Central Retinal Artery Occlusion after the Endovascular Treatment of Unruptured Ophthalmic Artery Aneurysm: A Case Report and a Literature Review

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OphA aneurysm that complicated by OphA occlusion with blindness after endovascular coiling and successfully recanalized by intra-arterial thrombolysis.

Case Report

Presentation

A 62-year-old female diagnosed by magnetic resonance angiography (MRA) in 2007 to have a right OphA aneurysm measuring about 5 mm in diameter. Follow-up MRA in 2015 showed enlargement of the aneurysm. Catheter angiography was performed and demonstrated asymptomatic increase in size of right OphA aneurysm to measure 6.9 × 5.9 × 4.6 mm with wide neck measuring 6 mm and denoted that the right OphA was originating from the body of the aneurysm (Fig. 1).

Intervention

Therapeutic decision was reached through multidisciplinary deliberation of both neurosurgeons and nonsurgical neurointerventionalists, and informed consent was obtained from the patient and her family. Balloon-assisted coiling was elected to attempt dense packing of the wide necked aneurysm. A 75 mg of clopidogrel and a 100 mg of aspirin were administered 4 days before the procedure. After general anesthesia and femoral puncture, systemic heparinization was started with a loading dose of 5,000 units and maintained with a dose of 1,000 units hourly and the activated clotting time (ACT) kept above 250 s throughout the procedure. The right femoral artery was used to place a 6F guiding catheter (Fubuki; ASAHI-INTECC, Aichi, Japan) indwelled into the right cervical ICA. Excelsior SL-10 microcatheter (Stryker, Kalamazoo, MI, USA) and one balloon catheter (4 × 10 mm Scepter C balloon; Microvention, Tustin, CA, USA) were then positioned in the ICA clinoid segment. Balloon test occlusion (BTO) of the OphA was performed using a balloon catheter. Rapid intervention is required for recovering visual disturbance in such a situation.

Keywords: central retinal artery, complication, endovascular therapy, local intra-arterial fibrinolysis, ophthalmic artery aneurysm

Introduction

Endovascular treatment of the internal carotid artery (ICA) paraclinoid aneurysms has been proposed as an alternative to surgical clipping.1–5) Nevertheless, the risk of impaired vision due to unexpected thromboembolism or occlusion of the ophthalmic artery (OphA) after coiling is still a major concern.6–8) Incidence of new visual deficits in paraophthalmic aneurysms treated surgically ranged from 4.8 to 7.5%.9,10) The neurointerventional therapies have done little to reduce these risks as the incidence of visual complications following endovascular coiling of paraophthalmic aneurysms are estimated to be approximately 5%.10,11) Rouchaud et al. found that the coverage of the OphA by flow diverters is not a harmless treatment because 17.9% of patients had permanent ophthalmic complications. These complications can be mild or severe, but some are only detected with a complete and extensive ophthalmic examination, which was not performed in most previous studies.12) We present a case of an
the right OphA. The right external carotid artery (ECA) angiography revealed the disappearance of the retinochoroidal blush as seen in the OphA BTO. These findings suggested thromboembolic event occurring through the right OphA (Fig. 3).

Endovascular procedure for the right OphA occlusion was performed under local anesthesia and systemic heparinization which started with a loading dose of 5,000 units and was maintained with a dose of 1,000 units hourly keeping the ACT above 300 s. A 6F-guiding catheter placed into the right cervical ICA and a carnelian PIXIE (Tokai Medical Products, Aichi, Japan) with its distal tip placed close to the orifice of the right OphA confirmed by selective angiography. Mechanical disruption of the thrombus using a micro-guidewire and local intra-arterial fibrinolysis (LIF) using 1,20,000 units Urokinase was performed with evident recanalization of the OphA with a normal anterograde flow until the third segment (Fig. 4). At the same time the visual acuity of the right eye was improving until the patient restore normal vision through. Heparin reverse was not carried out the final ACT was 238 s. Postoperative systemic heparinization was continued for 48 h. Dual antiplatelet therapy was continued 6 months after the procedure.

