Case Report

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A "Mismatched" connection: a rare case of indirect or dural carotid-cavernous fistula: a case based review

Ruchit Jain, MBBS[a], Nitin Jagdhane, MBBS, MS, MCh[b], Sameer Deshmukh, MBBS[c], Stefano M. Priola, MD[d], Vishal Chavda, MD[e], Bipin Chaurasia, MS[f,*]

Introduction and importance: A carotid-cavernous sinus fistula is an abnormal connection between the internal or external carotid artery and the venous system of the cavernous sinus. It represents a rare entity, and it is often misdiagnosed due to its overlapping symptoms with other conditions such as cavernous sinus thrombosis or orbital inflammation. Cerebral angiography continues to be the gold standard for diagnosis and surgical planning in patients with CCF, and the endovascular trans-venous approach still represents the primary line of treatment. Trans-arterial technique has become an excellent treatment option since the advent of embolic agents.

Case presentation: Here, the authors report the case of a 42-year-old male with a one-week history of impaired visual acuity, bilateral eye edema, eye redness, and ophthalmoplegia. A neuro-ophthalmological examination found proptosis, conjunctival chemosis, and sclera injection. Examination using MR venography and digital subtraction angiography (DSA) revealed an abnormal connection between the meningeal branch of the ICA, the ECA, and the cavernous sinus. The patient received endovascular treatment with Onyx injection into the facial vein with thrombosis of the anterior cavernous sinus. The patient presented an uneventful postoperative period and was symptoms-free on postoperative day 1.

Clinical discussion: CCF are rare and challenging conditions that require a multidisciplinary approach.

Conclusion: The endovascular treatment represents the gold standard and usually allows an effective interruption of the abnormal vascular connection with an almost immediate resolution of the preoperative signs and symptoms.

Keywords: carotid-cavernous fistula, cavernous sinus, proptosis

Introduction

Carotid-cavernous sinus fistulas (CCFs) are rare conditions based on an abnormal vascular connection between the carotid artery and the cavernous sinus[1].

They clinically present with a classic triad: proptosis, eye congestion, and signs of turbulent flow into the cavernous sinus. Treatment is crucial to interrupt the abnormal communication decreasing the venous pressure and restoring the normal venous outflow from the orbit[2–6].

HIGHLIGHTS

- A 42-year-old patient had impaired vision and proptosis, conjunctival chemosis, and sclera injection.
- Digital subtraction angiography (DSA) revealed an abnormal connection between the meningeal branch of the ICA, the ECA, and the cavernous sinus.
- The patient received endovascular treatment with Onyx injection into the facial vein with thrombosis of the anterior cavernous sinus. The patient presented an uneventful postoperative period and was symptoms-free on postoperative day 1.

Here, we report a rare case of bilateral carotid-cavernous fistula, and we review the classification, clinical presentation, pathophysiology, diagnosis and treatment of CCFs. We followed the SCARE 2023 criteria[7] to present our case. We report this case because indirect fistulas, particularly Barrow type D, are quite uncommon.

Case report

A 42-year-old male was admitted to our Institution with a 1-week history of impaired visual acuity, bilateral eye edema, eye redness, and ophthalmoplegia. A full neuro-ophthalmological examination was completed showing proptosis, conjunctival chemosis, and sclera injection (Fig. 1).

[a]Department of Surgery, Government Medical College, Miraj, [b]Department of Neurosurgery, Dr. D.Y. Patil Medical College, Navi Mumbai, Maharashtra, India, [c]Dr. Shankarrao Chavan Government Medical College, Nanded, Maharashtra, India, [d]Division of Neurosurgery, Health Sciences North, Northern Ontario School of Medicine University, Sudbury (ON), Canada, [e]Department of Pathology, Stanford school of Medicine, Stanford, CA, USA and [f]Department of Neurosurgery, Neurosurgery Clinic, Birgunj, Nepal

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*Corresponding author. Address: Department of Neurosurgery, Neurosurgery Clinic, Birgunj, Nepal. Tel.: +977 984 545 4636. E-mail: trozexa@gmail.com (B. Chaurasia).

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As further investigation, the patient underwent a brain MRI/MRA (Fig. 2), that showed an absence of flow into the left cavernous, which was suggestive of thrombosis. As a result, it was presumed that the patient had cavernous sinus thrombosis.

