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Ptosis as Partial Oculomotor Nerve Palsy Due to Compression by Infundibular Dilatation of Posterior Communicating Artery, Visualized with Three-Dimensional Computer Graphics: Case Report

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Abstract

Oculomotor nerve palsy (ONP) due to internal carotid-posterior communicating artery (PcomA) aneurysm generally manifests as partial nerve palsy including pupillary dysfunction. In contrast, infundibular dilatation (ID) of the PcomA has no pathogenic significance, and mechanical compression of the cranial nerve is extremely rare. We describe a 60-year-old woman who presented with progressive ptosis due to mechanical compression of the oculomotor nerve by an ID of the PcomA. Three-dimensional computer graphics (3DCG) accurately visualized the mechanical compression by the ID, and her ptosis was improved after clipping of the ID. ID of the PcomA may cause ONP by mechanical compression and is treatable surgically. 3DCG are effective for the diagnosis and preoperative simulation.

Key words: computer graphics, infundibular dilatation, oculomotor nerve palsy, posterior communicating artery, ptosis

Introduction

Mechanical compression of the oculomotor nerve manifests as partial nerve palsy, usually including pupillary dysfunction.[1,2] The association of oculomotor nerve palsy (ONP) and internal carotid-posterior communicating artery (IC-PC) aneurysms has been reported,[1,3,4] whereas ONP due to compression by an infundibular dilatation (ID) of the posterior communicating artery (PcomA) is extremely rare.

We describe a rare case of progressive ptosis occurring as partial ONP due to compression by an ID of the PcomA, which improved after clipping. Three-dimensional computer graphics (3DCG) were useful for the preoperative diagnosis and simulation.

Case Presentation

I. History and presentation

A 60-year-old woman was referred to the University of Tokyo Hospital with left ptosis. She had undergone levator resection for worsening of unilateral ptosis at the age of 58 years. However, the ptosis had continued to worsen gradually. She had no history of diabetes mellitus, hypertension, or other known medical problems. On admission, neurological examination revealed left mild ptosis, but no external ophthalmoparesis or pupillary dysfunction. Magnetic resonance (MR) angiography and digital subtraction angiography demonstrated left ID of the PcomA (Fig. 1A, B), and Allcock test demonstrated collateral blood supply from posterior circulation. Fast imaging employing steady-state acquisition (FIESTA) showed a point of contact between the oculomotor nerve and the ID of the PcomA (Fig. 1C). MR angiography and FIESTA were fused with 3DCG by multimodal fusion imaging.[5] These 3DCG images showed that the ID was located rostrolateral to the oculomotor nerve and had displaced the nerve (Fig. 2). These findings suggested that her progressive ptosis was due to mechanical compression of the oculomotor nerve by the ID of the PcomA. The potential risks and benefits of the craniotomy were discussed with her, and she gave informed consent. She was scheduled for surgery.

II. Operation

The patient underwent clipping of the ID at the origin of the PcomA via left frontotemporal craniotomy with motor evoked potential (MEP) monitoring. Intraoperative

