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CASE REPORT

Simultaneous Occurrence of Neovascular Glaucoma with Proliferative Diabetic Retinopathy and Carotid Occlusive Disease: A Case Report

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Abstract

Neovascular glaucoma secondary to carotid occlusive disease is uncommon. We report the case of a 60-year-old female who presented to the emergency department with right eye pain, redness, blurred vision, dizziness, and body pain for three days. Examination revealed an intraocular pressure (IOP) of 45 mmHg in the right eye. Carotid Doppler ultrasound, performed to evaluate for carotid occlusive disease, demonstrated 80% occlusion of the right internal carotid artery. Subsequent computed tomography angiography (CTA) confirmed significant stenosis (at least 70%) of the proximal right internal carotid artery. The patient was referred to vascular surgery and underwent a thromboendarterectomy of the right internal carotid artery. At nine months of follow-up, the patient demonstrated significant improvement in ocular pain and noticeable reduction in IOP to 16 mmHg. This case highlights the importance of clinical reasoning in identifying less common etiologies, such as carotid artery stenosis, in patients with treatment-resistant neovascular glaucoma.

Keywords: Glaucoma, Neovascular diabetic retinopathy, Carotid artery diseases, Diabetes mellitus

Introduction

Neovascular glaucoma (NVG) is secondary glaucoma characterized by the formation of new blood vessels (rubeosis iridis) on the iris and the proliferation of fibrovascular tissue in the anterior chamber angle, resulting in increased intraocular pressure (IOP), optic nerve damage, and visual loss. This disease is refractory to treatment and has a poor prognosis. It is commonly associated with retinal and anterior segment ischemia, such as proliferative diabetic retinopathy (PDR), central retinal vein occlusion (CRVO), and carotid occlusive disease (COD).[1]

Coat was the first to describe histopathological evidence of new blood vessel formation in the iris of a patient with central retinal vein occlusion.[2] This discovery significantly encouraged others to explore this previously unknown condition. Salus reported similar findings of new blood vessel formation in the eyes of patients with diabetes.[3] In one study, 36% of all cases of NVG arose from CRVO, 32% from proliferative diabetic retinopathy, and 13% from carotid artery occlusive disease.[4]

Early recognition of neovascular glaucoma secondary to carotid artery stenosis can be life-saving;

however, its rarity makes diagnosis challenging. With only a few cases reported in the literature, the condition often remains underrecognized. Carotid occlusive disease (COD) can exacerbate retinal and anterior segment ischemia, leading to further complications. This case illustrates the intricate relationship between these conditions and underscores the importance of a multidisciplinary approach to management.

Case Presentation

We report the case of a 60-year-old woman diagnosed with neovascular glaucoma secondary to proliferative diabetic retinopathy. During her hospital stay, the ophthalmology and vascular surgery teams at the Bahrain Defence Force Royal Medical Services, Military Hospital Bahrain, successfully managed her concomitant carotid occlusive disease with a combination of intravitreal anti-vascular endothelial growth factor (anti-VEGF) injections, pan-retinal photocoagulation (PRP), implantation of an Ahmed glaucoma valve, and thromboendarterectomy.

The patient had a history of type 2 diabetes mellitus, hypertension, and dyslipidemia. She was on regular follow-up at the eye clinic for bilateral neovascular glaucoma secondary to advanced diabetic eye disease. During this period, her condition remained stable, with well-controlled intraocular pressure (IOP) and diabetic retinopathy.