The symptoms persisted despite the medication. Further examination using MR venography (Fig. 3) and digital subtraction angiography (DSA) (Fig. 4) revealed an abnormal connection between the meningeal branch of the ICA, the ECA, and the cavernous sinus.

After case review at the neuroendovascular meeting, the patient was brought into the operating room for embolization (Fig. 5). Using a trans-venous approach through the left facial vein, Onyx was injected into the anterior cavernous sinus and thrombosis was achieved. The patient well tolerated the procedure and was symptom-free on postoperative day 1 (Fig. 6). The work has been reported in line with the SCARE 2023 criteria[7].

Discussion

Anatomy

The dural venous sinus (Fig. 7) includes the cavernous sinus, which is home to a number of neuro-vascular structures. It is located symmetrically lateral to the sella turcica, it measures about 1 cm in width and 2 cm in length, and it runs from the superior orbital fissure to the petrous part of the temporal bone.

The superficial middle cerebral vein, the superior and anterior ophthalmic veins, and the sphenoparietal sinus all supply the cavernous sinus with venous blood; the intercavernous sinuses, anterior and posterior, provide communication between the left and right cavernous sinuses[8,9].

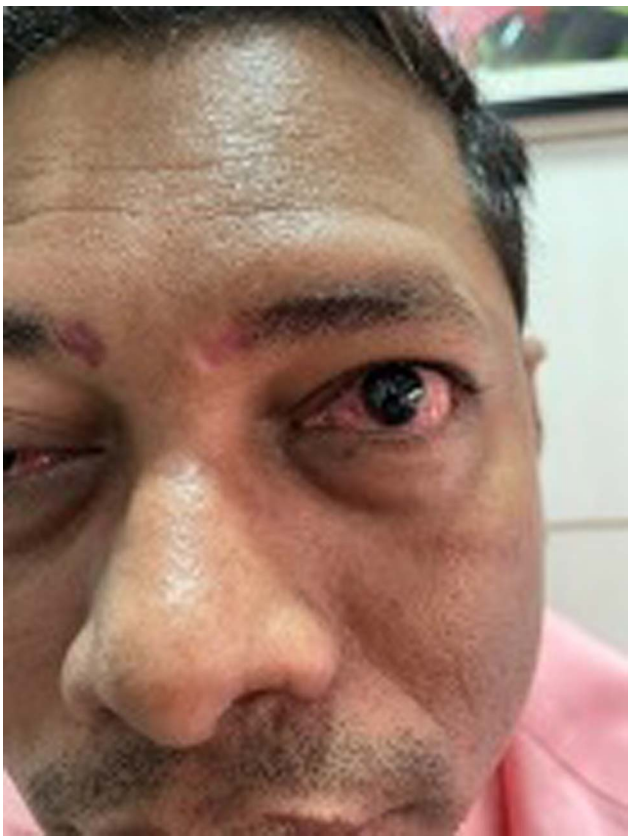

Figure 1. Neuro-ophthalmic assessment of the patient.

Arterial anatomy

Throughout its path through the CS, the ICA gives rise to a number of arterial branches, the largest of which is the meningo-hypophyseal trunk. This vessel emerges from the dorsal perimeter of the C5 segment of the ICA, just before the vertical part turns into the horizontal one.

The inferior hypophysial artery was initially described by Luschka in 1860 and it supplies the anterior hypophyseal artery, the tentorial artery, and the lateral clival artery. The tentorium's dura is also supplied by the artery of Bernasconi–Cassinari (lateral clival artery or dorsal meningeal artery).

The infero-lateral trunk, which supplies the portion of the cranial nerves going into the lateral sellar compartment, and the capsular artery of McDonnel are two more rather constant branches. The ICA and ECA's branches are constantly anastomosed even if it is not always angiographically evident.

Knowledge of these anastomotic channels is crucial in preventing complications during endovascular approaches[10].

Venous anatomy

For the understanding of the angiographical anatomy of cavernous sinus dural arteriovenous fistulas (CSDAVFs), it is essential to understand how the venous channels related to the cavernous sinus (Fig. 8).

