Severe Neovascular Glaucoma Exacerbation as a Complication of Carotid Artery Stenting: A Case Report

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Abstract

Introduction: Neovascular glaucoma (NVG) has been rarely reported as an acute complication of carotid endarterectomy, but there is scant literature available regarding this potential condition following carotid artery stenting (CAS). We present a case of severe worsening of NVG occurring after bilateral CAS with progressive deterioration of vision ultimately leading to blindness.

Case Description: A 66-year-old male with multiple stroke risk factors, bilateral cataract extraction, proliferative diabetic retinopathy of left eye, and nonproliferative diabetic retinopathy of right eye, and prior left eye pars plana vitrectomy presented with episodes of transient right eye vision loss in context of bilateral high-grade internal carotid artery stenoses. He underwent right CAS with subsequent elevation of bilateral intraocular pressures (IOPs) concerning for acute NVG. Over time, the patient had some interval improvement in IOPs and underwent planned left CAS. After the procedure, he again developed elevated IOPs, concerning for acute NVG which eventually led to right eye pars plana vitrectomy for vitreous hemorrhage and refractory IOP elevation. At 6-month follow-up from initial stenting, the patient was blind in both eyes. **Discussion:** We present a case of recurrent IOP elevations following CAS eventually resulting in bilateral eye blindness. This case is important not only as an illustration of an underrecognized postprocedural CAS complication but also as a demonstration of likely elevated risk of NVG following CAS for patients with other predisposing risk factors for ocular hypertension such as glaucoma, proliferative diabetic retinopathy, prior cataract extraction, and prior pars plana vitrectomy.

Keywords

carotid artery stenting, endovascular therapy, neovascular glaucoma

Introduction

Cerebral hyperperfusion syndrome is a rare, but preventable, complication after carotid endarterectomy (CEA) or carotid artery stenting (CAS).¹ Retrospective reviews have reported that following CAS, the incidence of cerebral hyperperfusion syndrome is around 1%.² Neovascular glaucoma (NVG), a more infrequent complication following carotid revascularization, is a severe form of glaucoma attributed to iris neovascularization, development of a closed anterior chamber angle, and subsequent development of very high intraocular pressure (IOP). Carotid artery obstructive disease is the third most common cause of NVG.^{3,4} However, the paradoxical development of NVG has been reported rarely as an acute complication of carotid revascularization, specifically CEA.^{5,6} To our knowledge, even fewer cases have been reported regarding the development of acute NVG following CAS.⁷⁻¹⁰ We report a case of severe NVG occurring after bilateral CAS conducted for

concern of amaurosis fugax and cerebral ischemia. The case involves recurrence of severe IOP elevations after right CAS and then subsequently left CAS, ultimately leading to blindness.

The paradoxical occurrence of NVG following carotid revascularization was reported by Coppeto et al in 1985 who

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hypothesized that decreased perfusion of the ciliary body from carotid artery obstruction significantly decreases aqueous humor production, but when perfusion is restored, aqueous production often returns to pre-ischemic levels leading to a dramatic increase in the IOP.¹¹ There is also a theory that increased perfusion leads to increased angiogenic growth factors that lead to ophthalmic changes resulting in neovascular glaucoma.⁶ In recent years, CAS has emerged as a therapeutic option for carotid revascularization, and due to scant evidence of standardized clinical protocols in place regarding NVG development after CAS, we report the following case.

Case Report

A 66-year-old male was admitted to our institution after experiencing episodes of right eye visual loss. He had a past medical history significant for hypertension, hyperlipidemia, poorly controlled diabetes mellitus type II, coronary artery disease status post bypass grafting, right eye nonproliferative diabetic retinopathy, left eye blindness secondary to neovascular glaucoma, and proliferative diabetic retinopathy with vitreous hemorrhage status post multiple intravitreal injections and pars plana vitrectomy with endolaser, bilateral eye cataract surgery, congestive heart failure, and end-stage renal disease not on hemodialysis.

Patient initially presented to ophthalmology outpatient clinic due to right eye pain, found to have concern for elevated IOP (right eye 24 mm Hg, left eye 31 mm Hg), and neovascularization of the left eye but not the right eye. He returned to ophthalmology clinic the next day for repeat IOP check and noted to have had 2 episodes of transient right eye vision loss also initially described as “blurring” and “dimming” of vision but without classic “window shade” phenomenon. Ophthalmological examination at that time noted improved IOP (visual acuity [VA], IOP: right eye 20/30, 19 mm Hg; left eye 1/200, 21–24 mm Hg). He was subsequently admitted to our institution due to concern for ocular ischemia, and magnetic resonance imaging (MRI) of the brain was remarkable for punctate area of diffusion restriction in the left centrum semiovale (see Figure 1A), while magnetic resonance angiography (MRA) of head and neck demonstrated critical stenosis of the proximal and petrous segments of the left internal carotid artery (ICA) and proximal right ICA. Given these findings, patient was administered clopidogrel load and continued on dual antiplatelet therapy with close monitoring and later noted to have 2 distinct episodes of right eye amaurosis fugax symptomatology, documented as “blackening of visual field, as a curtain coming down in front of his eye.”

