Phacoemulsification and intraocular lens implantation for cataract induced by ocular ischemic syndrome: 30-month follow-up

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We describe a 54-year-old patient with cataract induced by ocular ischemic syndrome who had phacoemulsification and intraocular lens implantation in 1 eye and, 30 months later, in the fellow eye. The visual acuity, visual field, fluorescein angiography of the fundus, and carotid Doppler ultrasound were recorded and compared during the 30 months. Although the carotid artery stenosis and retinal ischemia progressed, the visual acuity in both eyes improved after cataract surgery. The patient was very satisfied with the result.

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Ocular ischemic syndrome is a rare condition caused by ocular hypoperfusion due to atherosclerosis of the carotid arteries. It manifests as visual loss, orbital pain and, frequently, changes in the visual field, and various anterior and posterior segment signs.1 Anterior segment signs include iris neovascularization and secondary neovascular glaucoma and cataract.2,3 Posterior segment signs include narrowed retinal arteries, perifoveal telangiectasia, dilated retinal veins, and midperipheral retinal hemorrhages.2,3 We report the 30-month follow-up of a patient with carotid artery stenosis, cataract, and narrowed retinal vessels induced by ocular ischemic syndrome following phacoemulsification and intraocular lens (IOL) implantation.

CASE REPORT

In September 2011, a 54-year-old man presented to our clinic with sudden blurred vision without orbital pain in the left eye for 3 weeks. The patient had no medical history, including diabetes, hypertension, or trauma, and used no systemic medications. The uncorrected distance visual acuity (UDVA) was 20/40 in the right eye and 20/100 in the left eye. The uncorrected near visual acuity (UDVA) was 20/20 in the right eye and 20/100 in the left eye. The visual failure appeared to be due to the development of cataract or the ischemia of the retina.

Following the biometric measurements, the patient was treated successfully with phacoemulsification and IOL implantation. The UDVA was 20/20 on the first postoperative day.
The patient was referred to a vascular surgeon immediately after the fundus fluorescein angiography results were obtained. Considering the patient’s description of symptoms and the results of the ophthalmic examination and the carotid Doppler ultrasound, the vascular surgeon requested blood pressure, blood glucose, and serum lipid tests. All the results were normal. The vascular surgeon thought the ocular ischemia was strongly linked to the carotid artery stenosis, a type of symptomatic carotid artery stenosis infrequently seen. Aspirin, 75 mg per day, was prescribed to reduce the potential for stroke. As the patient’s serum lipid level was normal, no statin therapy was prescribed. The patient was advised to stop smoking, reduce alcohol intake, increase physical exercise, and adopt a low-fat diet. He was told to monitor his blood pressure, blood glucose, and serum lipid frequently.

In the 10th postoperative month, the visual-field loss had progressed in both eyes: temporal arcuate in the right eye (mean deviation −7.15 dB) and superior nasal arcuate in the left eye (mean deviation −8.00 dB) (Figure 2, A). The UDVA was 20/50 in the right eye and remained 20/20 in the left eye.

In March 2014, the patient presented to us with complaints of worsening vision in the unoperated eye. The UDVA was 20/70 in the right eye and 20/20 in the left eye. Posterior subcapsular lens opacity had developed.

Figure 1. Examination results in 2011. A: Fundus photograph of both eyes. B: The prolonged arm-to-retina circulation time (13 seconds) and retinal filling time (2 minutes and 5 seconds) was presented in fundus fluorescein angiography and no bone spicule-shaped pigment deposit was seen. C: Diffuse scotomas in the right eye (mean deviation of −6.91 dB) and nasal arcuate in the left eye (mean deviation of −7.46 dB). D: Blood-flow disturbance in the carotid artery secondary to carotid artery stenosis on both sides. E: A 5.4 mm × 3.1 mm atherosclerosis plaque was found in the right subclavian artery.
in the right eye. On ophthalmoscopy, arteries and veins were narrowed and whitening (Figure 2, B). A horizontally oriented optical coherence tomography (OCT) of the macula showed decreased thickness (179 µm in the right eye and 169 µm in the left eye), indicating ischemic damage to the nerve-fiber layer (Figure 2, C). The visual field loss had also progressed: temporal arcuate in the right eye (mean deviation \(-8.17\) dB) and superior inferior arcuate in the left eye (mean deviation \(-12.63\) dB) (Figure 2, D). On fundus fluorescein angiography, the arm-to-retina circulation time (22 seconds) and retinal filling time (2 minutes and 22 seconds) were longer than they had been 30 months earlier (Figure 3, A). Color Doppler ultrasound still showed blood-flow disturbance in the carotid artery secondary to carotid artery stenosis on both sides (Figure 3, B). The size of the right subclavian artery atherosclerosis plaque had increased to \(8.3\) mm \(\times\) \(3.2\) mm (Figure 3, C).