Follow-up finding

The patient maintained normal visual acuity and the fundus photograph showed no ischemic lesion in her right retina.

Discussion

Periprocedural thromboembolic complications from endovascular treatment of intracranial aneurysms are estimated to occur in 2–15% of patients. Aggressive treatment of acute intraprocedural thrombus formation with intra-arterial or intravenous administration of fibrinolytics

patient had no neurological deficit, and the patient was transferred to the recovery room.

Fifteen minutes later, the patient complained from blurred vision in her right eye. Although continuous heparin infusion was started, her visual disturbance progressed rapidly to complete blindness and total loss of light reflex in about 1 h after the endovascular procedure. The patient transferred emergently for cerebral angiography which demonstrated the complete occlusion of not only the treated aneurysm but also

Fig. 1 Right internal carotid artery three-dimensional reconstructed angiogram demonstrates a wide necked ophthalmic artery aneurysm with the ophthalmic artery originating from the body of the aneurysm.

Fig. 2 (A, B) Right common carotid artery lateral angiograms reveal (A) anterograde filling of the ophthalmic artery in the mid arterial phase and (B) choroidoretinal blush in the late arterial phase (arrow). (C, D) Right common carotid artery lateral angiograms with balloon test occlusion reveal (C) complete blockage of the anterograde internal carotid arterial flow, retrograde filling of the ophthalmic artery via the external carotid artery branches in the mid arterial phase (arrow), and (D) choroidoretinal blush in the late arterial phase (arrow).

Fig. 3 (A) Right internal carotid artery lateral angiogram immediately after endovascular coiling reveals patent ophthalmic artery. (B) Right internal carotid artery lateral angiogram after the onset of the visual symptoms reveals ophthalmic artery occlusion. (C, D) Right external carotid angiograms after the onset of the visual symptoms reveal (C) no retrograde flow to the ophthalmic artery and (D) absent of choroidoretinal blush in the late arterial phase.
Conventional treatment modalities for CRAO include ocular massage, anterior chamber paracentesis, intraocular pressure-lowering agents (e.g., mannitol, acetazolamide, and topical agents), hyperbaric oxygen, anticoagulants, and hemodilution, although efficacy regarding visual outcome of these treatments have not been clearly proved. Several case series and a meta-analysis indicated that LIF may improve visual outcome in patients with CRAO. Potential side effects of LIF include hemorrhagic or ischemic complication of the brain. For this reason, the benefit of LIF has also not generally been widely reported. Arnold et al. proved the efficacy of LIF for the acute CRAO within 6 h after the onset compared with conventional therapy. They injected Urokinase, in a dose ranging from 1,00,000 units to 10,00,000 units (mean dose 6,77,000 units) manually through a microcatheter placed in the proximal segment of the OphA. Prospective randomized multicenter clinical trial in Europe to compare treatment outcome after conservative standard treatment with LIF using recombinant tissue plasminogen activator for patients with CRAO within 20 h after the onset was stopped after the first interim analysis because of apparently similar efficacy and the higher rate of adverse reactions in LIF group. These data suggest that LIF for CRAO should be performed by skilled neurointerventionalists as soon as possible after the onset.

In our case, the BTO revealed intact collateral circulation between the ECA and OphA and the immediate post-coiling angiography showed preserved anterograde flow of the OphA. However, rapid reverse of systemic heparinization by protamine sulfate might induce progressing thrombosis in coiled intraaneurysmal sac, which involved in the origin of the right OphA. The progressing thrombosis reached the third segment of the OphA, and that is the reason the collateral flow from the ECA could not work. The patient suffered from complete visual loss in the right eye. Urokinase infusion through the microcatheter placed in the origin of the right OphA successfully dissolved the thrombus and made the OphA recanalized. The patient’s visual acuity completely recovered.

**Conclusion**

We need to highlight that during endovascular coiling of the OphA aneurysm the risk of visual loss due to thromboembolic complications cannot be ignored despite good retrograde flow during OphA occlusion test by a balloon catheter. Rapid endovascular intervention is required for recovering of visual disturbance in such a situation.

**Conflicts of Interest Disclosure**

The authors have no disclosure to report.

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