Case Report

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Ophthalmology Section

Co-existing Carotid-Cavernous Fistula and Central Retinal Vein Occlusion Presenting as Unilateral Vision Loss in a 30-Year-Old Male: A Case Report

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ABSTRACT

Carotid-Cavernous Fistulas (CCFs) are aberrant arteriovenous connections between the carotid arteries and the cavernous sinus. Although uncommon, they might manifest with subtle or deceptive symptoms, especially in young individuals with no past medical history. This case study describes the clinical appearance, diagnosis, and treatment of a direct CCF in a 30-year-old male. The patient had a history of trauma three months prior to presentation followed by chief complaints of diminution of vision and swelling in the right eye. There was sudden increase in pain and swelling since 2 weeks prior to presentation. Patient underwent CT scan, ocular examination, fundus evaluation which revealed the presence of a direct carotid cavernous fistula along with Central Retinal Vein Occlusion (CRVO), following which surgical intervention was done. The patient had successful endovascular embolisation. Following surgery, ocular symptoms and proptosis along with visual acuity improved significantly. Early detection of CCFs is crucial for avoiding vision-threatening consequences. Clinicians should be cautious of young patients who have unexplained red eye, proptosis, as well as visual impairment, particularly after trauma. Prompt diagnosis using appropriate imaging and swift intervention can result in positive outcomes.

Keywords: Carotid artery, Cavernous sinus, Endovascular embolisation, Proptosis, Ocular trauma

CASE REPORT

A 30-year-old male patient reported to the Outpatient Department of Ophthalmology, with the chief complaint of diminution of vision along with swelling in the right eye three months ago following a road traffic accident due to a two-wheeler. The symptoms were gradual on onset and progressive but were painless in nature and the patient did not consult any physician. Eventually the patient complained of sudden increase in swelling and pain since last two weeks of reporting to the department. Pain was throbbing in nature and was relieved after taking analgesic medication. The patient had no other significant ocular or systemic history besides trauma [Table/Fig-1].

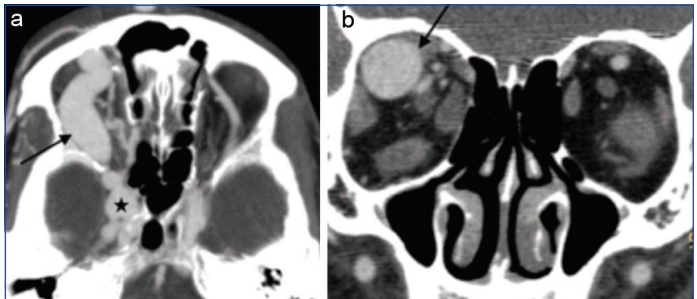
On examination of the right eye, visual acuity was limited to hand movements with present light perception but inaccurate projection of rays, and the eye showed oedema, ecchymosis, matted eyelashes, conjunctival prolapse, and a whitish discharge.

[Table/Fig-1]: Shows a 30-year-old male with right eye proptosis with severe haemorrhagic chemosis of the conjunctiva and bulging of the lower palpebral conjunctiva. The upper and lower eye lids are oedematous and inflammatory, with an erythematous protrusion indicating conjunctival prolapse and orbital congestion.

Extraocular examination revealed pulsating proptosis along with completely restricted movement in all gazes. Orbital margins were tender. Examination of the conjunctiva revealed considerable haemorrhagic chemosis along with diffuse congestion. Furthermore, episcleral injection was present, indicating dilated and engorged episcleral vessels, which are frequently linked with high episcleral venous pressure or inflammation. These findings are consistent with ocular surface inflammation as well as vascular congestion, which can be seen in conditions like CCF or other orbital vascular pathologies.

The right eye had a single, uneven, centrally positioned greyish-black pupil around 5 mm in diameter. The borders were regular, but the pupil was mid-dilated and non-reactive (no direct or consensual response) to light. These characteristics being frequently associated with severe ocular trauma, elevated intraocular pressure, or third nerve dysfunction.

A contrast-enhanced CT scan of the brain and orbit revealed a displaced fracture of the right frontal bone involving the walls of the right frontal sinus along with the roof of the right orbit [Table/Fig-2].

