Neovascular Glaucoma Post Carotid Endarterectomy: A Case Report and Review of the Literature

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Carotid endarterectomy is a standard vascular surgical procedure performed worldwide and encompasses multiple risks including cerebral hyperperfusion syndrome, stroke, carotid dissection and aneurysmal formation, all of which are well documented in the literature. However, neovascular glaucoma manifesting post carotid endarterectomy, is extremely rare and can have disastrous consequences if left undiagnosed. In this article, we present one such case of neovascular glaucoma manifesting post carotid endarterectomy and review the available literature on this uncommon entity.

Case Report
A 58-year-old male presented with sepsis secondary to diabetic foot infection, on a background of longstanding non-insulin dependent diabetes, complicated by neuropathy and diabetic retinopathy. Two days post-ulcer debridement, the patient complained of right eye visual disturbance, describing the episode as “a shade being drawn across the eye,” consistent with amaurosis fugax. A pre-operative ophthalmological review noted normal visual acuity and changes consistent with chronic ocular ischemia presumed to be secondary to right internal carotid artery stenosis.

A subsequent computed tomographic angiogram (CTA) of the head and neck confirmed subtotal occlusions to the proximal right internal carotid artery, both origins of the external carotid arteries and the origin of the left vertebral artery.

The patient subsequently underwent an urgent right carotid endarterectomy (CEA). Intra-operatively, he was noted to have an extremely tight stenosis of the right ICA. On the second postoperative day, he complained of worsening visual acuity with associated vomiting and headaches. An urgent ophthalmology review revealed a non-reactive right pupil, an elevated intraocular pressure of 47 mmHg and changes consistent with neovascular glaucoma.

Concern over cerebral hyperperfusion syndrome post carotid endarterectomy culminated in cerebral magnetic resonance imaging (MRI), which revealed no acute intracranial pathology, and specifically no evidence of cerebral oedema. The patient underwent intravitreal anti-vascular endothelial growth factor (anti-VEGF) administration with serial measurements of intraorbital pressures and retinal photocoagulation. Blood pressure control was also maintained at a systemic target of 150 mmHg with medical therapy. He recovered well with no complications on follow-up a month later.

Discussion
Carotid endarterectomy has numerous complications, one of which is cerebral hyperperfusion syndrome. It occurs as a result of transient increase in cerebral perfusion after revascularisation of the cerebral blood supply as well as alteration of the cerebral vascular autoregulatory mechanisms. Cerebral hyperperfusion syndrome is defined as a cerebral blood flow increase of more than 100% baseline. The classic triad of headache, convulsions and neurological deficit soon manifests and is followed by cerebral oedema, hemorrhage and eventual death. In our patient who experienced headaches and vomiting, this was our utmost concern and the reason for the urgent cerebral MRI.

The manifestation of neovascular glaucoma post CEA, however, is extremely rare. Only 11 case reports exist as outlined in our extensive search of the literature (Table 1).1–9

In our table of collated cases of neovascular glaucoma manifesting post CEA available in the literature (Table 1), we note that all patients presented with vague ocular symptoms, which included eye pain, glare, blurred vision and headache. In our case, the patient developed amaurosis fugax after debridement of his foot wound but had no
Table 1

<table>
<thead>
<tr>
<th>Case</th>
<th>Carotid endarterectomy side</th>
<th>Carotid degree of ICA stenosis</th>
<th>Co-morbidities</th>
<th>Pre-CEA ocular symptoms</th>
<th>Post-CEA ocular symptoms</th>
<th>Onset of symptoms post CEA (days)</th>
<th>Intraocular pressure (mmHg)</th>
<th>Treatment of neovascular glaucoma</th>
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<tbody>
<tr>
<td>1</td>
<td>Right</td>
<td>Right</td>
<td>Diabetes mellitus</td>
<td>R eye blurred vision</td>
<td>R eye pain and tearing</td>
<td>14</td>
<td>18</td>
<td>44–46 Medical PRP*</td>
</tr>
<tr>
<td>2</td>
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<td>Left</td>
<td>Diabetes mellitus</td>
<td>L eye blurred vision</td>
<td>Not stated</td>
<td>5</td>
<td>21–23</td>
<td>Medical PRP*</td>
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<tr>
<td>3</td>
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<td>Diabetes mellitus</td>
<td>Right orbital pain</td>
<td>R eye pain</td>
<td>4</td>
<td>16</td>
<td>Trabeculotomy</td>
</tr>
<tr>
<td>4</td>
<td>Left</td>
<td>Left</td>
<td>Diabetes mellitus</td>
<td>R eye decreased visual acuity</td>
<td>R eye blurred vision</td>
<td>1</td>
<td>10–18</td>
<td>Medical PRP*, Trabeculectomy</td>
</tr>
<tr>
<td>5</td>
<td>Same as above</td>
<td>Right</td>
<td>Diabetes mellitus</td>
<td>Right orbital pain</td>
<td>R eye blurred vision</td>
<td>4</td>
<td>24</td>
<td>Medical PRP*, Trabeculectomy</td>
</tr>
<tr>
<td>6</td>
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<td>Diabetes mellitus</td>
<td>L eye decreased visual acuity</td>
<td>R eye blurred vision</td>
<td>1</td>
<td>18</td>
<td>Medical PRP*, Trabeculectomy</td>
</tr>
<tr>
<td>7</td>
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<td>Right</td>
<td>Diabetes mellitus</td>
<td>Right orbital pain</td>
<td>R eye blurred vision</td>
<td>4</td>
<td>24</td>
<td>Medical PRP*, Trabeculectomy</td>
</tr>
<tr>
<td>8</td>
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<td>Diabetes mellitus</td>
<td>L eye decreased visual acuity</td>
<td>R eye blurred vision</td>
<td>1</td>
<td>18</td>
<td>Medical PRP*, Trabeculectomy</td>
</tr>
<tr>
<td>9</td>
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<td>R eye blurred vision</td>
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<td>24</td>
<td>Medical PRP*, Trabeculectomy</td>
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<tr>
<td>10</td>
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<td>Right</td>
<td>Diabetes mellitus</td>
<td>Right orbital pain</td>
<td>R eye blurred vision</td>
<td>1</td>
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<td>Medical PRP*, Trabeculectomy</td>
</tr>
<tr>
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<td>L eye decreased visual acuity</td>
<td>30</td>
<td>Medical PRP*, Trabeculectomy</td>
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</table>

