-AChA aneurysm to cause oculomotor nerve palsy.

Sacrifice of the AChA will result in severe neurological deficits such as motor weakness,² so accurate neuroimaging information must be obtained prior to the operation. MR angiography, conventional angiography, and/or 3D-CT angiography should be performed to ascertain whether the aneurysm is an IC-PcomA or IC-AChA aneurysm, even if some neurosurgeons insist that conventional angiography is not always necessary before surgery for an unruptured aneurysm.

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