In August 2024, during a scheduled follow-up visit, the patient presented to the eye clinic with a two-day history of right eye pain. The pain had a sudden onset, was localized to the right eye without radiation, and was described as sharp. It was not associated with changes in vision or halos. There was no history of ocular trauma or recent contact lens use. On examination, best-corrected visual acuity was 6/6 in the right eye and 6/12 +2 in the left eye, with IOP measuring 32 mmHg and 22 mmHg, respectively. Slit lamp examination of the right eye revealed clear conjunctiva and cornea, a reactive pupil, and a deep, clear anterior chamber. The ophthalmologist also observed aggressive iris neovascularization, early cataractous lens changes, clear vitreous, a fundus cup-to-disc (CDR) of 0.5 with a mild pale rim, a clear macula, laser marks, and a flat retina in the right eye. In the left eye, slit-lamp examination revealed similar anterior segment findings with faint neovascularization and early cataractous lens changes. The vitreous was clear, and fundus examination demonstrated a CDR of 0.3 with a healthy rim, laser marks, and a grossly flat retina. Gonioscopy showed new vessels at the anterior chamber angle in the right eye.

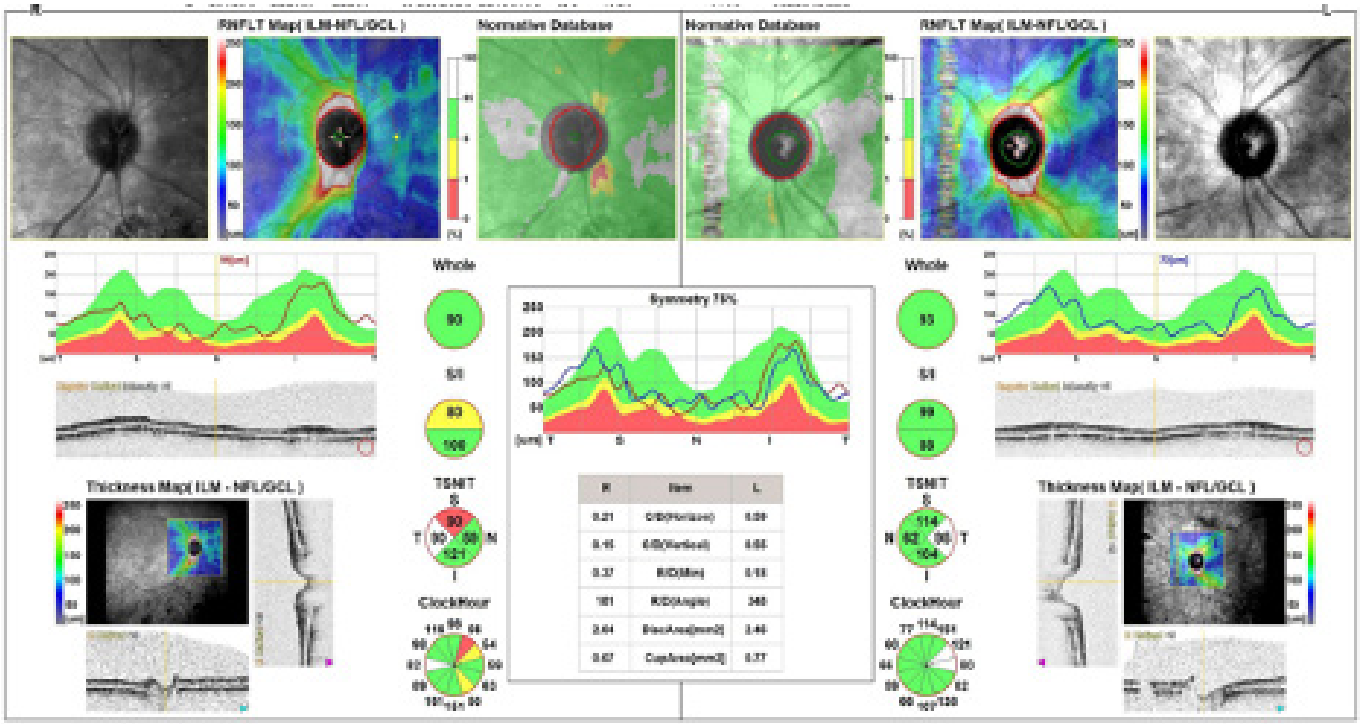

Figure 1: Pre-operative Optical Coherence Tomography (OCT)

Based on the ocular examination findings, the patient was treated with medication, intravitreal injections, and pan-retinal photocoagulation (PRP). This treatment led to regression of the rubeosis; however, despite maximum tolerable anti-glaucoma medications, the patient's right eye IOP remained poorly controlled. Therefore, a decision was made to implant an Ahmed glaucoma valve in the right eye to improve aqueous outflow. The procedure was completed without complications, and the patient's IOP remained well controlled for the following three months.