In our collated data, all patients who presented with neovascular glaucoma had stenosis of the internal carotid artery greater than 70%. Six cases had carotid stenosis of 90% or more. The prevalence of tight stenoses preoperatively suggests that this may be a precursor for the development of neovascular glaucoma post CEA due to the greater degree of restored reperfusion. Diabetes and hypertension were present in 7 of the 11 cases. All patients in the review presented with vague ocular symptoms, which included eye pain, blurred vision, glare and headaches prior to carotid endarterectomy. The onset of ocular symptoms post CEA ranged from 1 to 14 days, suggesting that ophthalmological review and followup should be tailored to a time period of 2 weeks. In the nine cases, which reported intraocular pressures, a preoperative range of 12–24 mmHg was noted. Intraocular pressures increased remarkably by up to 3-fold post CEA and this can be attributed to increased perfusion of the eye and the associated manifestation of neovascular glaucoma. Treatment of neovascular glaucoma in all cases involved medical therapy in conjunction with ophthalmologic intervention. The majority of cases (six cases) utilised panretinal laser photocoagulation. Surgery via trabeculotomy was used in four cases and a Molteno implant was used in one case.

Our patient had pre-existing chronic ocular ischemia preoperatively. Chronic ocular ischemia is a rare form of ischemia of the eye in patients with existing carotid disease. The early and often asymptomatic stage of chronic ocular ischemia is referred to as venous stasis retinopathy (VSR) which can occur in up to a third of patients with symptomatic carotid disease. In addition to this, patients with carotid occlusive disease can present with a variety of other ocular symptoms including teichopsia, anterior segment ischaemia,
ocular neovascularisation, glaucoma, cataracts, diabetic retinopathy and macular degeneration. In patients who were symptomatic with ocular events, the presence of significantly worse atherosclerotic disease was noted in the ipsilateral internal carotid artery, external carotid artery, aortic arch branch disease and vertebral artery.

These findings illustrate the fact that patients with carotid occlusive disease can present with a myriad of preoperative ocular symptoms which may progress and worsen post CEA. This warrants close monitoring of ocular symptoms as well as ophthalmology review with any worsening or progression of symptoms.

Neovascular glaucoma usually occurs in proliferative diabetic retinopathy and arises secondary to formation of abnormal new blood vessels, which inhibit drainage of aqueous humour, from the anterior segment of the eye. The subsequent buildup of aqueous humour leads to the symptoms of glaucoma. Treatment consists of intravitreal administration of agents directed against vascular endothelial growth factors (anti-VEGF agents), panretinal photocoagulation (PRP), glaucoma shunts or deep vitrectomy as outlined in Table 1. Clinical indicators of improvement are mainly based around intra-ocular pressure measurements as well as fundoscopic monitoring.

There are multiple theories for the occurrence of neovascular glaucoma post carotid endarterectomy. One theory describes the pathophysiology as a result of chronic ocular ischemia, leading to iris neovascularisation, which alters the resorptive capacity of the eye for aqueous humour. Upon revascularisation, there is a sudden increase in aqueous humour production by the ciliary body with resorptive capacity remaining unchanged. This imbalance in aqueous humour production and resorption results in a drastic increase in intraocular pressure and subsequent neovascular glaucoma. Another prevalent theory describes a model of increased angiogenic growth factor production as a result of stimuli from increased perfusion, leading to ophthalmic changes that result in neovascular glaucoma.

**Conclusion**

There is a positive correlation between ocular pathology and the presence of existing carotid disease. Regardless of the cause, neovascular glaucoma is a rare but serious complication of carotid endarterectomy. The manifestation of neovascular glaucoma in patients post carotid endarterectomy, although rare, can have disastrous consequence if missed.

We advocate pre-operative ophthalmology review for patients undergoing carotid artery endarterectomy particularly in patients with existing comorbidities like hypertension and diabetes that are associated with ocular symptoms. If ocular symptoms post CEA are discovered, then an urgent ophthalmology review is recommended to exclude neovascular glaucoma as a potential cause in addition to other investigations such as a cerebral MRI.

**Disclosure Statement**

The authors have nothing to disclose and also state no conflict of interest in the submission of this manuscript.

**References**