The anterior part of the CS receives regular venous drainage from the orbit through the superior and inferior ophthalmic veins. Additionally, the sphenoparietal sinus serves as the typical entry point for the superficial middle cerebral veins into the CS. The circular sinus is created when the two separated CSs interact via the anterior and posterior intercavernous sinuses. To reach the jugular bulb, posterior drainage passes through the basilar plexus, the superior petrosal sinuses, and the inferior petrosal sinuses. Connections can be found inferolaterally through the dural veins that drain into the pterigoid plexus.

Classification

CCFs are usually classified according anatomy, origin, and hemodynamic features.

Based on their hemodynamic characteristics we are able to identify high-flow and low-flow CCFs. Etiologically we can classify CCF as spontaneous or traumatic[11].

According to their angio-architecture, CCFs can be divided into 4 groups (Fig. 9) (Table 1):

(1) Direct fistulas (Barrow type A)
(2) Indirect or dural fistulas (Barrow types B, C, and D).

The internal carotid artery (ICA) and the cavernous sinus are directly connected in direct fistulas. Typically, they are high-flow fistulas[12–27].

On the contrary, indirect or dural CCFs are low-flow fistulas that connect the cavernous sinus and cavernous artery branches. Specifically, meningeal branches of the ICA are involved in Barrow type B fistulas, ECA branches are in Barrow type C fistulas, and meningeal branches from both the ICA and ECA are involved in Barrow type D fistulas. Type D spontaneous dural CCFs are the most common[28].

