Case Report

A Case of Complete Recovery of Fluctuating Monocular Blindness Following Endovascular Treatment in Internal Carotid Artery Dissection

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Background: Monocular blindness may appear as the first symptom of internal carotid artery dissection (ICAD). However, there have been no reports that monocular visual loss repeatedly occurs and disappears in response to postural change in ICAD. *Methods:* A 33-year-old woman presented with transient monocular blindness (TMB) following acute-onset headache. TMB repeatedly occurred in response to postural change. Two days later, she experienced transient dysarthria and right hemiparesis in upright position. Pupil size and light reflex were normal, but a relative afferent pupillary defect was positive in the left eye. Diffusion-weighted imaging showed no acute lesion, but perfusion-weighted imaging showed perfusion delay in the left ICA territory. Digital subtraction angiography demonstrated a false lumen and an intraluminal filling defect in proximal segment of the left ICA. *Results:* Carotid stenting was performed urgently. After carotid stenting, left relative afferent pupillary defect disappeared and TMB was not provoked anymore by upright posture. At discharge, left visual acuity was completely normalized. *Conclusions:* Because fluctuating visual symptoms in the ICAD may be associated with hemodynamically unstable status, assessment of the perfusion status should be done quickly. Carotid stenting may be helpful to improve the fluctuating visual symptoms and hemodynamically unstable status in selected patient with the ICAD. **Key Words:** Transient monocular blindness—internal carotid artery—dissection—stenting.

Various visual symptoms and signs, such as Horner syndrome, scintillations, transient monocular blindness (TMB), permanent visual loss, and ischemic ocular syndrome, can be caused by internal carotid artery dissection (ICAD).[1,2] TMB has been reported in 6% to 38% of patients with ICAD.[1] However, to the best of our knowledge, there have been no reports that TMB caused by ICAD waxes and wanes in response to postural change.

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We report a case showing a complete recovery of fluctuating TMB following endovascular treatment in patient with ICAD. Our institutional review board approved this case report: patients' informed consent was not required because this case report was retrospective.

Case Report

A 33-year-old woman presented with TMB in left eye following acute-onset headache. Monocular blindness developed episodically only in the upright position and was relieved quickly by lying down. Two days later, transient dysarthria and right hemiparesis occurred in upright position and were alleviated by lying down. She was transferred to our emergency center for further evaluation. She denied any trauma and medical history. Her vital signs were stable. Snellen chart revealed a reduced visual acuity of the left eye (OS 20/30 and OD 20/17) in a supine position. Pupil size and light reflex were normal, but there was a relative afferent pupillary defect (RAPD) in the left eye. Photostress recovery test showed a prolonged recovery time in the left eye. Fundoscopic examination was normal bilaterally. In a supine position, she did not show any focal neurologic deficits including monocular visual loss.

Diffusion-weighted imaging (DWI) showed no diffusion restriction (Fig 1, A), but perfusion-weighted imaging (PWI) showed a perfusion delay in the left ICA territory. Time-to-peak map showed a delayed time to peak more than 4 seconds in the left hemisphere compared to those in the right (Fig 1, B). Especially, left border-zone area showed a mean transit time delay more than 6 seconds (Fig 1, C). Susceptibility-weighted imaging (SWI) showed prominent multiple hypointense vessels in the left hemisphere (Fig 1, D). Contrast-enhanced magnetic resonance (MR) angiography showed a focal stenosis of the left proximal ICA (Fig 1, E). Time-of-flight MR angiography showed a faint flow through the left middle cerebral artery and ICA and numerous cortical branches of the left posterior cerebral artery compared to those of the right (Fig 1, F). Digital subtraction angiography (DSA) demonstrated an intraluminal filling defect and a false lumen in the proximal segment of the left extracranial ICA, which was consistent ICAD (Fig 1, G). DSA showed collateral blood flow via anterior communicating artery and left ophthalmic artery.

The patient was medicated with 75 mg of clopidogrel and 100 mg of aspirin in another hospital for 2 days before the procedure. DSA was performed with a 6Fr, guiding catheter (Flexor Shuttle Select; Cook Medical, Bloomington, IN). A distal protection device (Filter wire EZ embolic protection device; Boston scientific, Fremont, MA) was placed under roadmap guidance into the distal cervical ICA. Prestenting ballooning was performed using the 5 mm × 40 mm, Sterling Monorail Balloon (burst pressure of 6 atm, Boston scientific, Fremont, MA), but the dissection lesion was not improved. The Carotid WALLSTENT (7 mm × 50 mm, Boston scientific, Fremont, MA) was placed across the dissection segment of left ICA. Poststenting ballooning was performed using the 3 mm × 20 mm, Catheter PTCA Balloon Ryujin (burst pressure of 14 atm, Terumo, Tokyo, Japan). Poststenting angiography demonstrated good apposition of the stent throughout the dissection segment of the left ICA with no residual stenosis (Fig 1, H). Restoration of normal flow through the ICA and the intracranial vessels were identified on the final angiographic runs (Fig 1, I).

After the procedure, the left RAPD disappeared immediately. A day after the procedure, the decreased visual acuity of the left eye was normalized. Also, TMB did not develop in upright posture. The patient was maintained on a daily regimen of aspirin (100 mg) and clopidogrel (75 mg) for 6 months.

Discussion

TMB is not an uncommon manifestation in ICAD, but fluctuating TMB in response to postural change has not been reported previously in ICAD.[1,2] There are 2 possible mechanisms for explaining the TMB in ICAD: 1) embolic mechanism and 2) hypoperfusion mechanism.[1,3,4] In our case, the ophthalmoscope examination showed no occlusion of the retinal arteries by embolus: therefore, the embolic mechanism could be ruled out. The hypoperfusion mechanism in our case could be backed by neurologic and MR findings. The monocular blindness was not developed in supine position, but the RAPD of the left eye was observed, which suggested that retina or optic nerve may be affected by a decreased ocular blood flow due to ICAD even in supine position. MR imaging findings also support the hypoperfusion mechanism. DWI showed no diffusion restriction, but PWI showed a marked perfusion delay in the left ICA territory; it is often called as "a total DWI/PWI mismatch."[5] Especially, perfusion delay was significant in the left border-zone area, which could explain why right hemiparesis developed in upright posture and disappeared in supine position. SWI showed the prominent multiple hypointense vessels in the left hemisphere compared to the right hemisphere; it means an increased ratio of deoxyhemoglobin/oxyhemoglobin by increased oxygen demand in the left hypoperfused hemisphere.[6] Therefore, the patient was considered to be in hemodynamically unstable condition even in supine position and have a high risk of neurologic deterioration. The changing position from supine to upright posture may aggravate a cerebral hypoperfusion caused by ICA occlusion. The collateral flow may be helpful to overcome the hypoperfusion and prevent ischemic symptoms of the left hemisphere in upright posture. But, considering the left positive RAPD even in supine position, the left ocular hypoperfusion may be more severe