The patient was treated successfully with phacoemulsification and IOL implantation for cataract in the right eye. The UDVA was 20/25 on the first postoperative day.

With all the vascular treatments, the carotid artery stenosis and right subclavian artery atherosclerosis progressed slowly during the 30-month follow-up. The patient did

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**Figure 2.** Examination results in 2012 and 2014. A: Temporal arcuate in the right eye (mean deviation \(-7.15\) dB) and superior nasal arcuate in the left eye (mean deviation \(-8.00\) dB) in 2012. B: Both arteries and veins became narrowed and white in 2014. C: Optical coherence tomography of the macula demonstrated decreased thickness (179 µm in the right eye and 169 µm in the left eye) in 2014. D: Temporal arcuate in the right eye (a mean deviation \(-8.17\) dB) and superior inferior arcuate (mean deviation \(-12.63\) dB) in the left eye in 2014.
not consider a carotid endarterectomy as he feared the surgical risk. He has continued the medical management until the present.

DISCUSSION

The blood supply to the eye comes from the retinal central vascular system of the ophthalmic artery and the ciliary vascular system. The ophthalmic artery stems from the ipsilateral internal carotid artery. If occlusion or stenosis occurs in the carotid artery, the blood perfusion to the ophthalmic artery becomes insufficient, leading to signs and symptoms of anterior and posterior ocular ischemia. The mean age of patients with ocular ischemic syndrome has been reported as 65 years, and men are affected twice as often as women. In 20% of cases, the involvement is bilateral. The exact incidence of ocular ischemic syndrome is not known, but it is estimated at 7.5 cases per million every year. In 20% of cases, the involvement is bilateral. The exact incidence of ocular ischemic syndrome is not known, but it is estimated at 7.5 cases per million every year.

The evidence for this patient with ocular ischemic syndrome was carotid artery stenosis, posterior subcapsular cataract, and narrowed retinal vessels. As the patient was 54 years old, the cataract could be senile or complicated. However, the complaint about the sudden blurred vision made us pay more attention to the fundus and complicated cataract. The opaque lens could be due to ischemia and dystrophy. After cataract surgery, we noted that the IOP in both eyes was 5 to 6 mm Hg lower. This would also be beneficial for the ischemic retina because the ocular blood flow and perfusion pressure were increased in the vessels supplying the retina.

The differential diagnosis of this case should be central retinal artery occlusion and retinitis pigmentosa. In this case, although the retinal artery was narrowing and whitening, there was no retinal edema, cherry-spot of the macular, or bone spicule-shaped pigment deposit. On fluorescein angiography of the fundus, these would have been easily differentiated. The patient’s postoperative visual acuity exceeded our expectation, and he was very satisfied with the results. The reason might be that the central visual field was not affected. Also, unlike acute ischemia–reperfusion injury of the retina, the chronic narrowing of the carotid artery and retinal blood vessels might make the retinal neurons gradually adapt and tolerant of the hypoperfusion.

Internal carotid artery stenosis and subclavian artery atherosclerosis are important causes of ischemic stroke, and the treatment of them has been a topic of intense debate over the past 30 years. Despite numerous trials and years of clinical research, the optimal management remains controversial. Now, combination therapy with antiplatelet, antihypertensive, and antidiabetic agents, and treatment of hypercholesterolemia, together with smoking cessation, is the current best medical management. Antiplatelet therapy has been shown to reduce the incidence of stroke by 25%. Patients with less than 50% stenosis do not benefit from carotid endarterectomy surgery. Most patients with less than 70% stenosis, particularly if they are more than a month from the last symptoms, are probably best treated only medically.
Although ocular ischemic syndrome is a rare condition, its complications may lead to irreversible vision loss. Ophthalmologists must be cautious with these symptoms and obtain follow-up data from relevant examinations to identify the underlying causes. Carotid artery stenosis is usually severe when symptoms of ophthalmic ischemia are present. Early intervention may be effective in the treatment of carotid artery stenosis, thus reducing the incidence of ophthalmic and cerebral complications.

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