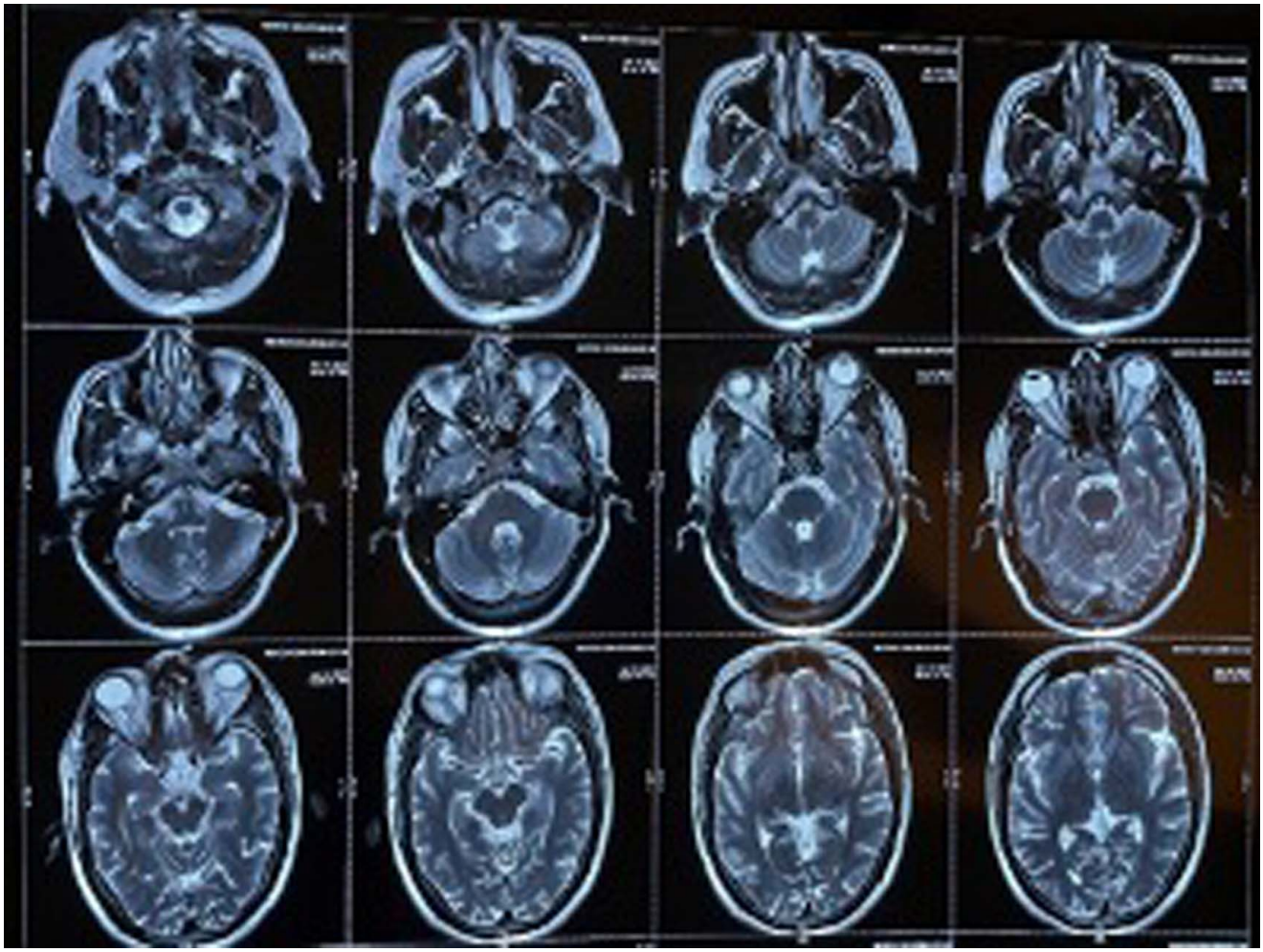

Figure 2. MRI showing thrombosis of cavernous sinus.

The inferior cavernous sinus artery is the ICA trunk that is most usually implicated; however, dural fistulas can also affect the meningo-hypophyseal trunk and its branches. The internal maxillary artery, middle and accessory meningeal arteries, ascending pharyngeal artery, anterior deep temporal artery, and posterior auricular artery are the branches of the external carotid artery that are most frequently affected[29].

Etiology

Traumatic CCFs are usually type A direct fistulas and frequently show a single direct contact between the ICA and the cavernous sinus. Nonetheless, the cavernous sinus wall typically contains several micro-fistulas and various dural feeders in spontaneous fistulas[30]. Because a type A shunt with high-flow characteristics might emerge after spontaneous rupture of an intracavernous ICA aneurysm, spontaneous CCFs may fit into any of the four angiographic classifications established by Barrow[31].

The most frequent cause of direct CCFs is traumatic disruption of the vessel wall. Direct CCFs may result from blunt and penetrating head trauma as well as iatrogenic harm[32,33].

Around 20% of type A CCFs are thought to be spontaneous and unrelated to any history of trauma[5,34]. They typically result from the rupture of an atherosclerotic artery or a cavernous segment aneurysm[5,36]. Ehlers-Danlos syndrome, fibromuscular dysplasia, and pseudoxanthoma elasticum are also risk factors linked to spontaneous type A, CCFs[5,37,38].

Pathophysiology and clinical features

The presence of important neuro-vascular structures into the cavernous sinus affects the symptomatology of CCFs. They comprise cranial nerves III (oculomotor nerve), IV (trochlear nerve), V1 (ophthalmic nerve), V2 (maxillary nerve), and VI (abducens nerve)[41].

Venous hypertension results from the direct transmission of extremely pressured arterial blood into the cavernous sinus and the draining veins via a CCF. The clinical manifestation of CCF is a direct result of increased intracavernous pressure and altered flow patterns. The size, as well as the exact position, duration, sufficiency, and route of venous drainage, along with the existence of arterial/venous collaterals, are all factors that affect the clinical characteristics of CCFs[33].

Exophthalmos, bruit, and conjunctival chemosis are the classic Dandy's triad symptoms that signify a direct, high-flow CCF. Although the full clinical trial is not always present, the majority of patients have proptosis (90%), chemosis (90%), diplopia (50%), cephalic bruit (25%), pain (25%), trigeminal nerve