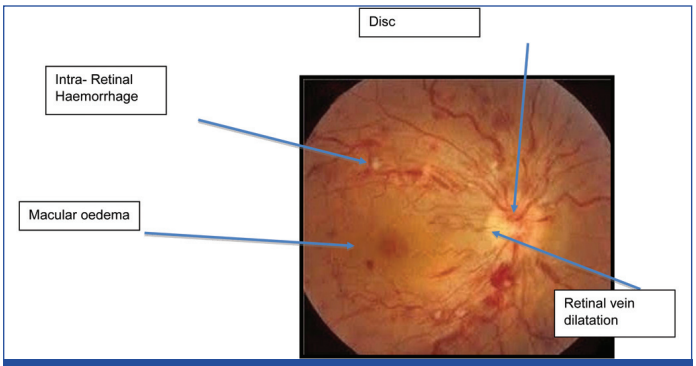


[Table/Fig-2a,b]: Contrast enhanced CT Head suggestive of right frontal bone fracture, Dilated right superior ophthalmic vein (arrow), bulging right cavernous sinus (asterisk) and bulky extraocular muscles on the right-side.

[Table/Fig-2a] shows a well-defined, rounded soft-tissue density lesion in the superior-lateral orbit, causing downward displacement of the globe along with mass effect on surrounding orbital structures, with adjacent fat stranding (marked by the star) suggesting secondary inflammation or pressure-related changes and [Table/Fig-2b] shows well-defined, round, soft-tissue density mass in the superior aspect of the right orbit causing displacement of adjacent orbital structures (marked by arrow).

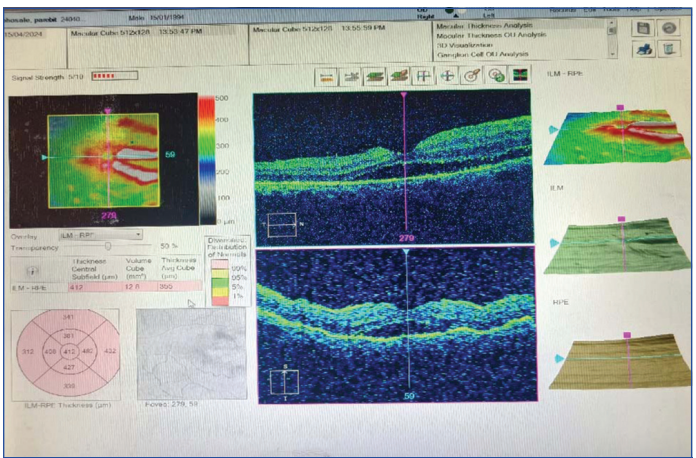
The imaging also revealed right eye proptosis with dilated and convoluted superior ophthalmic veins. There was also bulging and expansion of the right cavernous sinus, which supported the diagnosis of a CCF on the right-side. Notably, the globes and extraocular muscles seemed normal bilaterally, and the optic nerves and chiasm revealed no anomalies, indicating that the visual pathways were structurally intact at the time of imaging.

Fundus examination of the right eye exhibited clear ocular media and a hyperaemic optic disc. The retinal blood vessels were tortuous with superficial haemorrhages in all four quadrants, indicating vascular congestion. The foveal reflex was dull with intraretinal thickening at the macula. These findings were suggestive of CRVO in the right eye [Table/Fig-3].



[Table/Fig-3]: The right eye of the patient showing 4-point sign: disc hyperaemia, retinal vein dilatation, macular oedema and intra-retinal haemorrhages.

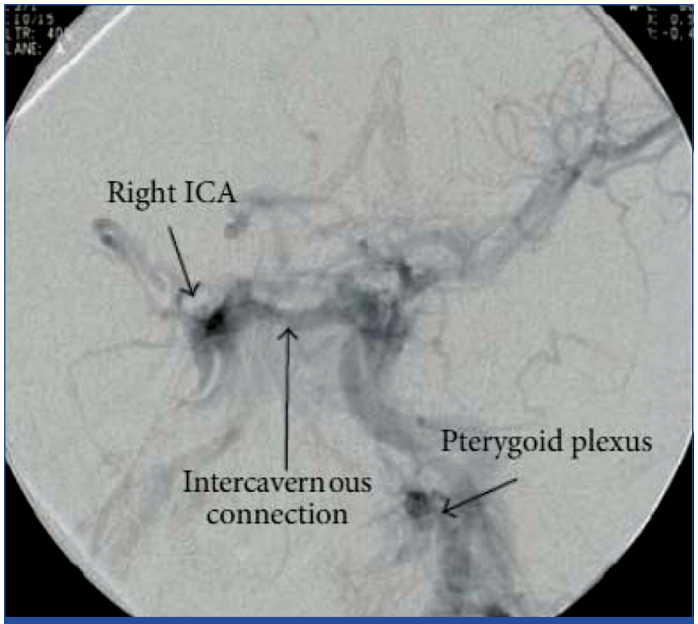
Optical Coherence Tomography (OCT) revealed presence of subretinal fluid at the macula which was suggestive of Central Serous Chorioretinopathy (CSCR) [Table/Fig-4].