Due to concern for clinically symptomatic right ICA high-grade stenosis, in the setting of near-total occlusion of contralateral ICA, CEA was not considered an optimal therapeutic option and the patient underwent right-sided CAS successfully under conscious sedation without any immediate complication (see Figure 1B). Patient was admitted to intensive care unit postoperatively with systolic blood pressure (BP) goal of 100

to 120 mm Hg and continued atropine, brimonidine, dorzolamide, prednisolone, and timolol eyedrops for glaucoma. During postoperative day (POD) #1, patient was noted to have worsening right eye vision, and ophthalmology consultation revealed elevated IOP (right eye 26–29 mm Hg, left eye 22–24 mm Hg). Repeat right carotid ultrasound revealed stent patency without evidence of re-stenosis. The prednisolone acetate eye drops were modified to more frequent dosing while he was maintained on atropine, brimonidine, dorzolamide, and timolol drops for medical management of IOP. On POD #2, his examination revealed persistently elevated IOP (right eye 28 mm Hg, left eye 31 mm Hg) and by POD #5, he was noted by the ophthalmologist to have persistent ocular hypertension on maximum medical therapy for elevated IOP (right eye 34 mm Hg, left eye 35 mm Hg). Hospitalization was prolonged due to renal dysfunction and respiratory distress, patient had recurrent episodes of decreased right eye VA and repeat assessment by ophthalmology on POD #9 showed elevated IOP (right eye 30–32 mm Hg, left eye 26–27 mm Hg) but noted that visual fluctuations in the right eye were improving (VA: right eye 20/200, left eye hand motion), and medical management was continued. Post-discharge, follow-up examination approximately 3 weeks after stenting revealed some IOP improvement (right eye 22 mm Hg, left eye 18 mm Hg) with worsened right eye VA 20/100 –2 as compared to pre-hospital examination. Over the next 5 weeks, the patient visited outpatient ophthalmology 4 additional times for close monitoring with documented complaints of right eye decreased vision, which was managed non-operatively with eye drops for neovascular glaucoma (continued on atropine, brimonidine, dorzolamide, methylprednisolone, and started on travoprost drops) prior to planned left CAS.

Approximately 2 months following right CAS, the patient's ophthalmology examination had IOPs (right eye 23–24 mm Hg, left eye 19 mm Hg) but right eye VA (uncorrected) of 20/150 +2. He was admitted soon thereafter for left CAS due to concern for symptomatic stenosis in the setting of punctate infarct seen on prior MRI brain in left centrum semiovale. Stenting was performed without any reported procedural complications under conscious sedation (see Figure 1C). Intraoperatively, patient had peak systolic BP 168 mm Hg and peak diastolic BP 88 mm Hg, with post-operative systolic BP goal <120 mm Hg. Although he had prior reported critical stenosis of petrous left ICA on MRA head, cerebral angiogram showed that post-stenting, patient had physiologic filling of the ACA territory and the left MCA with good capillary blush in venous drainage without large vessel dropout. Patient was discharged home on POD #2 but presented to ophthalmology clinic approximately 5 days post-stenting with elevated IOP (right 33–34 mm Hg, left 25–26 mm Hg). During the subsequent month, the patient ultimately remained with uncontrolled IOPs requiring pars plana vitrectomy, endolaser, and endocyclophotocoagulation of the right eye, which was subsequently repeated approximately 4 weeks later. On interval follow-up with neurology approximately 4 months after initial stenting

Figure 1. A, Magnetic resonance imaging (MRI) brain axial images demonstrating punctate diffusion restriction in the left centrum semiovale with apparent diffusion coefficient (ADC), diffusion-weighted imaging (DWI), and T2-fluid-attenuated inversion recovery (T2 FLAIR images), respectively. B, Cerebral digital subtraction angiogram images demonstrating right carotid artery stenosis pre-stenting (left) and post-stenting (right). C, Cerebral digital subtraction angiogram images demonstrating left carotid artery stenosis pre-stenting (left) and post-stenting (right).

of right ICA, he was noted to have no light perception in either eye. His most recent neuro-ophthalmology examination at our institution more than 3 years from initial stenting redemonstrated that he had no light perception in either eye, nonreactive pupils, and florid neovascularization of the iris of both eyes.

Discussion

This case presentation highlights a rare but potentially devastating complication of carotid revascularization and is one of a limited number of case reports in the literature that demonstrates this effect specifically after CAS. To the best of our

knowledge, only 4 other reports are published regarding NVG following CAS.⁷⁻¹⁰ Our case is similar to Lee et al's case in that our patient also had prior cataract surgery. It is similar to the case by Loving et al and one of the cases reported by Kawano et al in that they had concern for NVG on ophthalmological examination prior to carotid revascularization.

Our report is unique, however, in that IOP elevation occurred in both eyes following carotid revascularization and notably occurred not only after right CAS but also subsequently after left CAS months later. It is unclear why IOP elevation was also seen in the eye contralateral to the side of carotid stenting, although there is a prior reported case of this effect occurring after CEA which was hypothesized to be due to collateral blood flow.¹²

This case is complex due to the patient's pre-existing ophthalmological history but also illustrates the elevated risk of developing NVG following CAS for patients with other independent risk factors for iris neovascularization and NVG: prior cataract extraction, prior pars plana vitrectomy, and diabetic retinopathy.^{3,7,13,14}

Conclusion

In conclusion, we present a case report involving acute increase in bilateral IOP following CAS, likely due to NVG. Similar pathology has been previously reported after CEA with the devastating complication of chronic vision loss secondary to ocular hypertension. These complications have very rarely been reported following CAS. We recommend that all patients with risk factors for development of glaucoma receive pre-operative ophthalmologic screening prior to carotid revascularization, especially in the non-emergent setting. Additionally, routine bedside tonometry may be a useful tool after CAS, particularly in those with predisposing risk factors for glaucoma or ocular hypertension.

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Declaration of Conflicting Interests

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