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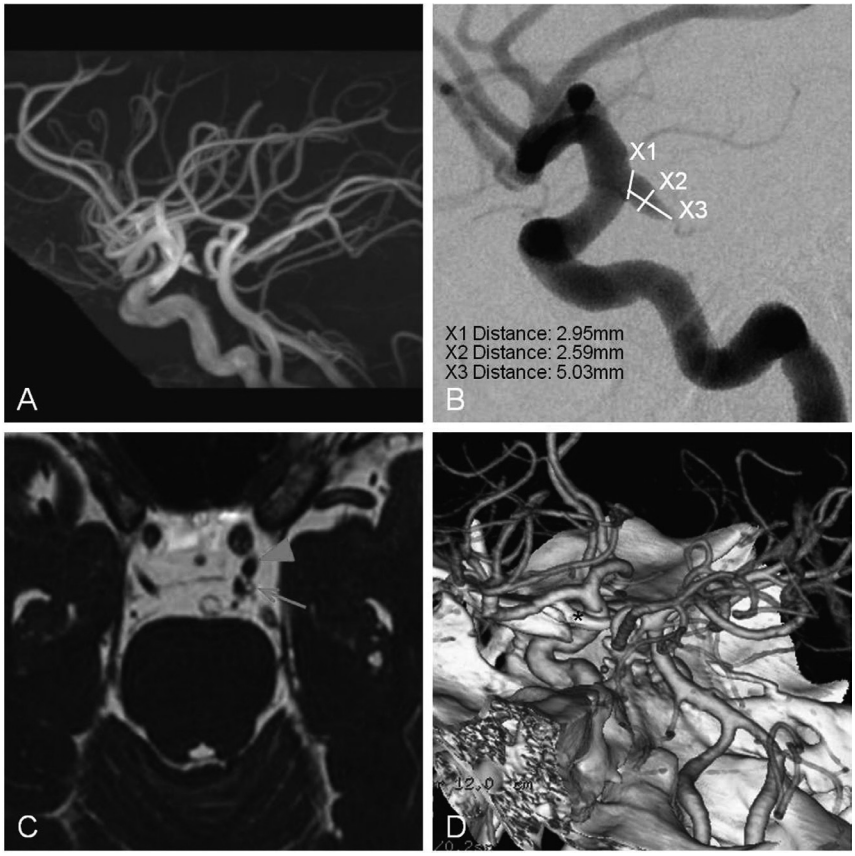

Fig. 1 Lateral preoperative magnetic resonance angiography (A) and digital subtraction angiography of the left internal carotid artery (B) showing a funnel-shaped dilatation. C: Preoperative fast imaging employing steady-state acquisition showing a contact point between the oculomotor nerve (*arrow*) and the infundibular dilatation (ID) (*arrowhead*). D: Postoperative three-dimensional computed tomography scan showing the aneurysm clip (*asterisk*) and disappearance of the ID.

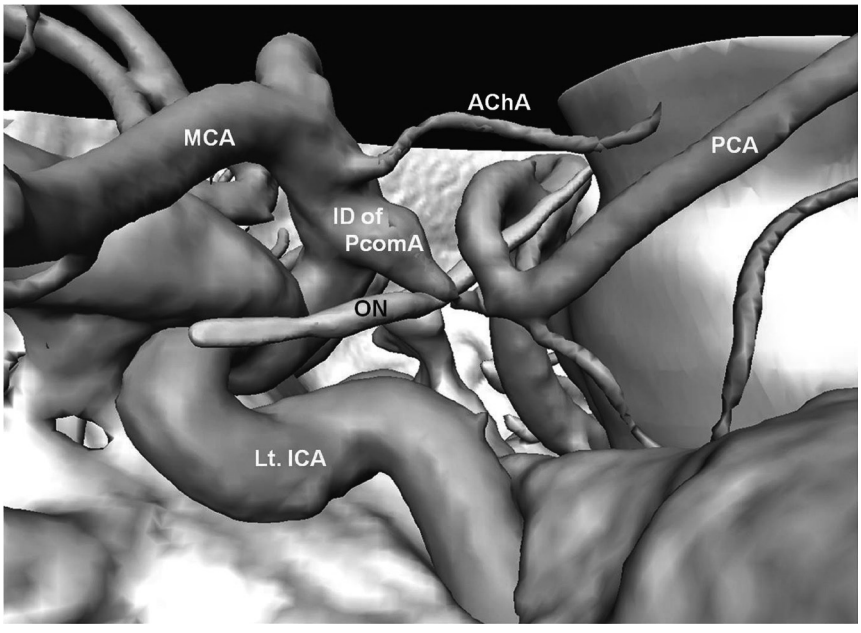

Fig. 2 Preoperative three-dimensional computer graphics image showing that the posterior communicating artery is dilated at its branch point and courses lateral to the oculomotor nerve. The infundibular dilatation has displaced the oculomotor nerve. AChA: anterior choroidal artery, ID: infundibular dilatation, Lt. ICA: left internal carotid artery, MCA: middle cerebral artery, ON: oculomotor nerve, PCA: posterior cerebral artery, PcomA: posterior communicating artery.

