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Case Report

Isolated Oculomotor Nerve Palsy after Mechanical Thrombectomy for Middle Cerebral Artery Occlusion: A Case Report

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Objective: Complications of mechanical thrombectomy (MT) should be identified and managed because they often worsen clinical outcomes. Here we present a case of post-MT embolization of the artery supplying the oculomotor nerve, which has not previously been reported as a complication of MT.

Case Presentation: An 81-year-old woman visited our hospital within 2 hours of the sudden onset of left hemiparesis and impaired awareness. MRA showed right middle cerebral artery (MCA) M1 segment occlusion and a possibly salvageable penumbra. We performed thrombectomy for right MCA occlusion with successful recanalization. In the final angiography view, the marginal tentorial artery was almost invisible. Ten hours after thrombectomy, the patient developed complete right oculomotor nerve palsy. Subsequent MRI showed ischemic lesions, but none in the oculomotor nucleus, and there were no lesions compressing the oculomotor nerve. We presume that embolization of the marginal tentorial artery caused oculomotor nerve palsy. The intracranial middle and distal portions of the oculomotor nerve are supplied by the superior branches of the inferolateral trunk and by the marginal tentorial artery.

Conclusion: Occlusion of the marginal tentorial artery can cause oculomotor nerve palsy, although this has not previously been reported. Our case suggests that neurointerventional surgeons should evaluate patency of branches of the inferolateral trunk and the meningohypophyseal trunk during the procedure of MT.

Keywords oculomotor nerve palsy, mechanical thrombectomy, complication, marginal tentorial artery, middle cerebral artery occlusion

Introduction

Mechanical thrombectomy (MT) has recently become a standard therapy for acute ischemic stroke caused by large vessel occlusion. Good outcomes depend not only on successful reperfusion but also on noticing and managing complications. One potential complication is embolization in previously unaffected territories. While this may occur anywhere, embolization of arteries supplying cranial nerves is very rare, which makes this issue difficult to identify.

Here, we describe a patient who experienced isolated oculomotor nerve palsy after MT due to embolization of an artery supplying a cranial nerve. Isolated oculomotor nerve palsy due to embolization has never been reported. We hope our report will help draw attention to the possibility of post-MT cranial nerve palsy.

Case Presentation

An 81-year-old woman with ulcerative colitis visited our hospital within 2 hours of the sudden onset of left hemiparesis. Neurological examination showed impaired awareness, left hemiparesis, left-sided hypesthesia, and left hemispatial neglect. The National Institutes of Health Stroke Scale (NIHSS) score was 16. Electrocardiogram showed atrial fibrillation.

MRA showed right middle cerebral artery (MCA) M1 segment occlusion. Diffusion-weighted images (DWI)

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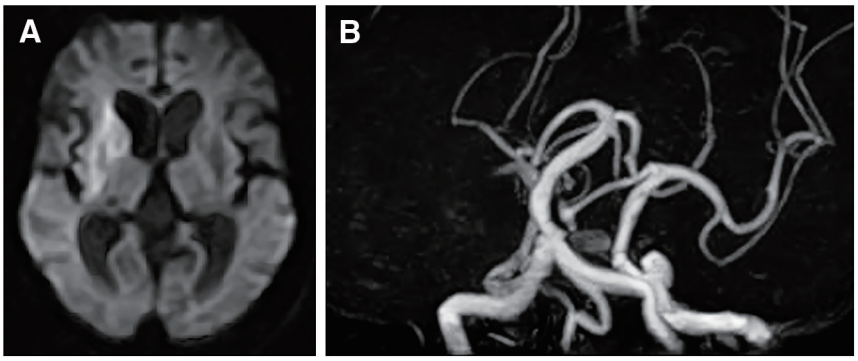

Fig. 1 Head MRI and MRA just before thrombectomy. Diffusion-weighted images show high intensity of the right basal ganglia (**A**), and MRA shows occlusion of the M1 segment of the MCA (**B**). MCA: middle cerebral artery