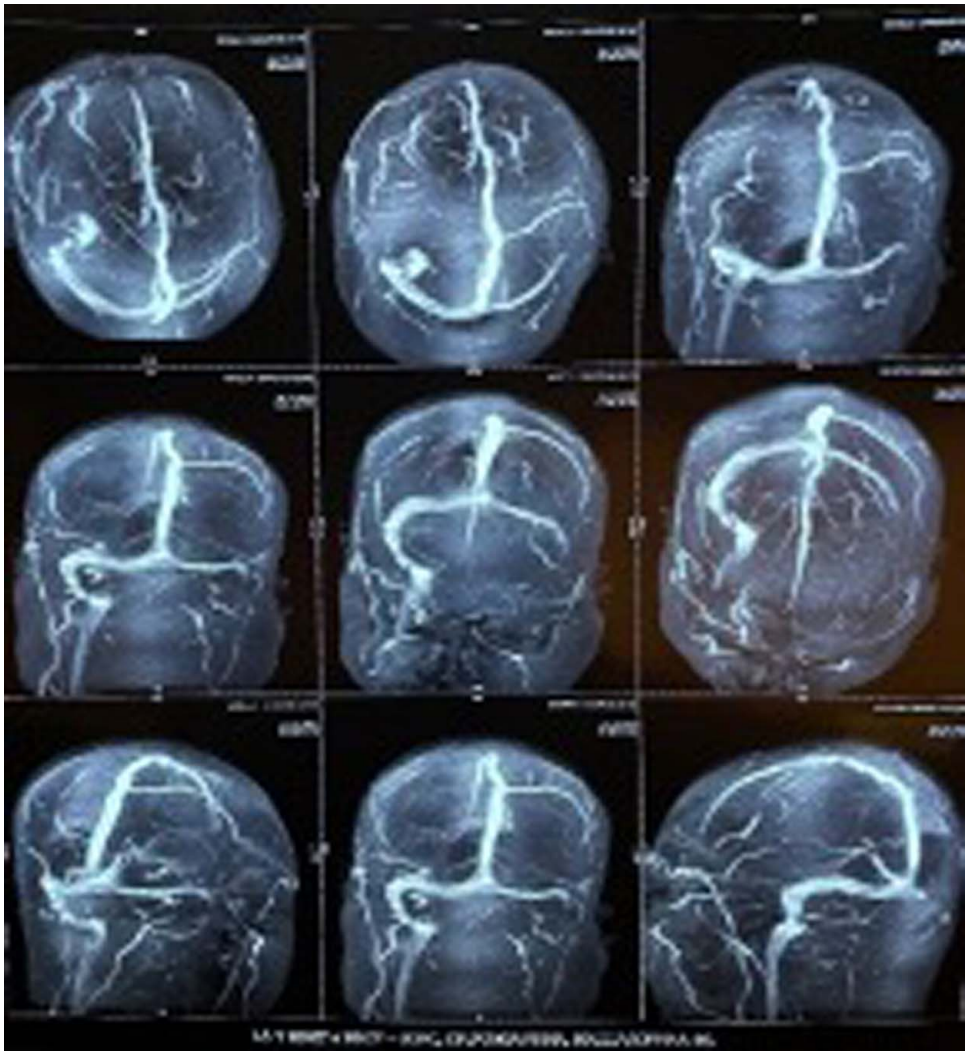

Figure 3. MR venography.

dysfunction, high intraocular pressure, and vision loss (up to 50%) upon presentation[35].

Elevated pressure in the veins that drain the orbit may result in secondary glaucoma, orbital venous congestion. Increased intraocular pressure and intravenous pressure can compromise retinal perfusion, which will drastically reduce visual acuity[33]. One of the most concerning CCF complications is visual loss, which requires urgent medical attention. Subconjunctival hemorrhages can be noticed as a result of dilated arterialized veins rupturing, and increased corneal exposure can also harm the cornea. In about 5% of patients, intracranial hemorrhage occurs; this is likely caused by altered venous drainage into the sphenoparietal sinus with obstruction of other drainage channels, which leads to cerebral cortical venous hypertension[5,35].

Indirect CCFs frequently lack the traditional triad of symptoms. Indirect CCFs do not present with symptoms as abruptly as do direct CCFs. Insidiously developing symptoms and indicators of indirect CCFs include proptosis, conjunctival injection, or progressive glaucoma in the majority of patients[5,29,35,39,40,42]. The hallmark of dural CCFs is exacerbation and remission of signs and symptoms, which may be caused by cavernous sinus thromboses and rerouting of venous flow in different directions[35].

Diagnosis

In the initial work-up of a potential CCF, noninvasive imaging techniques such computed tomography (CT), magnetic resonance (MR), CT angiography, MR angiography, and Doppler are frequently employed.

A CT scan of the orbit typically shows the afflicted eyeball proptosed, the extraocular muscles enlarged, the superior ophthalmic vein (SOV) dilated and tortuously twisted, and the ipsilateral cavernous sinus enlarged. With ocular edema and aberrant flow voids in the afflicted cavernous sinus, MR imaging results in CCFs are comparable to those seen on CT[5,32,35].

