Bilateral Oculomotor Nerve Palsies Due to Posterior Cerebral Arterial Compression Relieved by Microvascular Decompression

—Case Report—

Hidemitsu NAKAGAWA, Shin NAKAJIMA, Yoshikazu NAKAJIMA,
Yoshihiro FURUTA, Okihiro NISHI* and Kayo NISHI*

Department of Neurosurgery, The Center for Adult Diseases, Osaka, Osaka;

**Nishi Ophthalmological Hospital, Osaka*

Abstract

A 59-year-old male developed peripheral oculomotor nerve paresis due to compression by the left posterior cerebral artery (PCA), which was successfully treated by microvascular decompression. Two months later, a similar oculomotor nerve paralysis due to the same mechanism occurred contralaterally and was also treated by microvascular decompression. The previous condition was probably caused by arteriosclerotic changes in the PCA, and the following condition by postsurgical adhesion of the arachnoid membrane. The possibility of vascular compression should be considered when oculomotor nerve palsy rapidly develops, although not proven by angiography.

Key words: microvascular decompression, oculomotor nerve palsy, posterior cerebral artery

Introduction

Oculomotor nerve palsy frequently results from compression by an aneurysm at the junction of the internal carotid and the posterior communicating arteries.^{2,17,18} Here, we report a case of bilateral oculomotor nerve compression by the posterior cerebral artery (PCA), which was relieved by microvascular decompression.

Case Report

A 59-year-old male was hospitalized with left oculomotor nerve paresis in June, 1988 (Fig. 1A). He had no past history of severe headache or family history of neurological disease. The cerebrospinal fluid, erythrocyte sedimentation rate, and other laboratory findings were normal. The glucose tolerance test did not show a diabetic pattern. A computed tomographic (CT) scan found no abnormalities. A left carotid angiogram demonstrated a

fetal-type posterior communicating artery and a small aneurysm, 0.3 cm in diameter, at the internal carotid artery proximal to the junction with the posterior communicating artery (Fig. 2 *left*). A right carotid angiogram found no aneurysm but marked arteriosclerotic changes (Fig. 2 *right*). Left vertebral angiograms showed the P₂ portion of the PCA to form a curve with an anterior convexity, possibly due to arteriosclerosis (Fig. 3).

On July 8, 1988, left frontotemporal craniotomy was performed to prevent aneurysmal bleeding and investigate possible vascular compression of the oculomotor nerve. The aneurysm was too small to cause compression of the oculomotor nerve, being a 3.0 mm junctional dilatation. From the top of the dilatation, another posterior communicating artery, not demonstrated on the angiograms, extended to the P₂ portion of the PCA forming the curve on the angiograms. Thus, the posterior communicating arteries were duplicated, and the curved P₂ portion of the PCA severely compressed the oculomotor nerve (Fig. 4A). The nerve was depressed and discolored at the compression site and edematous and swollen at the periphery. The arachnoid membrane

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Fig. 1 A: Left oculomotor nerve palsy at first admission. B: Right oculomotor nerve paralysis at second admission. C: Recovery from the right oculomotor nerve paralysis 3 months after second operation.

was not hypertrophic. After adequate dissection of the arachnoid membrane around the oculomotor nerve and PCA, the nerve was decompressed by inserting a sponge. The neck of the junctional dilatation was then clipped because it had aneurysmal character.

The impaired ocular movements completely resolved 1 month after the operation, and ptosis subsided 2 weeks later. He was discharged with slight double vision.

Two months after the operation, he suddenly experienced diplopia unaccompanied by headache and, 2 days later, right ptosis. Neurological examination on the second admission showed complete right ptosis (Fig. 1B) with complete absence of adduction, elevation, and depression of the right eye and anisocoria (4 mm right, 3 mm left). The cerebrospinal fluid was normal. We assumed the oculomotor nerve paralysis to be caused by vascular compression, so a second operation was performed.

A right subtemporal craniotomy discovered elevation of the oculomotor nerve by a tense arachnoid membrane at the tentorial edge. The nerve was severely compressed by the PCA (P_2 portion) at the

proximal portion and abnormally hyperemic and swollen at the periphery. The superior cerebellar artery was not attached to the oculomotor nerve (Fig. 4B). The nerve was separated from the arachnoid membrane and decompressed by inserting a sponge between the PCA and the nerve.