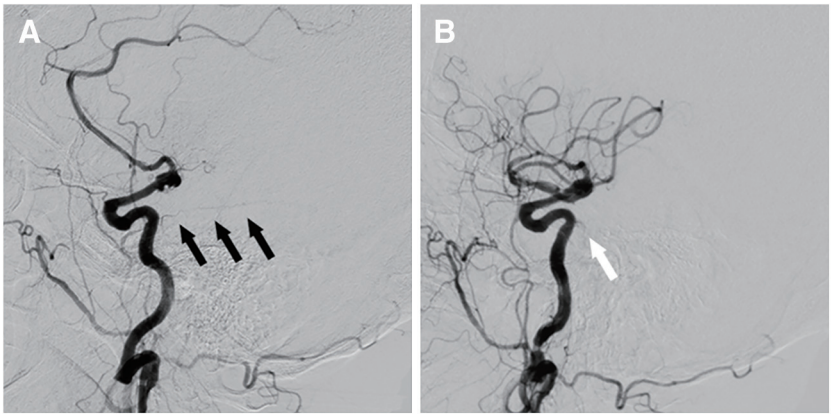

Fig. 2 Lateral angiographic view of the right carotid artery before (**A**) and after (**B**) thrombectomy. The marginal tentorial artery is shown before thrombectomy (black arrows) (**A**), but the flow in the same arterial phase become invisible distally after thrombectomy (white arrow) (**B**).

showed hyperintensity in the basal ganglia (**Fig. 1A** and **1B**). The DWI–Alberta stroke program early CT score (DWI-ASPECTS) was 8. We expected a possibly salvageable large ischemic penumbra and decided to perform MT.

The patient received an intravenous infusion of 0.6 mg/kg recombinant tissue-type plasminogen activator within 40 minutes of admission. We performed groin puncture of the right femoral artery within 1 hour of admission. The patient received an intravenous bolus of about 70 IU/kg heparin, followed by an hourly bolus of 18 IU/kg. The brachiocephalic artery arose from the lower level of the aortic arch, and the right internal carotid artery (ICA) was elongated. Thus, a 9-Fr Optimo balloon-guiding catheter (Tokai Medical, Aichi, Japan) was positioned in the right ICA but it was unstable. Control angiography showed right MCA M1 segment occlusion. Looking backward, the marginal tentorial artery arose prominently from the right ICA (**Fig. 2A**). A tri-axial system was used to deploy a Solitaire X 6.0 mm × 40 mm (Medtronic, Irvine, CA, USA) stent across the thrombus. The stent was positioned in the MCA M1 segment through a Phenom 027 catheter (Medtronic) via an ACE68 reperfusion catheter (Penumbra, Alameda, CA, USA). We performed continuous aspiration prior to intracranial vascular embolectomy (CAPTIVE), a technique for recanalization of right MCA occlusion.[1] Although the first attempt was unsuccessful, the second attempt led to complete recanalization except for the marginal tentorial artery. The flow of this artery was slower in the final angiography view (**Fig. 2B**).

One hour after MT, the patient's right pupil was slightly dilated. Ten hours later, she had a fixed dilated pupil and lateral and downward eyeball deviation with ptosis. These findings were indicative of complete oculomotor nerve palsy. MRI showed infarctions of the basal ganglia and the frontal lobe, no bleeding, and no carotid-cavernous fistula (**Fig. 3A** and **3B**). She received betamethasone 6 mg per day for a week for oculomotor nerve neuropathy. MRI on the 12th day after MT showed chronological changes in preexisting infarctions. Her pupil reactivity, ptosis, and eye movement improved gradually over the course of her hospitalization. Three months after MT, dilation and reactivity of her right pupil improved completely, and slight right ptosis and mildly impaired eye movement were apparent. Her left eye movement was intact. Left hemispatial neglect almost improved. Lethargy and left hemiparesis persisted. The modified Rankin Scale score was 5.