The gold standard for endovascular intervention planning, classification, and conclusive diagnosis of CCFs is cerebral angiography (Fig. 10). It does allow a detailed identification of the size and location of the fistula, the distinction between direct and indirect lesions, the presence of any associated cavernous carotid aneurysms, the presence of complete or partial stealing phenomena, as well as the assessment of the global cortical

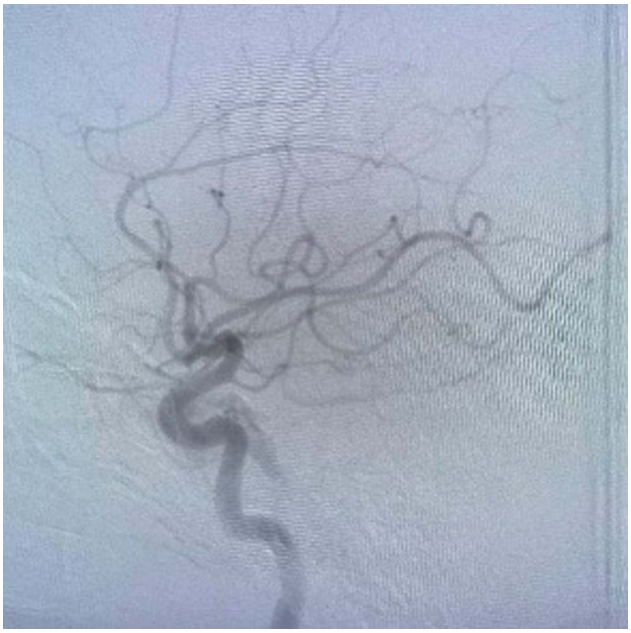

Figure 4. Digital subtraction angiography.

arterial circulation and collateral flow through the circle of Willis.

Color Doppler study can measure flow direction and velocity, identifying the arterial flow in the orbital veins in CCF instances[43]. The SOV's flow reversal suggests a CCF.

Treatment

Conservative management

Due to additional thrombosis of the affected cavernous sinus segment, spontaneous resolution of dural fistulas can occasionally take place within days to months following clinical presentation. As a result, it is standard practice to treat the patient's ocular symptoms medically. This includes prism therapy or patching for diplopia, topical beta-blockers and acetazolamide for elevated intraocular pressure, lubrication for proptosis-related keratosis, and/or systemic corticosteroids as necessary.

As a further noninvasive treatment for indirect CCFs, manual external carotid-jugular compression therapy may be started[5]. By reducing arterial intake while concurrently increasing outlet venous pressure and encouraging spontaneous thrombosis within the fistula, compression therapy seeks to temporarily reduce arteriovenous shunting[44].

Figure 5. Preoperative assessment.

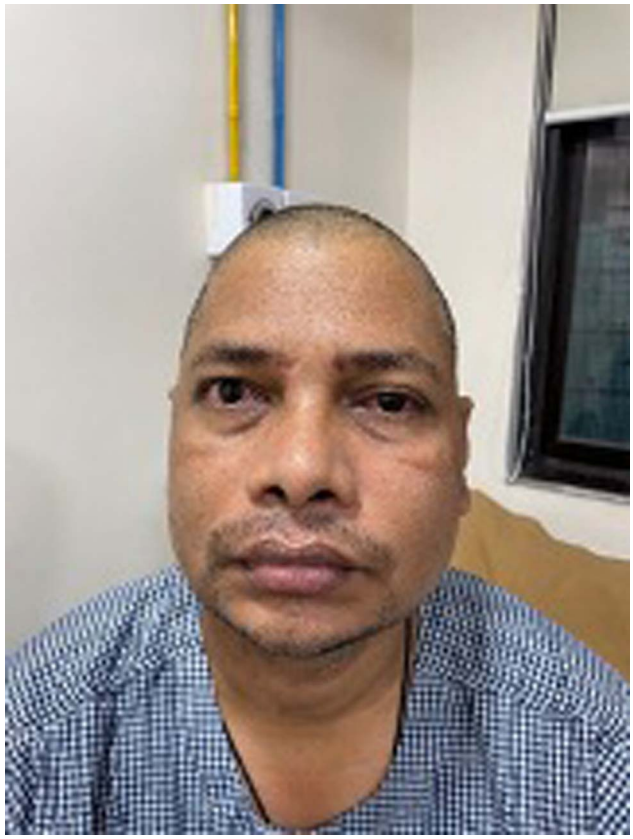

Figure 6. Postoperative result.