Four months after Ahmed glaucoma valve implantation, the patient presented to the emergency department with a three-day history of right eye pain, redness, blurred vision, dizziness, and body pain. Ocular examination revealed a visual acuity of counting fingers (CF) in the right eye, an IOP of 45 mmHg, conjunctival congestion, corneal edema, and rubeosis iridis. These findings were consistent with a diagnosis of recurrent neovascular glaucoma. The patient received maximal anti-glaucoma therapy in addition to anti-VEGF and PRP. Although most new vessels showed regression, a large channel extending from the anterior chamber angle persisted, and the patient's IOP remained poorly controlled. In view of the patient's comorbidities, risk factors, and refractory changes in the patient's IOP, the primary ophthalmologist maintained a high index of suspicion and advised a carotid Doppler ultrasound to rule out carotid occlusive disease. The carotid Doppler ultrasound showed 80% occlusion of the right internal carotid artery.

Ultrasound Doppler of the Carotid Arteries

Diffuse atherosclerotic changes of the examined arteries with loss of intimal lucency and subintimal/media thickening were noted. A calcified atherosclerotic plaque was identified in the proximal right internal carotid artery, causing approximately 80% diameter stenosis, with peak systolic velocity tripled at the stenosis site. A calcified atherosclerotic plaque was also noted in the right carotid bulb.

Based on these findings, the patient was referred to the vascular surgery department, and a carotid computed tomography angiography (CTA) was performed to confirm the diagnosis.

CTA Carotid

CTA of carotid arteries demonstrated calcified intimal plaques in the right common carotid artery, bulbu, and proximal aspect of the right internal carotid artery, with significant stenosis (at least 70%) of the proximal right internal carotid artery.