findings revealed the oculomotor nerve was compressed by the ID from the lateral direction, as suggested by the preoperative 3DCG images, and had an indentation (Fig. 3). The ID was tightly adhesive to the oculomotor nerve. At first, we took the microvascular decompression (MVD) of the oculomotor nerve into consideration,

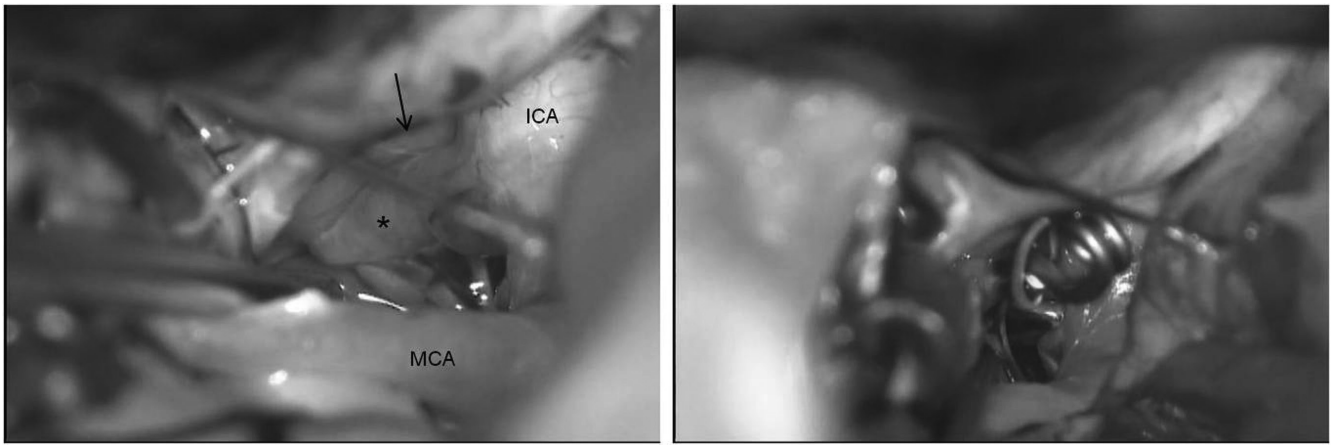

Fig. 3 Intraoperative photographs, preclipping view (*left*) showing that the infundibular dilatation (*asterisk*) had displaced the medially located oculomotor nerve (*arrow*), and post-clipping view (*right*). ICA: internal carotid artery, MCA: middle cerebral artery.

but MVD was difficult because effective decompression was not achieved due to tight adhesion. Finally, we chose the clipping of the ID at the origin of the PcomA. Intraoperative indocyanine green videoangiography after clip application detected no filling of the ID and showed anterograde patency of PcomA perforators. Intraoperative MEP amplitude did not change.

III. Postoperative course

No cerebral infarction was detected on postoperative diffusion weighted image. The patient's ptosis improved for 1 month. Postoperative CT angiography did not detect the ID (Fig. 1D).

Discussion

Oculomotor nerve, the third cranial nerve, supplies motor innervation to the levator palpebrae superioris, superior rectus, middle rectus, inferior rectus, and inferior oblique muscles, and parasympathetic innervation to the sphincter muscles of the iris and to the ciliary body.[1] ONP has various etiologies: diabetes mellitus, hypertension, multiple sclerosis, trauma, and compressive lesions such as tumors and aneurysms.[6] Compression of the oculomotor nerve in the subarachnoid space usually manifests as partial isolated nerve palsy including pupillary dysfunction.[1,6] On the other hand, only one case of unilateral ptosis has been reported as the only manifestation of the ONP due to mechanical compression.[7]