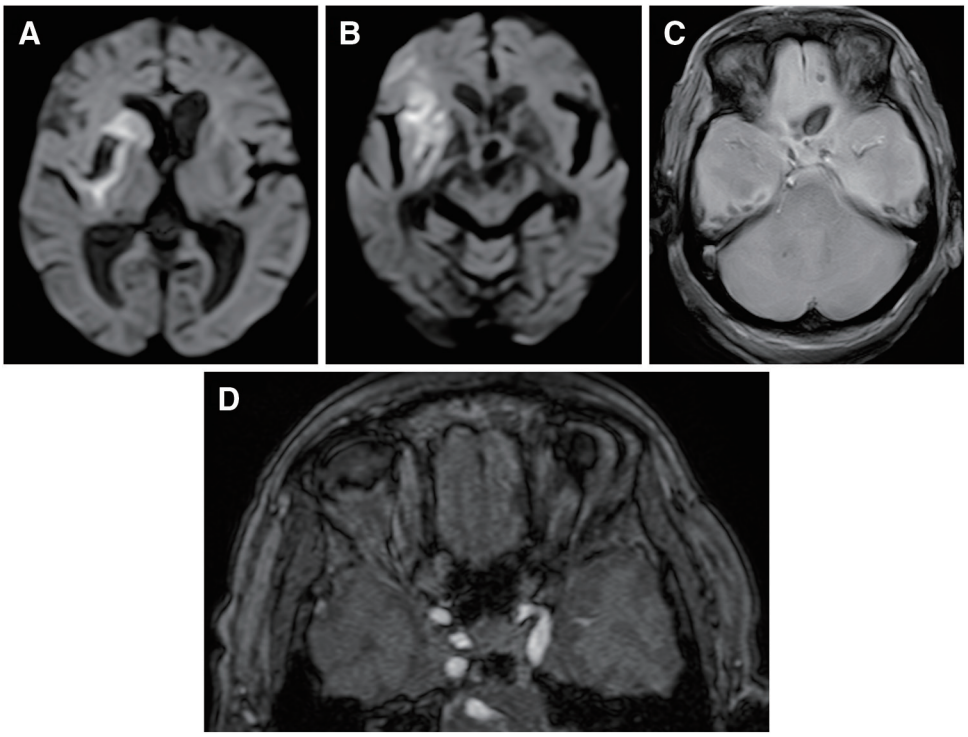

Fig. 3 Head MRI on the day after thrombectomy (**A**–**D**). Diffusion-weighted images show high intensity of the basal ganglia and frontal lobe, with an intact oculomotor nucleus (**A** and **B**). T2*WI shows no hematoma and nothing compressing the oculomotor nerve (**C**). MRA shows no findings indicating an arteriovenous fistula, including hyperintensity in the cavernous or intracavernous sinus and in the superior ophthalmic vein (**D**). T2*WI: T2* weighted image

Discussion

Complications of MT often occur and may worsen clinical outcomes or lengthen hospitalizations. Randomized controlled trials have reported that the incidence of device- or procedure-related complications ranges from 4% to 29%.[2] Possible complications include groin hematoma, iatrogenic dissection, vasospasm, intracranial hemorrhage, and embolization within the same or a new vascular territory.[2] Among previously described complications, oculomotor nerve palsy may occur due to intracranial hemorrhage or compression by a dissecting aneurysm, but it is unlikely to be caused by ischemic injury secondary to embolization. Because no MRI findings in our case suggested dissection or hemorrhage, oculomotor nerve palsy was presumably caused by embolization of the marginal tentorial artery.

The oculomotor nerve emerges from the inner side of the cerebral peduncle, passes through the interpeduncular cistern and the upper part of the cavernous lodge, penetrates the orbit through the superior orbital fissure, and divides into a superior and inferior branch to innervate the ocular muscles, except for the lateral rectus and superior oblique muscles.[3] The proximal intracranial portion of the oculomotor nerve is supplied by posterior circulation, mainly the thalamoperforating artery that arises from the posterior communicating artery, and supplementary branches that arise directly from the posterior cerebral, posterior communicating, superior cerebellar, and basilar arteries. The intracranial middle and distal portions of the oculomotor nerve are consistently supplied by the superior branches of the inferolateral trunk and the marginal tentorial artery.[4]