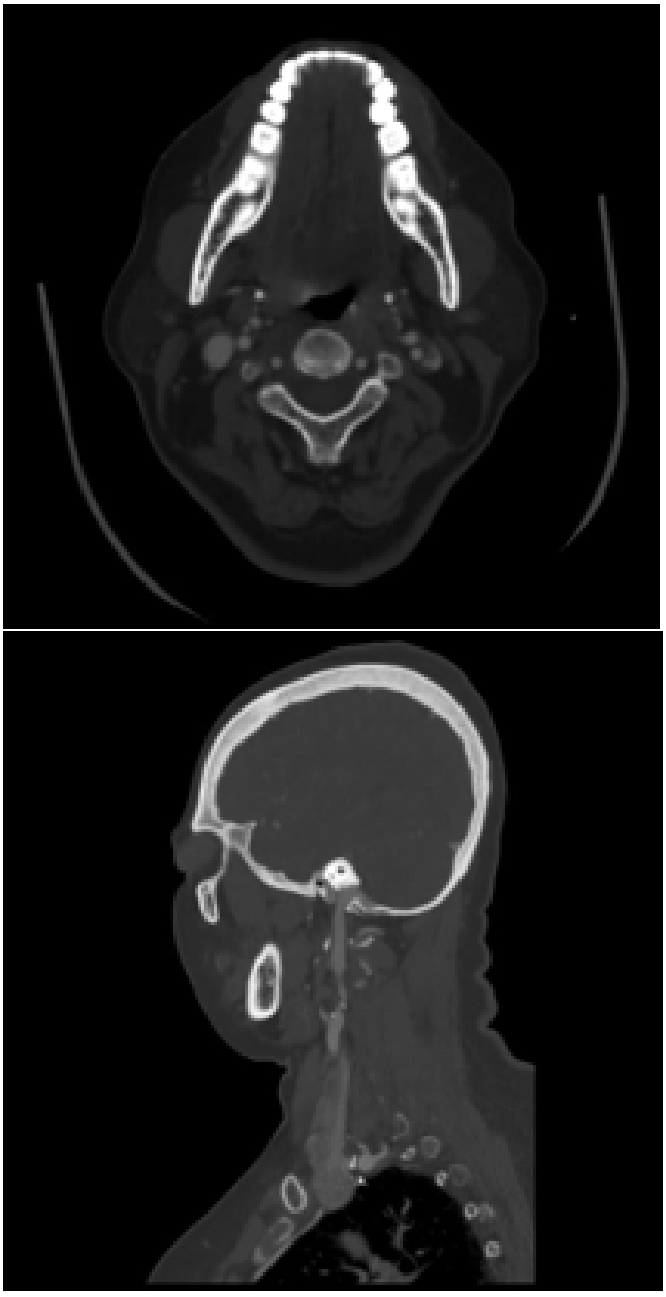

Figure 2: CTA carotid - axial and sagittal views

As part of a multidisciplinary team discussion, the patient underwent a thromboendarterectomy of the right internal carotid artery. Following surgery, regression in the patient's new vessel channel was observed during follow-up at the eye clinic, while the patient's IOP remained well controlled with a single anti-glaucoma medication. The patient has remained stable and has been doing well for over a year.