Microvascular peripheral nerve infarction caused by thickening and hyalinization of the vessel wall due to diabetes mellitus affects the core of the nerve, but spares the superficial fibers.[8] In contrast, mechanical compression of oculomotor nerve affects superficial fibers.[9] The subarachnoidal portion of the oculomotor nerve consists of a group of larger caliber somatic fibers that innervate the extraocular muscles and the levator palpebrae superioris muscle, and a group of smaller caliber parasympathetic fibers that route to the ciliary ganglion.[10] The larger somatic fibers are located in the core of the oculomotor nerve, and the smaller parasympathetic fibers are located in a superficial dorsomedial position beneath the epineurium in the subarachnoidal portion of the oculomotor nerve in the dog, monkey, and human in autopsy cases.[9] Compression of the nerve against medially placed structures caused mydriasis much more effectively than other directions in the dog and monkey.[9] Therefore, the parasympathetic fibers of the oculomotor nerve are vulnerable to mechanical compression from the medial direction.

In general, PcomA and IC-PC aneurysms are located medial to the oculomotor nerve. No PcomA was found to course lateral to the oculomotor nerve in 110 cadaver specimens.[11] This anatomical location may contribute to the tendency for compressive lesions to manifest as partial ONP including abnormal pupillary findings. Therefore, IC-PC aneurysms and IDs located lateral to the oculomotor nerve can cause ONP with atypical presentation. In the present case, preoperative 3DCG images showed that the ID of the PcomA compressed the oculomotor nerve from the rostrolateral direction, as confirmed by the operative findings. Her ptosis was improved after clipping. This history supports our supposition. Meanwhile, clipping of the ID had the potential risk of cerebral infarction related to the PcomA perforators. If feasible, MVD might be ideal. Actually we chose clipping of the ID to avoid the risk of mechanical oculomotor nerve injury and rupture of the ID due to adhesiotomy. This case took 1 month for the recovery of oculomotor nerve palsy. The duration in this case seems to be relatively longer than that of the general case of IC-PC aneurysms, which might be attributed to the pathogenic difference of the nerve injury such as the adhesion.[12]

ID is described as funnel-shaped symmetrical widening at branch points of major cerebral arteries and principally occurs at the junctions of the internal carotid artery (ICA) and PcomA.[13,14] ID at these locations is defined with a maximum of 3 mm diameter and faces the ICA, whereas the PcomA arises from the apex.[13,15,16] ID is apparent on 7–25% of otherwise normal angiograms.[14,16–18] In general, ID is considered to be a normal anatomic variant of no pathogenic significance.[19,20] On the other hand, IDs have undergone aneurysmal development and rupture.[13,19,21–23] Mechanical compression of cranial nerves by unruptured ID is extremely rare.[24] Considering that ONP is caused by IC-PC aneurysms and sometimes PcomAs,[25,26] ID of the PcomA is likely to cause ONP.

In the present case, FIESTA showed that the ID was in contact with the oculomotor nerve, but did not reveal that the ID had displaced the oculomotor nerve, since the cranial nerves and vessels showed almost same signal intensities on FIESTA.[27] In contrast, the 3DCG images were fused with gadolinium contrast-enhanced FIESTA, which has a high detectability of the cranial nerves, and time-of-flight MR angiography, which has a high detectability of the vessels, and hence exhibited this displacement accurately.[27,28] 3DCG is effective for detecting the offending vessels in neurovascular compression syndrome in patients with facial spasm and trigeminal neuralgia.[5] Therefore, 3DCG is likely to be useful for the diagnosis of oculomotor nerve mechanical compression by aneurysms, PcomAs, and IDs. Moreover, 3DCG can detect mechanical compression of the oculomotor nerve in patients with unilateral ptosis of unknown etiology. This subject requires further study.

Conclusion

We report an extremely rare case of ptosis manifesting as partial ONP due to mechanical compression by an ID of the PcomA in which 3DCG allowed accurate preoperative diagnosis and simulation. Mechanical compression by ID of the PcomA can cause partial ONP and is treatable surgically.

Conflicts of Interest Disclosure

The authors report no conflicts of interest concerning the materials or methods used in this study or the findings specified in this article.

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