The marginal tentorial artery usually originates as the terminal branch of the meningohypophyseal trunk.[5] It passes through the roof of the cavernous sinus and courses posterolaterally along the free edge of the tentorium.[5] It sends branches to the oculomotor and trochlear nerves and to the medial portion of the Gasserian ganglion.[5]

We presume that embolization of the marginal tentorial artery caused oculomotor nerve palsy in our case. Slow flow in the marginal tentorial artery indicated a narrowed origin or distal branch occlusion, including the branches, of the artery. As the possible mechanisms, it was considered that the marginal tentorial artery was occluded by the fragments released

from the main thrombus during the thrombectomy. Another possibility is that the artery was occluded by the thrombosis due to the impaired endothelial function, which was induced from the mechanical stress by the catheter and the stent. We hypothesize that our patient's oculomotor nerve was more dependent on the marginal tentorial artery, and it was the reason why the trochlear nerve and Gasserian ganglion were intact. Isolated ocular motor nerve palsy, which is defined as dysfunction of a single ocular motor nerve (oculomotor, trochlear, or abducens), is often caused by microvascular ischemia.[6] This suggests that the susceptibility of the cranial nerves to ischemia may differ among patients.

Oculomotor nerve palsy due to ischemic injury is very rare and was reported to have occurred in cases of intracranial or extracranial ICA dissection.[7–9] However, oculomotor nerve palsy was not necessarily caused by ischemic injury in these reports, because it can arise from compression secondary to ICA dissection. No previous reports have identified embolization of the supplying arteries as the cause of oculomotor nerve palsy.

Our patient had an elongated access route, which made it difficult to stabilize the catheters and to control the microcatheter and the stent retriever. As a result, a longer procedure time and multiple attempts were necessary, both of which might increase the risk of complications, and it was important to pay careful attention to angiographic findings. We should have considered embolization of the marginal tentorial artery as a cause of oculomotor nerve palsy, even though this was not reported by previous studies. We considered careful follow-up to be necessary in this case.

Conclusion

Our case is the first in which embolization of the supplying artery occurred after MT. This finding suggests that neurointerventional surgeons should carefully evaluate patency of branches of the inferolateral trunk and the meningohypophyseal trunk during the procedure of MT. Careful follow-up is required when occlusion occurs.

Disclosure Statement

The authors declare that they have no conflicts of interest.

References

1) McTaggart RA, Tung EL, Yaghi S, et al. Continuous aspiration prior to intracranial vascular embolectomy (CAPTIVE): a technique which improves outcomes. *J Neurointerv Surg* 2017; 9: 1154–1159.
2) Pilgram-Pastor SM, Piechowiak EI, Dobrocky T, et al. Stroke thrombectomy complication management. *J Neurointerv Surg* 2021; 13: 912–917.
3) Koskas P, Héran F. Towards understanding ocular motility: III, IV and VI. *Diagn Interv Imaging* 2013; 94: 1017–1031.
4) Hendrix P, Griessenauer C, Foreman P, et al. Blood Supply of the Cranial Nerves. In: Nerves and Nerve Injuries. Academic Press, Cambridge, 2015, 427–438.
5) Banerjee AD, Ezer H, Nanda A. The artery of Bernasconi and Cassinari: a morphometric study for superselective catheterization. *AJNR Am J Neuroradiol* 2011; 32: 1751–1755.
6) Kung NH, Van Stavern G. Isolated ocular motor nerve palsies. *Semin Neurol* 2015; 35: 539–548.
7) Jung S, Yoon C. A rare case of oculomotor nerve palsy after endovascular treatment in a patient with internal carotid artery dissection. *J Stroke Cerebrovasc Dis* 2021; 30: 105555.
8) Mokri B, Silbert PL, Schievink WI, et al. Cranial nerve palsy in spontaneous dissection of the extracranial internal carotid artery. *Neurology* 1996; 46: 356–359.
9) Campos CR, Massaro AR, Scaff M. Isolated oculomotor nerve palsy inspontaneous internal carotid artery dissection: case report. *Arq Neuropsiquiatr* 2003; 61: 668–670.