[Table/Fig-4]: OCT evaluation showing the evidence of subretinal fluid which was suggestive of central serous retinopathy.

Based on the clinical findings as well as imaging studies (contrast-enhanced CT and digital subtraction angiography) [Table/Fig-2a,b,5], patient was started on i.v. methylprednisolone (pulse therapy)- 1 gm/day for three days along with adjunctive ocular lubricants and topical antimicrobial cover.

The patient was then scheduled for embolisation of corticocavernous fistula. Under all the aseptic precautions, the right femoral artery access was taken with 6F sheath. Cannulation of right common carotid artery was done with 6F envoy guiding catheter following the selective cannulation of right ICA was done. USG guided right facial



[Table/Fig-5]: Digital Subtraction Angiography (DSA) showing left-sided high-flow direct Type A Carotid-Cavernous Fistula (CCF), filling defect of the right cavernous sinus and right pterygoid plexus via inter-cavernous connections.

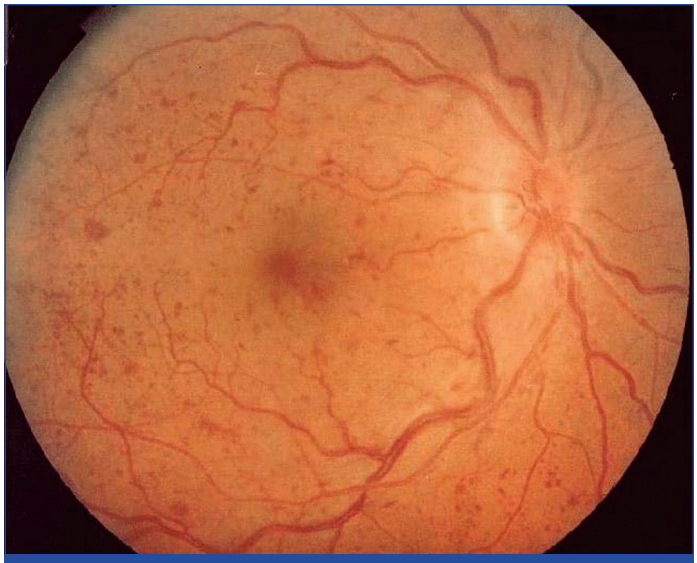
vein was punctured and 5F destination sheath was placed. The carotid–cavernous fistula was crossed using a Progreat catheter and a 0.014° Traxcess wire. Embolisation was done using 3 coils of Boston Scientific Interlock 14 mm×50 cm, Boston Scientific Interlock 14 mm×50 cm and Boston Scientific Interlock 10 mm×50 cm. The postsurgical angiogram demonstrated exclusion of the fistula. The right middle cerebral artery, anterior cerebral artery and internal cerebral artery appeared normal.

The second surgical procedure was undertaken for craniofacial arteriovenous malformation embolisation. Under all the aseptic precautions the right femoral artery was accessed using 5F sheath. Selective cannulation of branch of the right internal carotid artery was done using a picard. The cannulation of the capillary channels near the region of pituitary region was followed by Progreat microcatheter being passed through demonstrating Arteriovenous Malformation (AMV). Embolisation was done using glue and lipiodol. The completion angiogram revealed the presence of good embolisation of the AMV.

