Case Report

Ocular ischemic syndrome due to severe internal carotid artery stenosis improved by intracranial stent placement: A case report

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ABSTRACT

Background: Treatment of cervical internal carotid artery (ICA) stenosis has contributed to the improvement of ocular ischemic syndrome. However, there have been few cases of visual impairment caused by ocular ischemic syndrome due to intracranial ICA stenosis, which improved through intracranial stent placement.

Case Description: A 76-year-old man presented with right-sided paralysis. Radiographic examination revealed severe stenosis of the left intracranial ICA (distal cavernous-infracarotid portion) and a watershed infarction of the left cerebral hemisphere. Conservative therapy including antiplatelet drugs was initiated, but severe visual acuity disturbance in his left eye occurred 1 month after onset. The antegrade ocular artery flow recovered after urgent intracranial stent placement, and his vision improved immediately after the procedure.

Conclusion: Visual impairment presenting as ocular ischemic syndrome can occur due to severe stenosis of the intracranial ICA, and treatment of these lesions could improve the symptoms.

Keywords: Internal carotid artery stenosis, Intracranial, Ocular ischemic syndrome, Stent placement

INTRODUCTION

Ocular ischemic syndrome, including visual impairment, amaurosis fugax, and pain, occurs secondary to severe atherosclerotic carotid artery stenosis, resulting in 50% reduction in ipsilateral perfusion pressure. Treatment of cervical internal carotid artery (ICA) stenosis, such as carotid artery stenting (CAS) and carotid endarterectomy (CEA), has contributed to the improvement of ocular ischemic syndrome. Here, we describe a case of an ocular ischemic syndrome due to intracranial ICA stenosis which improved with intracranial stent placement.

CASE REPORT

A 76-year-old man presented with right-sided paralysis. Cerebral magnetic resonance imaging revealed a watershed infarction in the left hemisphere, and cerebral angiography revealed severe stenosis of the left intracranial ICA (distal cavernous-infradicaliculid portion), without stenosis of the cervical ICA [Figures 1 and 2a]. Collateral flow through anterior communicating artery was absent,
but through posterior communicating artery was confirmed insufficiently. Single-photon emission computed tomography (SPECT) demonstrated that his left cerebral hemisphere reserve was reduced. He immediately started conservative treatment with antiplatelet drugs and rehabilitation, and extracranial-intracranial bypass was planned within 3 months after the ictus. However, he presented with severe visual acuity disturbance in the left eye 1 month after onset, and ophthalmic evaluation revealed a central retinal artery obstruction in the left eye, and the ocular circulation time evaluated by arm retinal circulation time (A-R time) was prolonged.

Decreased blood flow to the ocular artery due to stenosis progression of the left ICA was suspected. Urgent cerebral angiography performed 2 days after the occurrence of visual impairment demonstrated that the left ICA was almost completely occluded, and the ocular artery was confirmed through anastomosis from the external carotid artery [Figure 1b]. Percutaneous angioplasty (PTA) was performed to improve visual acuity. The ICA flow improved immediately after PTA but decreased thereafter. After several attempts at PTA, an intracranial stent (Wingspan, Stryker Neurovascular, Salt Lake City, Utah, USA) was placed to maintain the improved flow of the ICA [Figure 1c]. After stent placement, the antegrade ocular artery flow recovered, and his vision improved immediately.

The A-R time was reduced from 54 s to 11 s, and retinal angiography also showed improvement in the visualization of blood vessels [Figure 2a and b]. In addition, SPECT showed improvement in the left cerebral blood flow reserve [Figure 3a-f], and he was discharged 2 weeks later without deficits. During the 1-year follow-up, his visual acuity and ICA flow remained stable.

**DISCUSSION**

Ocular ischemic syndrome is caused by ocular hypoperfusion due to stenosis or occlusion of the cervical ICA. There have been cases of improvement in the blood flow to the retina and ocular ischemic syndrome, such as CAS and CEA, after the treatment of the cervical ICA stenosis. The positive effect of CAS and CEA on the compromised ophthalmic artery flow in patients with ocular ischemic syndrome is well established. Regarding intracranial ICA stenosis, Marx et al. reported three cases of percutaneous transluminal angioplasty and stenting (PTAS) for ocular ischemic syndrome due to the intracranial ICA stenosis, but those were precavernous and proximal cavernous portions of the ICA. Therefore, there have been no cases of ocular ischemic syndrome due to stenosis of the distal cavernous-infraclinoid portion of the ICA. In the present case, the antegrade ocular artery flow recovered and his vision improved immediately after treatment. It is reasonable to speculate that improvement in blood flow in the ICA contributes to the increased blood flow in the ocular artery, irrespective of whether it is cervical or intracranial ICA.

To evaluate ocular blood flow and retinal circulation, the A-R time measured by fluorescein fluorescence fundus examination was used in this case. In patients with severe cervical ICA stenosis, the A-R time was prolonged and was improved after CAS at a high rate. In the present case,
the A-R time significantly improved after intracranial stent placement. We speculate that A-R time might be strongly associated with intracranial carotid artery stenosis and is predictive of ocular ischemic syndrome.

Complications related to PTA have been reported: cerebral embolism, vessel dissection, hemodynamic compromise, and cerebral hyperperfusion syndrome. Regarding visual acuity, angioplasty of the ICA could increase ocular blood flow, but intraocular pressure elevation due to increased aqueous humor might result in decreased visual acuity. Several reports of glaucoma deterioration after CAS and CEA have been published. Although the visual acuity of the patient improved after the treatment in this case, ocular complications might occur with a similar condition in the intracranial carotid artery stenosis.

The Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis trial supports the use of aggressive medical management rather than PTAS with the Wingspan system in patients with intracranial arterial stenosis. Although there have been cases of intracranial ICA stenosis that improved transient memory impairment and hemiparesis, there is no case in literature elucidating improvement in visual impairment due to ocular ischemic syndrome after PTAS treatment for the distal cavernous-infraclinoid portion of the ICA. In this case, we provided the best medical treatment; however, severe visual acuity disturbance developed, and PTAS was successfully performed with improvement in the ocular symptoms. PTAS could be an option for salvage therapy for ocular ischemic syndrome due to intracranial ICA stenosis.

CONCLUSION

The improvement in blood flow in the ICA contributes to an increased blood flow in the ocular artery irrespective of whether it is cervical or intracranial ICA in patients with ocular ischemic syndrome. PTAS might be considered as a treatment option for ocular ischemic syndrome due to intracranial ICA stenosis when the best medical treatment fails.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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Figure 3: Preoperative single-photon emission computed tomography (SPECT) demonstrated that his left cerebral hemisphere reserve was reduced (a-c). Postoperative SPECT showed improvement in the left cerebral blood flow reserve (e-g). (a and d) Cerebral blood flow (CBF) at rest; (b and e) CBF after acetazolamide challenge (Diamox CBF); (c and f) cerebral vascular reserve defined as (Diamox CBF-resting CBF)/resting CBF×100%).
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