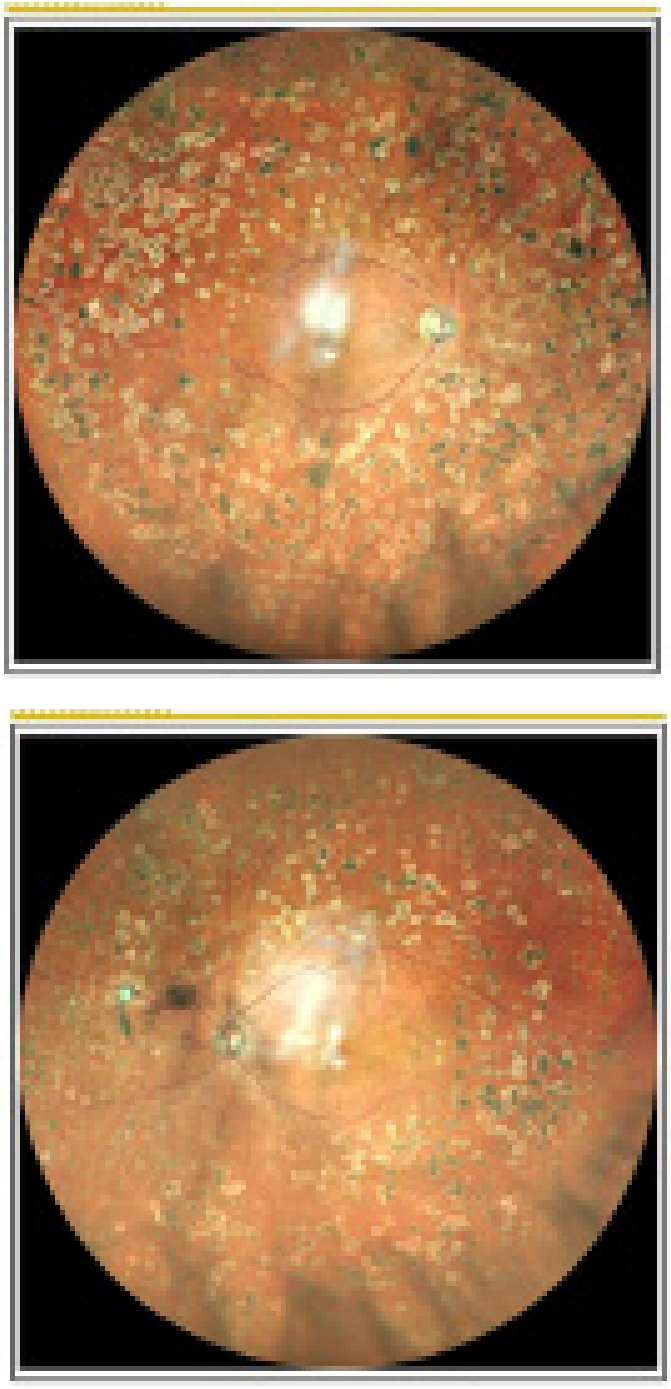

Figure 3: Fundus photo showing pan-retinal photocoagulation

Discussion

Neovascular glaucoma is caused primarily by three conditions: proliferative diabetic retinopathy, central vein occlusion, and, less commonly, ocular ischemic syndrome (OIS).[5]

The ocular ischemic syndrome is a rare disorder associated with carotid artery stenosis or occlusion, resulting in decreased blood supply to the eye and orbit.[6] Evidence-based research has shown that atherosclerosis plays a role in its pathogenesis by limiting the blood supply to the retina.[7] Several studies have identified risk factors for OIS, including hypertension (73%), diabetes mellitus (56%), and myocardial infarction (4%).[8] These risk factors are the same as those attributed to carotid artery disease. The estimated 5-year mortality rate following disease onset is approximately 40%.[8]

The literature contains limited reports of neovascular glaucoma secondary to carotid artery stenosis. This underscores the rarity of neovascular glaucoma occurring simultaneously secondary to PDR and COD.

The simultaneous occurrence of NVG, PDR, and carotid occlusive disease presents a unique challenge. Retinal ischemia, a consequence of diabetic retinopathy, stimulates VEGF production, resulting in neovascularization and glaucoma. The carotid occlusive disease further compromises retinal perfusion, exacerbating the ischemic process. These two distinct processes are interdependent in the formation of neovascular glaucoma.

A key pillar of the guidelines is anti-vascular endothelial growth factor therapy, which is effective in reducing the neovascularization of the iris the central pathogenic mechanism of this disease.

Another treatment modality is pan-retinal photocoagulation (PRP), which effectively addresses retinal ischemia and reduces the stimulus of neovascularization. Our patient underwent two sessions of PRP in addition to anti-VEGF treatment; however, the clinical response was minimal, as evidenced by the rapid recurrence of neovascularization after each course of treatment. As the next line of treatment, surgical intervention with a right-eye Ahmed glaucoma valve was performed after medical treatment yielded minimal results.

Surgery was indicated due to the severe neovascular glaucoma and the risk of irreversible vision loss. A right-eye Ahmed glaucoma valve procedure was performed, creating a filtration bleb and a reduction in intraocular pressure. Though the presence of neovascularization complicated the procedure, it was successful, achieving a postoperative IOP of 15 mmHg. Figure 4 shows the post-operative OCT.