The patient was closely monitored postoperatively, with a repeat ophthalmic assessment to determine intraocular pressure, retinal perfusion, and orbital congestion regression. This method not only regulated high-flow CCF, but it also reduced venous stasis that contributes to CRVO, conserving visual potential and preventing additional ocular morbidity [Table/Fig-6]. The patient tolerated the surgery well and was transferred to the critical care unit for further neurological and ophthalmic monitoring where patient was started on oral prednisolone as per body weight in tapering doses and ocular lubricants. Patient was discharged on postoperative 3rd day and follow-up was scheduled after seven days. Visual symptoms and orbital signs improved gradually over the next few days [Table/Fig-7] with vision improving to BCVA 6/36 P and significant external ocular restoration as well.

DISCUSSION

The confluence of CRVO along with CCF after trauma is an uncommon but clinically important occurrence [1]. CCF, especially the direct (high flow) form, is frequently linked with severe craniofacial trauma and can cause venous congestion in the orbit resulting from increased pressure in the superior ophthalmic vein and cavernous sinus. This increased venous pressure can impair retinal venous outflow, possibly leading to CRVO [2]. The observed CRVO in our patient is most likely caused by a trauma-induced CCF followed by retinal vascular stasis.



[Table/Fig-6]: Post-embolisation fundus showing resolving subretinal fluid, decreased vascular dilatation and resolving haemorrhages.

[Table/Fig-7]: In contrast to the previous appearance, proptosis and conjunctival prolapse in the right eye were resolved. Minor ectropion, lid oedema and conjunctival congestion remain apparent.
Eventually the patient was lost to follow-up.

Direct CCFs are typically caused by head trauma, such as motor vehicle accidents or penetrating injuries, but can also be caused by the rupture of an intra-cavernous aneurysm or head surgery [2-4]. Our patient arrived with direct CCF caused by a motor car collision. Clinical signs of direct CCFs are often sudden in start with fast development, needing immediate treatment [2,5]. In contrast, indirect CCFs are idiopathic and have a more insidious start [6]. Indeed, the severity of symptoms is determined by venous return capacity, as well as the quantity and rate of blood flow. Most cases have a combination of anterior and posterior drainage, with the former going into the ophthalmic veins and the latter going into the petrosal sinuses. The most severe symptomatology is found in cases with anterior drainage. Orbital symptoms are less severe when venous drainage is posterior [7].

The main symptoms of CCFs are orbital bruit, which can be heard by patients as well as objectively determined by the physician, proptosis, chemosis, arterialisation of the episcleral veins with the characteristic corkscrew-like aspect, eye lidoedema, diplopia, visual impairment of varying degrees, and elevated intraocular pressure. Ophthalmologic examination may also detect dilatation of retinal veins, intraretinal haemorrhages, minor optic disc enlargement, and even non-rhegmatogenous retinal and choroidal detachments [8].

One of the most feared CCF consequences is vision loss. Direct CCF may result in sight- threatening consequences such as severe exposure keratopathy, corneal ulcerations, and central retinal artery blockage [9]. There is currently no data on the incidence of vision loss if direct CCF is not treated adequately and on time. The differential diagnosis of CCFs include cerebral aneurysms, vascular abnormalities of the eyes, inflammation of the orbits, retro-orbital cellulitis, thyroid exophthalmos, retrobulbar haemorrhage, lacrimal gland tumour, cavernous sinus thrombosis, and vasculitis [10].

Endovascular therapy is usually successful for carotid cavernous fistulas. If the endovascular approach is deemed inappropriate or ineffective, surgical intervention becomes an option. The cavernous sinus fistula can be repaired directly [8]. Matsuda Y et al., indicated that accessing the cavernous sinus via the pterygoid venous plexus can be a useful alternate approach when traditional venous channels (e.g., inferior petrosal sinus) are inaccessible. The authors observed effective fistula closure while maintaining ICA patency with complete resolution of ocular symptoms, highlighting the use of this unusual but safe transvenous approach in complicated anatomy [11]. In other two cases of direct CCF reported by Lin LM et al., it was seen that despite the changed vascular architecture caused by earlier stenting, effective coil embolisation was possible via the inferior petrosal sinus or alternate vein pathways. Both the patients achieved angiographic fistula closure and clinical improvement, demonstrating transvenous treatment as a feasible and safe alternative when arterial access is restricted by the flow diverter [12]. Hamano E et al., described in a technical note with detailed bidirectional (arterial and venous) double-catheter method that enabled precision microcatheter placement and controlled coil deployment. The approach allowed for full obliteration of the fistula while preserving ICA flow. The method increased procedural stability, safety, and packing density, which was useful in anatomically demanding direct CCFs [13].

