Left acute neovascular glaucoma after right carotid endarterectomy

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ABSTRACT

Carotid endarterectomy is a commonly performed vascular surgical procedure with well-known complications, such as stroke and nerve injury. Neovascular glaucoma (NVG) is an exceedingly rare complication after carotid endarterectomy that can result in loss of vision. All previous reports of NVG after carotid endarterectomy have occurred on the same side as the carotid surgery; in this report, we present a case of left-sided NVG after right carotid endarterectomy for contralateral ocular ischemic syndrome. We aim to emphasize the importance of early recognition and treatment of this serious complication as rapid intervention has the potential to save sight. (J Vasc Surg Cases and Innovative Techniques 2018;4:112-4.)

CASE REPORT

A 69-year-old man presented for an elective right carotid endarterectomy because of left ocular ischemic syndrome. Past medical history included type 2 diabetes, hypertension, transient ischemic attacks, and bilateral cataract surgery.

The patient gave a history of several weeks of worsening vision in his left eye. Arterial duplex ultrasound and computed tomography angiography of the patient’s carotid arteries demonstrated complete left internal carotid artery occlusion and an 80% stenosis of his right internal carotid artery. Ophthalmologic examination was consistent with left ocular ischemic syndrome, and it was thought that this was due to recent left carotid occlusion and contralateral tight stenosis with reduced flow through the circle of Willis (Fig). An elective right carotid endarterectomy was scheduled.

After an uncomplicated operation, the patient reported reduction in his left eye pain and improved vision. However, on day 3 postoperatively, the patient developed recurrent left eye pain and decreased visual acuity, with nausea and a headache. Magnetic resonance imaging of the brain ruled out any acute intracranial disease as well as cerebral hyperperfusion syndrome. The patient’s left eye pain worsened despite oral opioid analgesia, and a fixed, dilated pupil was observed. At this point, the patient was transferred to a tertiary center for urgent ophthalmologic assessment because of concern of acute glaucoma.

After ophthalmologic review, it was noted that the patient’s visual acuity deteriorated to hand movements only from a previously documented 6/60 Snellen acuity. The cornea was hazy with microcystic edema, there was anterior chamber activity, and the intraocular pressure (IOP) was elevated at 35 mm Hg. The patient was treated with a topical alpha-adrenergic agent and a topical carbonic anhydrase inhibitor as well as oral acetazolamide. Topical prednisolone was started for the anterior chamber inflammation. After administration of these agents, the patient’s eye pain and headache quickly improved, and IOP was found to be 14 mm Hg the following day.

DISCUSSION

NVG is a severe form of glaucoma attributed to new blood vessels in the anterior chamber angle obstructing aqueous humor outflow secondary to posterior segment ischemia. The obstructed aqueous outflow leads to a high IOP and optic nerve damage. There is a high rate of severe visual loss associated with the disease, with a final visual acuity of only light perception being reported in around 70% of patients in a Brazilian study. A number of conditions (both ocular and systemic) are associated with NVG; the most common are central retinal vein occlusion, proliferative diabetic retinopathy, ocular ischemic syndrome, and central retinal artery obstruction. Preoperatively, our patient was diagnosed with ocular ischemic syndrome, putting him at higher risk for
development of NVG. Approximately 50% of patients with ocular ischemic syndrome develop raised IOP, most commonly from development of NVG. However, IOP is commonly normal or even low, secondary to ischemia of the ciliary body and reduced production of aqueous humor. Our patient was known to have a chronically low pressure preoperatively. This scenario highlights the need for surgeons to take a targeted history from their patients about any conditions predisposing to NVG, with the aim of identifying patients who are at risk of this postoperative complication. Although it would be too burdensome for all patients to undergo an ophthalmology assessment before carotid endarterectomy, those patients with significant visual disturbances should be investigated before surgery.

In their literature review, Ng et al reported only 12 documented cases of NVG after carotid endarterectomy, making this an exceptionally rare postoperative complication. A case of NVG has recently been recorded after carotid angioplasty and stenting. Of note, all previously reported cases have involved ipsilateral NVG in relation to carotid intervention, that is, the glaucoma manifested on the same side as the carotid operation. Our report is unique in that the NVG occurred on the side opposite to carotid surgery. A possible explanation for this phenomenon is that the patient’s left eye was receiving collateral blood supply from the right carotid system, and once luminal stenosis of the right internal carotid artery was relieved as a result of surgery, rapid left eye revascularization occurred. It is postulated that such revascularization results in a sudden increase in aqueous humor production by the ciliary body of the eye that is unable to be equilibrated because of reduced aqueous humor outflow. If it is not controlled, the resulting rise in IOP can lead to optic nerve damage.

In our case, the patient exhibited fairly typical symptoms of NVG, including loss of visual acuity, eye pain, and headache. Other documented symptoms include photophobia and exophthalmos. Acute onset of these symptoms postoperatively, particularly in patients with pre-existing ocular disease, should alert the clinician to the possibility of NVG. The time at onset of glaucoma in this case is in keeping with other reports, with 7 of 12 previous cases having occurred between 1 and 5 days postoperatively. The latest onset occurred 16 days after surgery, highlighting that NVG does not always occur in the acute postoperative period. At the onset of symptoms, appropriate measures were put in place for our patient to ensure that he was not having an acute cerebrovascular event, such as a hemorrhagic or ischemic stroke, and cerebral hyperperfusion syndrome was ruled out with magnetic resonance imaging.

On discharge, the patient was managed with topical ocular agents followed by intravitreal bevacizumab, a vascular endothelial growth factor (VEGF) inhibitor. VEGF plays a major part in mediating active intraocular neovascularization in patients with ischemic retinal diseases and has become a major therapeutic target. However, whereas treatment options with VEGF inhibitors or panretinal photocoagulation might be used in an attempt to control the neovascularization process, surgical procedures are necessary in some cases to achieve normal levels of IOP and to avoid optic nerve damage. A number of such surgical procedures are used in conjunction with medical therapy to treat NVG, including cyclocryotherapy, trabeculotomy, posterior lip sclerotomy, and implantation of drainage devices. This demonstrates that the management of NVG is often time-consuming and costly.

CONCLUSIONS

NVG, a rare but significant complication after carotid endarterectomy, can lead to blindness. Patients with pre-existing ocular syndromes associated with NVG must be identified preoperatively. Close monitoring and urgent ophthalmologic assessment are required for any patient complaining of a change in visual acuity and ocular pain in the days after carotid endarterectomy. We advise that patients with pre-existing ocular conditions predisposing to NVG have carotid endarterectomy performed only in centers with rapid access to an ophthalmology team as a delay in the management of NVG may lead to complete loss of vision.

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