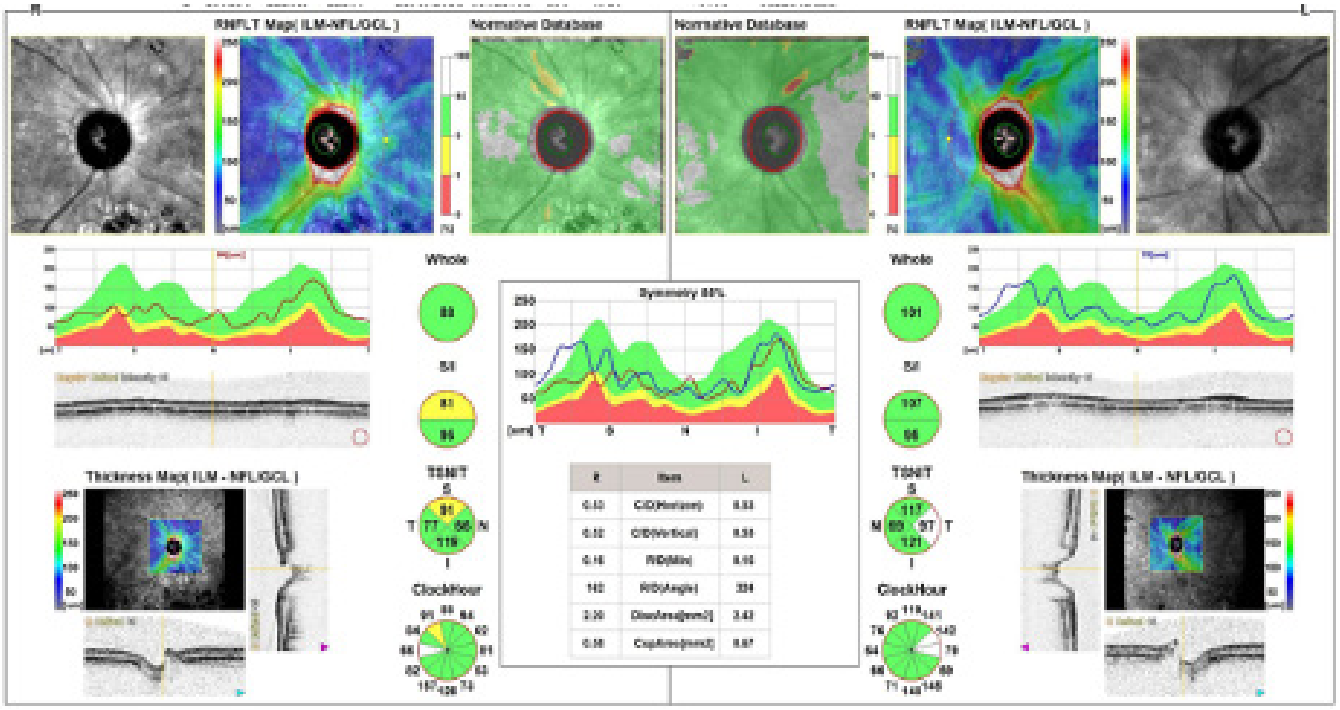

Figure 4: Post-operative Optical Coherence Tomography (OCT)

To address the proliferative diabetic retinopathy, the surgeon performed pan-retinal photocoagulation (PRP) in the left eye. The goal of this treatment was to reduce retinal ischemia and VEGF production, thereby preventing further neovascularization.

The underlying cause of this patient's neovascular glaucoma carotid artery stenosis likely explains the limited response to standard guideline-based medical therapy. The stenosis reduced the blood flow through the main branches of the ocular arterial supply, affecting both the retina and anterior segment. This also accounts for the marked improvement in the patient's IOP and regression of neovascularization following carotid endarterectomy. This case highlights the importance of clinical reasoning in identifying less common causes of disease processes, such as carotid artery stenosis, as contributors to treatment-resistant neovascular glaucoma.

Outcomes and Follow-Up

At the nine-month follow-up, the patient's ocular pain showed significant improvement, with IOP reduced to 16 mmHg. Visual acuity in the left eye remained stable at 6/6, with stabilization of retinal findings. The patient was advised to maintain strict glycemic control and was referred to a vascular specialist for further evaluation and management of carotid occlusive disease.

Conclusion

This case underscores the importance of recognizing the interplay between neovascular glaucoma, proliferative diabetic retinopathy, and carotid occlusive disease. A multidisciplinary approach involving ophthalmology and vascular specialists is crucial for optimal management. Early intervention and comprehensive treatment strategies can improve outcomes and help preserve both vision and overall health of the patients with these complex conditions. Regular follow-up and monitoring are essential to prevent recurrence and to manage potential complications.

Conflict of Interest

Nil

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