Case Report

Effective intraocular pressure management and vision preservation in secondary glaucoma associated with carotid-cavernous fistula: A case report

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ABSTRACT

Carotid-cavernous fistulas (CCFs) are abnormal arteriovenous connections between the cavernous sinus and branches of the internal or external carotid arteries. Although uncommon, CCFs are an important cause of secondary glaucoma due to increased episcleral venous pressure, and they present significant diagnostic and therapeutic challenges. Uncontrolled intraocular pressure (IOP) can lead to irreversible blindness, highlighting the urgency of timely and effective management. We report the case of a 65-year-old woman with a history of hypertension and dyslipidemia who presented with persisting bilateral redness and dry sensation in both eyes (BE), along with visual decline, left-sided proptosis, conjunctival injection (more severe in the left eye [LE]), and a palpable bruit. IOP was elevated in the LE despite maximal topical therapy. Imaging with computed tomography angiography and digital subtraction angiography confirmed a Type C CCF. This case highlights the importance of addressing the underlying cause of secondary glaucoma, as maximal anti-glaucoma medications alone may be insufficient if the primary pathology remains unresolved.

Key words: Carotid cavernous fistula, Carotid compression, Intraocular pressure, Secondary glaucoma, Visual acuity

Carotid-cavernous fistulas (CCFs) are rare vascular shunts between the carotid artery and the cavernous sinus (CS) [1]. Increased episcleral venous pressure (EVP) and venous congestion can impair the drainage of aqueous humor through the trabecular meshwork and Schlemm's canal, increasing intraocular pressure (IOP) and subsequently leading to secondary glaucoma [2]. Glaucoma or elevated IOP has been reported in 6.5 to 79% of patients with CCF [3]. Managing secondary glaucoma following CCF is complex and often requires a multidisciplinary approach. IOP control may not be achievable with anti-glaucoma medications; addressing the underlying vascular pathology is crucial for effective IOP control and for preventing irreversible glaucomatous damage.

We present the case of a 65-year-old woman diagnosed with secondary glaucoma due to indirect (Type C) CCF. This case is reported to highlight the diagnostic and therapeutic challenges of secondary glaucoma associated with indirect CCF, particularly when IOP remains uncontrolled despite maximal medical therapy. The case emphasizes the importance of identifying and addressing the underlying vascular pathology, as conventional anti-glaucoma medications alone may be insufficient. In addition, this report demonstrates the role of non-invasive carotid compression therapy as an effective conservative option in selected low-flow CCF cases, resulting in IOP stabilization and visual preservation.

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CASE REPORT

A 65-year-old woman was referred with persistent redness and a dry sensation in both eyes (BE) for 7 months, accompanied by intermittent periorbital swelling. She denied experiencing visual disturbances, periorbital pain, or pain associated with eye movements. Her medical history was significant for hypertension and dyslipidemia, both of which were well-managed with medications.

On presentation, she was hemodynamically stable with blood pressure of 135/80, pulse rate of 78 beats/min, respiratory rate of 18 breaths/min, and oxygen saturation of 98% on room air. There was no history of trauma, prior ocular disease, or surgery.

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On local examination, best-corrected visual acuity (BCVA) was 6/7.5 in the right eye (RE) and 6/19 in the left eye (LE). Proptosis was evident in the LE, conjunctival injection, and prominent corkscrew vessels in BE, more pronounced in the LE (Fig. 1a and 1b). IOP measured 17 mmHg in the RE and 27 mmHg in the LE, despite ongoing treatment with topical anti-glaucoma medications. Fundoscopic examination revealed a cup-to-disc ratio (CDR) of 0.3–0.4 in BE. Computed tomography angiography and digital subtraction angiography (DSA) confirmed a low-flow Type C CCF (Fig. 2a and b).

Initial management consisted of a combination of oral and topical anti-glaucoma medications. However, IOP in the LE remained persistently elevated despite maximal medical therapy. Micropulse laser treatment was attempted but failed to control IOP, while CDR progressively increased to 0.6–0.7. Repeat DSA reconfirmed the diagnosis, and carotid compression therapy was recommended.

The patient was instructed to perform self-administered intermittent carotid compression multiple times daily, while continuing topical and systemic anti-glaucoma medications. Clinical signs improved significantly. Complete resolution of redness in the RE, gradual improvement in the LE (Fig. 3), along with stabilization of IOP (Fig. 4a) and improvement in BCVA (Fig. 4b). On subsequent follow-up visits, IOP remained within the target range, VA was maintained. There was a gradual reduction of redness in the LE, and no recurrence of ocular congestion.

DISCUSSION

CCFs are abnormal arteriovenous connections characterized by the redirection of blood flow from a high-flow arterial system [2]. The Barrow classification divides CCFs into four subtypes. Direct fistulas (Type A) are high-flow communications between the ICA and the CS. In contrast, indirect fistulas (Type B, C, and D) are low-flow shunts. Type B involves meningeal branches of ICA, Type C meningeal branches of ECA, and Type D consists of contributions from both ICA and ECA [4]. Direct CCFs most commonly occur after trauma in young males and account for 75% of CCFs. Meanwhile, indirect CCFs are typically insidious in onset and more common among elderly, hypertensive post-menopausal women and account for 30% of cases [1,5]. In the present case, the patient demonstrated known predisposing factors that have been linked to the formation of indirect CCF.