CONCLUSION(S)

This case emphasises the significance of keeping a high index of suspicion for direct CCF in young patients who arrive with unexplained unilateral red eye, proptosis, ocular discomfort, and abrupt vision impairment, especially if they have a history of trauma. Early diagnosis by adequate imaging and extensive ocular assessment is critical, since delayed detection can result in permanent vision loss and other significant problems. The patient's successful restoration of visual function following endovascular embolisation demonstrates the efficacy of timely interventional therapy. Overall, the case emphasises the importance of early detection, fast diagnostic workup, and coordinated multidisciplinary therapy to optimise visual and anatomical results in patients with traumatic CCF.

REFERENCES

[1] Blair K, Czyz CN. Central retinal vein occlusion. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK525985/.
[2] Gemmete JJ, Chaudhary N, Pandey A, Ansari S. Treatment of carotid cavernous fistulas. Curr Treat Options Neurol. 2010;12(1):43-53. Doi: 10.1007/s11940-009- 0051-3.
[3] Ringer AJ, Salud L, Tomsick TA. Carotid cavernous fistulas: Anatomy, classification, and treatment. Neurosurg Clin N Am. 2005;16(2):279-95. viii. Doi: 10.1016/j.nec.2004.08.004.
[4] Guimarães AC, De Carvalho GM, Chone CT, Pfeilsticker LN. Carotid cavernous fistula: A rare complication of maxillofacial trauma. Head Neck Oncol. 2014;6(3):23.
[5] Barry RC, Wilkinson M, Ahmed RM, Lim CS, Parker GD, McCluskey PJ, et al. Interventional treatment of carotid cavernous fistula. J Clin Neurosci. 2011;18(8):1072-79. Doi: 10.1016/j.jocn.2010.12.026.
[6] Ellis JA, Goldstein H, Connolly ES, Jr, Meyers PM. Carotid-cavernous fistulas. Neurosurg Focus. 2012;32(5):E9. Doi: 10.3171/2012.2.FOCUS1223.

[7] Ajhoun Y, El Khoyali A, Aissa I, Laaribi N, Mouzari Y, Reda K, et al. Direct carotid-cavernous fistula: An atypical presentation. Pan Afr Med J. 2019;34:204. Doi: 10.11604/pamj.2019.34.204.19445.
[8] Naesens R, Mestdagh C, Breemersch M, Defreyne L. Direct carotid-cavernous fistula: A case report and review of the literature. Bull Soc Belge Ophtalmol. 2006;(299):43-54.
[9] de Keizer R. Carotid-cavernous and orbital arteriovenous fistulas: Ocular features, diagnostic and hemodynamic considerations in relation to visual impairment and morbidity. Orbit. 2003;22(2):121-42. Doi: 10.1076/orbi.22.2.121.14315.
[10] Pülhorn H, Chandran A, Nahser H, McMahon C. Case report: Traumatic carotid- cavernous fistula. J Trauma Nurs. 2016;23(1):42-44. Doi: 10.1097/JTN.0000000000000174.
[11] Matsuda Y, Hiramatsu M, Sugiu K, Hishikawa T, Haruma J, Nishi K, et al. Transvenous embolization of the direct carotid-cavernous fistula via the pterygoid plexus: Illustrative case. J Neurosurg Case Lessons. 2023;5(11):CASE22558. Doi: 10.3171/CASE22558.
[12] Lin LM, Colby GP, Jiang B, Pero G, Boccardi E, Coon AL. Transvenous approach for the treatment of direct carotid cavernous fistula following Pipeline embolization of cavernous carotid aneurysm: A report of two cases and review of the literature. BMJ Case Rep. 2014;2014:bcr2014011235. Doi: 10.1136/bcr-2014-011235.
[13] Hamano E, Satow T, Hori T, Takahashi JC, Kataoka H. A case of direct carotid-cavernous fistulae successfully treated by bidirectional double catheter technique: A technical note. J Neuroendovasc Ther. 2022;16(6):307-12. Doi: 10.5797/jnet.cr.2021-0052.

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