Hyperperfusion syndrome: Aggravation of neovascular glaucoma after stenting for internal carotid artery stenosis

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ABSTRACT

Introduction: Cerebral hyperperfusion syndrome (CHS) is an exceedingly rare yet serious complication after abrupt revascularization of the carotid artery circulation with endarterectomy or stent placement. Case Report: A 70-year-old male who had severe bilateral internal carotid artery (ICA) stenosis and a history of neovascular glaucoma (NVG) was referred to our institution for single staged right ICA stenting. Twenty-four hours after the procedure, he developed a painful red right eye with a fixed dilated pupil and complained of a right-sided periorbital throbbing headache. Tonometry of the right eye revealed a substantially elevated intraocular pressure (IOP). Conclusion: There are few documented cases of aggravation of NVG after ICA stenting. To reduce the risk of NVG exacerbation as a complication of post-procedural CHS, close monitoring of perioperative IOP should be considered.

Keywords: Carotid revascularization, Cerebral hyperperfusion, Neovascular glaucoma

INTRODUCTION

Carotid artery angioplasty and stenting (CAAS) is an endovascular procedure used to reduce the risk of cerebrovascular events in patients with severe carotid artery stenosis [1]. This procedure is also used for symptomatic patients in states of chronic cerebral hypoperfusion. This phenomenon may produce ocular ischemia, leading to neovascularization of the iris and ciliary body, resulting in NVG [2]. Chronic states of cerebral hypoperfusion lead to compensatory dilation of cerebrovasculature [3]. This dysregulation of cerebrovasculature is a risk factor for post-procedural CHS [4]. Known complications of CAAS include perioperative stroke, myocardial infarction, cervical hematoma, nerve injury, infection, and carotid restenosis [3–6]. There have been scarce reports of an association between CAAS and exacerbation of pre-existing NVG as a direct result of post-operative CHS [7]. In this report, we present a unique case of an exacerbation of NVG following CAAS. The patient provided verbal consent for this report.
CASE REPORT

A 70-year-old man presented to the Denver Veterans Affairs Medical Center for an elective CAAS for severe carotid stenosis with ocular ischemic syndrome complicated by NVG. The patient’s past medical history included peripheral artery disease, type-2 diabetes, congestive heart failure, and chronic obstructive pulmonary disease. He had an 80 pack-year smoking history. He was seen by ophthalmology two months prior to the procedure and had an IOP of 29 mmHg. He had neovascularization of the right iris with resultant NVG that was medically managed. There was no evidence of diabetic retinopathy. One month prior to the procedure, computed tomography angiography (CTA) showed profound atherosclerosis of bilateral carotid arteries (Figure 1A). The stenosis extended from the origins of the common carotid arteries to the bilateral supraclinoid internal carotids with 80–90% cross-sectional stenosis. The patient underwent uncomplicated CAAS of the right ICA. Post-procedural CTA showed a widely patent vessel (Figure 1B).

The morning after the procedure the patient experienced a right-sided, throbbing headache and severe periorbital pain. Examination revealed redness of the right eye with a fixed 8 mm pupil. The left pupil was 3 mm and responsive to light. Gonioscopy revealed peripheral anterior synechiae in that eye. Right IOP was 37–40 mmHg. Treatment was initiated with timolol, dorzolamide, latanoprost, and brimonidine eye drops. Intraocular aflibercept was administered and emergent anterior chamber drainage was performed, resulting in normalization of IOP. The next day, the patient’s IOP was again found to be elevated at 29 mmHg. Treatment was modified to include acetazolamide eye drops. Over the next several days, the patient’s headache and periorbital pain resolved. His right pupil became mildly responsive and dilation reduced to 5 mm with a 3 mm left pupil (Figure 2). Prior to discharge, his right IOP was 23 mmHg.

DISCUSSION

Manifestation or exacerbation of NVG is a rare complication of CAAS or carotid endarterectomy and can occur between 24 hours and 14 days following recanalization of the carotid arterial circulation.

The most commonly proposed mechanism is that sudden improvement in ocular circulation stimulates rapid increase in production of aqueous humor from the previously neovascularized ciliary body.

However, the drainage capacity of the anterior chamber does not increase nor accommodate for this increased production of aqueous humor. This mismatch can lead to a marked increase in IOP [2].

Acutely elevated IOP can present with headache, eye pain, and blurry vision. This constellation of ocular symptoms shares considerable overlap with cerebral CHS, which most commonly features altered mental status, ipsilateral headache, seizures, and focal neurologic deficits. Tight control of blood pressure in the periprocedural period is a mainstay in the management of CHS, however, it is likely insufficient for preventing ocular complications. Even with tight blood pressure control and aggressive medical management of IOP, the majority of reported cases required direct ophthalmologic intervention, as in our patient [7–10].

With few cases in the literature, it is unknown which patients are at increased risk for post-procedural exacerbation of NVG. Risk factors for pre-existing ischemic NVG include high-grade carotid stenosis, uncontrolled hypertension, and poor glycemic control. These same factors appear to be predictive of post-procedural CHS development [4, 8]. We suggest close post-procedural motoring of IOP for patients with pre-existing neovascularization of the iris or NVG. For these patients, IOP should be optimized pre-operatively.

It has been suggested that carotid revascularization be performed in a graded, stepwise fashion to prevent sudden changes in cerebrovascular perfusion. A study in 2009 by Yoshimura et al. showed a significant decrease in CHS following staged vascular intervention when
compared to single-staged CAAS [11]. It is uncertain if this would have prevented the exacerbation of NVG in our patient.

Failure to recognize or appropriately manage acute exacerbation of glaucoma could result in catastrophic vision loss. Likewise, failure to adequately control blood pressure and IOP in cases of CHS could lead to devastating neurologic and ophthalmic consequences. Any ocular signs or symptoms, including periorbital headache, should prompt consideration of an urgent ophthalmologic evaluation. Both conditions, recognized early, have favorable clinical outcomes.

CONCLUSION

In this case report, the patient likely developed NVG due to chronic ocular ischemia caused by severe carotid artery stenosis. After stent placement and a sudden significant increase in perfusion to the ciliary body of the iris, an acute exacerbation of the pre-existing NVG was observed. We urge clinicians to consider optimizing pre-procedural IOP and careful monitoring of post-procedural IOP for patients with known NVG. This intervention may prevent serious ocular complications.

REFERENCES


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Author Contributions

Bailey A Loving – Conception of the work, Design of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Jacob Walker – Conception of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Guarantor of Submission

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Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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