The CS contains multiple cranial nerves and ICA, making ocular symptoms and signs common in CCFs. Both types of CCFs generally cause elevated IOP [5]. Conception PA and FlorCruz II NV [6] reported that 78% of eyes had elevated IOP, and Ishijima *et al*. [7] reported elevated IOP in 64.3%. Glaucoma can cause irreversible blindness, especially in cases of untreated CCF [3]. Two mechanisms for IOP elevation in CCFs are the open-angle mechanism, due to increased EVP impairing aqueous outflow, and the closed-angle mechanism, involving choroidal and ciliary body congestion leading to anterior chamber swelling. In this case, IOP elevation was consistent with the open-angle mechanism, which aligns with previous studies reporting

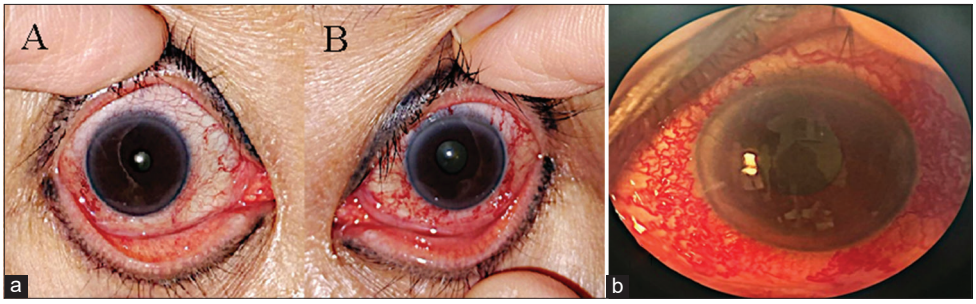

Figure 1: (a) Initial presentation of right eye (a) and left eye (LE) (b) demonstrating conjunctival injection, chemosis, and engorgement of episcleral vessels, (b) slit-lamp image of the LE showing corkscrew appearance

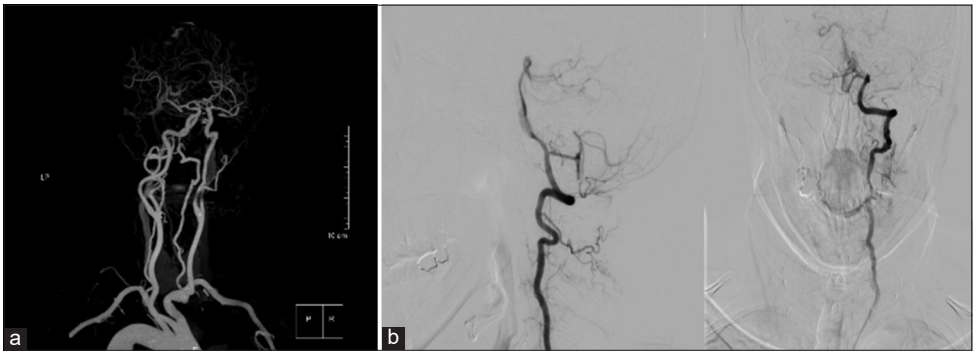

Figure 2: (a) Computed tomography angiography demonstrating indirect carotid-cavernous fistula (Type C) with abnormal vascular channels and (b) digital subtraction angiography revealing a low-flow indirect carotid-cavernous fistula (Type C) with venous drainage into the cavernous sinus

this mechanism in the majority of cases. In a previous study, 7 out of 9 eyes (77.8%) developed elevated IOP through the open-angle mechanism. Comparable findings were noted in another study, which reported open-angle involvement in 85% of cases [7].

In indirect CCFs draining into the superior ophthalmic veins (SOV) and inferior ophthalmic veins (IOV), symptoms include pulsatile proptosis, diplopia, episcleral congestion and chemosis, a red eye, and ocular bruit. On the other hand, posteriorly draining fistulas, through the petrosal sinuses, symptoms may be mild and difficult to interpret (white-eye syndrome) [8,9]. In our case, clinical findings were indicative of drainage into the SOV and IOV, as evidenced by proptosis, red eye, and an orbital bruit.

The optimal management of secondary glaucoma following indirect CCF can be quite challenging. Spontaneous closure of CCF may occur in 11–70% of cases, making observation and conservative management preferred in low-risk cases [2,8]. Topical agents remain the first-line therapy for CCF-induced IOP. However, due to elevated EVP, aqueous suppressants may have limited efficacy, and prostaglandin receptor agonists may also be less effective due to arterial blood reflux [2]. In another study, it was found that 78% of CCF cases experienced increased IOP, with 36.5% achieving control with medications alone. In contrast, in our case, anti-glaucoma medications alone were insufficient to control IOP [7].