One and a half months after the operation, both the impaired ocular movement and ptosis well improved. Within 2 months, the impaired adduction had completely recovered and, 1 month later, elevation of the right eye was only slightly limited together

Fig. 2 *left*: Left carotid angiogram, lateral view, demonstrating an aneurysm (arrow) of the internal carotid artery proximal to the origin of the posterior communicating artery. *right*: Right carotid angiogram, lateral view, showing arteriosclerotic changes such as elongation and irregularities of the arteries.

Fig. 3 Left vertebral angiograms, anteroposterior (*left*) and lateral (*right*) views, demonstrating marked anterior protrusion (arrow) at the P_2 portion of the PCA.

Fig. 4 Microsurgical photographs and diagrams. A: First operation through the left frontotemporal approach. Duplicate posterior communicating arteries (PComA) are shown: small one extending from the top of the junctional dilatation (JD) to the P_2 portion of the PCA, and large one distal to the junctional dilatation. The P_2 portion of the PCA compresses the oculomotor nerve (III) forming an anterior convexity (arrow). B: Second operation through the right subtemporal approach. After separating the tense arachnoid membrane and mobilizing the P_2 portion of the PCA, the compressed part (arrow) and swollen peripheral portion of the oculomotor nerve are exposed. BS: brainstem, ICA: internal carotid artery, ret: brain retractor, tent: tentorium, SCA: superior cerebellar artery.

with slight ptosis (Fig. 1C). These residual signs completely disappeared, and he became asymptomatic after 1 year. The anisocoria slightly improved but remained detectable even after 1 year.

Discussion

Vascular compression of the root entry/exit zones or central segments (glial part) of the cranial nerves can cause facial spasm, trigeminal neuralgia, glossopharyngeal neuralgia,⁹⁾ hypertension,¹⁰⁾ vertigo,^{11,12)}

and spasmodic torticollis.⁵⁾ These conditions are all hyperactive dysfunctions.

Hyperactivity of the oculomotor nerve causing ocular muscle spasm is possibly caused by compression of the central nerve segment.¹⁶⁾ However, impaired visual acuity and visual fields caused by vascular compression of the optic nerve^{7,14)} are considered to be due to neuropathy. The oculomotor nerve palsy in the present case was likewise a clinical manifestation of neuropathy caused by vascular compression. Generally, hyperactive dysfunction occurs when the central nerve segment with a central myelin sheath, rather than the junctional area between the central and peripheral myelin, suffers vascular compression. In contrast, neuropathy occurs when the cisternal nerve segment without a central myelin is compressed by a vessel. All these conditions can be treated by microvascular decompression.

In addition to aneurysms and vascular anomalies,⁸⁾ the posterior communicating artery may compress the oculomotor nerve.¹³⁾ PCA (P_1 portion) compression of the oculomotor nerve was demonstrated by postmortem examination in a case of suspected ophthalmoplegic migraine (OM)¹⁵⁾ and in a case of megadolichobasilar anomaly.¹⁾ These reports also indicated that microvascular decompression relieved oculomotor nerve palsy caused by vascular compression.

Spontaneous recovery from oculomotor nerve palsy is sometimes seen in cases of OM^{4,19)} or diabetes mellitus.^{3,6)} However, the present patient had no history of headache suggesting migraine and laboratory examinations found no sign of diabetes mellitus. At operation, bilateral oculomotor nerves were found to be markedly compressed, with distinct depressions due to vascular compression and peripheral abnormalities. These findings suggest that spontaneous recovery was unlikely.

Our patient first presented with oculomotor nerve paresis due to compression by the PCA, which was treated by microvascular decompression. Two months later, a similar oculomotor nerve paralysis diagnosed as due to the same mechanism occurred contralaterally and was also treated by microvascular decompression. The previous condition was probably caused by arteriosclerotic changes in the PCA, and the following condition by postsurgical adhesions of the arachnoid membrane, although not proven by the operative findings. The possibility of vascular compression should be considered when palsy of all muscles controlled by the oculomotor nerve develops rapidly, even if angiography demonstrates no abnormalities, provided that no other causes can be identified.

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Address reprint requests to: H. Nakagawa, M.D., Department of Neurosurgery, The Center for Adult Diseases, Osaka, 1-3-3 Nakamichi, Higashinari-ku, Osaka 537, Japan.