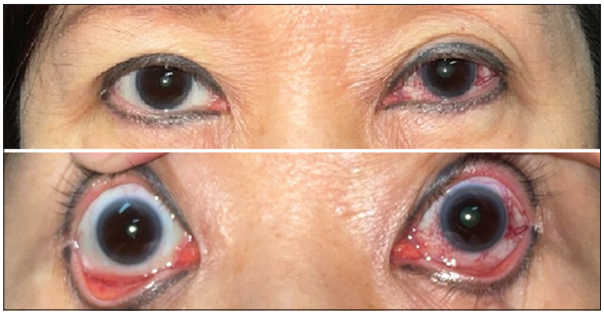

Figure 3: Complete resolution observed in the right eye and marked improvement of the left eye following combination of carotid compression and anti-glaucoma medications

Khurana *et al*., reported that 64.1% of patients with CCF had elevated IOP or glaucoma, mainly through an open-angle mechanism. IOP control was achieved in 70.73% using combined treatment, indicating that medications alone are often insufficient [3]. In our case, a combination of non-invasive carotid compression therapy was initiated alongside anti-glaucoma medications. Conservative management with manual external compression of the cervical carotid artery several times a day may be effective in low-flow fistulas without cortical venous drainage or progressive visual/neurological decline. Carotid compression therapy has been reported to achieve closure rates of approximately 17% in direct CCFs and 30% in indirect CCFs [2,10].

When maximal medical therapy fails to control IOP, the definitive treatment for CCF is endovascular embolization, with reported success rates ranging from 70 to 100% [2]. However, in our case, embolization was not pursued due to DSA findings showing minimal shunting that was difficult to visualize. Moreover, embolization can impose a considerable systemic burden, particularly in elderly patients, making it a less favorable option [7]. Therefore, in cases such as ours, it is important to consider less invasive alternatives. A low-risk procedure, such as carotid compression therapy, can be a viable approach to reduce or close the fistula as much as possible.

A noticeable reduction in IOP was observed following the initiation of carotid compression therapy by the patient, accompanied by an improvement in BCVA. These clinical improvements strongly suggest that addressing the underlying vascular anomaly was crucial in achieving better ocular pressure control. This observation aligns with findings from a case study in which IOP returned to normal levels after closure of the CCF. Their study further emphasized that conventional antiglaucoma medications may be less effective, and that definitive management of the fistula, either through spontaneous closure or interventional procedures, can result in more effective IOP regulation [7]. This was consistent with our case, where despite the use of

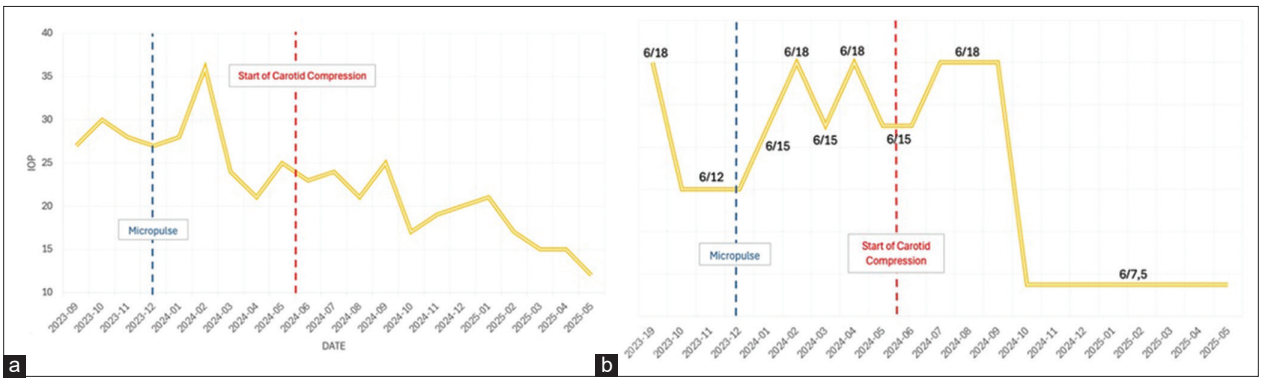

Figure 4: (a) Intraocular pressure (IOP) trend. Note the gradual and sustained decline in IOP following the initiation of carotid compression therapy; (b) VA trend. Note the gradual and sustained decline in IOP following the initiation of carotid compression therapy

multiple IOP-lowering agents, adequate pressure control was only achieved following initiation of non-invasive carotid compression therapy.

CONCLUSION

This case highlights the importance of addressing the underlying cause of secondary glaucoma, as anti-glaucoma therapy alone may be insufficient if the primary pathology remains unresolved. Indirect CCFs can sometimes resolve spontaneously; however, in cases where sight- threatening glaucoma develops, a more proactive approach is warranted. In this patient, frequent external carotid artery compression was initiated to promote spontaneous closure of the fistula, in conjunction with intensive glaucoma therapy, to achieve rapid IOP control and prevent further visual deterioration.

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