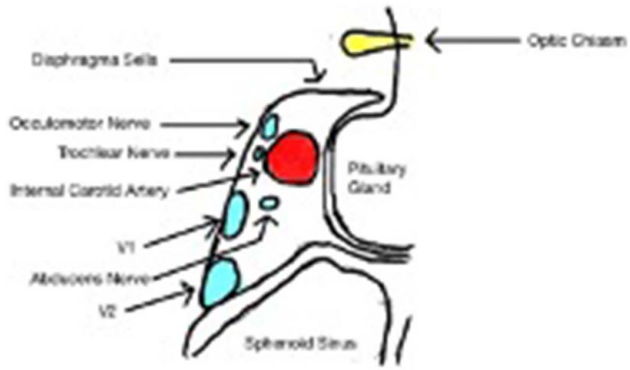

Figure 7. Cavernous sinus anatomy.

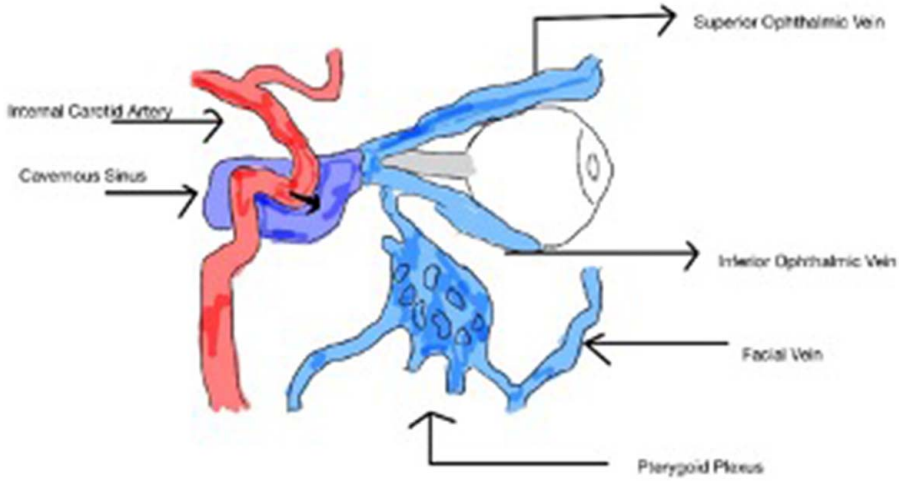

Figure 8. Superior ophthalmic vein, cavernous sinus, and inferior ophthalmic vein.

Surgical intervention

Surgery interventions can include stereotactic radiosurgery and endovascular therapy.

Stereotactic radiosurgery

For the treatment of CCFs, stereotactic radiosurgery has become a viable alternative therapy option and has been studied in numerous institutions. Gamma knife radiosurgery can be utilized as a stand-alone procedure or as a follow-up treatment after endovascular intervention[45,46] The 22 months average delay between treatment and full symptom relief is a significant downside, even if preliminary results indicate that radiosurgery is a safe and effective alternative treatment for indirect CCFs[42]. Moreover, the use of radiosurgery as a first-line treatment is hindered by the inability to manage crises and traumatic fistulae[35].

Endovascular approach

There are two routes for the endovascular approach.

Arterial route

Indirect low-flow CCF trans-arterial embolization is typically difficult due to the small size, intricate architecture, and numerous artery feeders. Also, the decision to use the trans-arterial method as the major therapy of spontaneous indirect CCFs is constrained by potential side effects (such as thromboembolic stroke, cranial nerve palsies, etc.). Thus, for high-flow indirect CCFs, trans-arterial embolization is normally only utilized to decrease arterial inflow prior to trans-venous occlusion and as a workable alternative when trans-venous attempts fail[5,35].

Indirect CCFs that are traumatic are managed differently than those that are spontaneous. Trans-arterial embolization may be

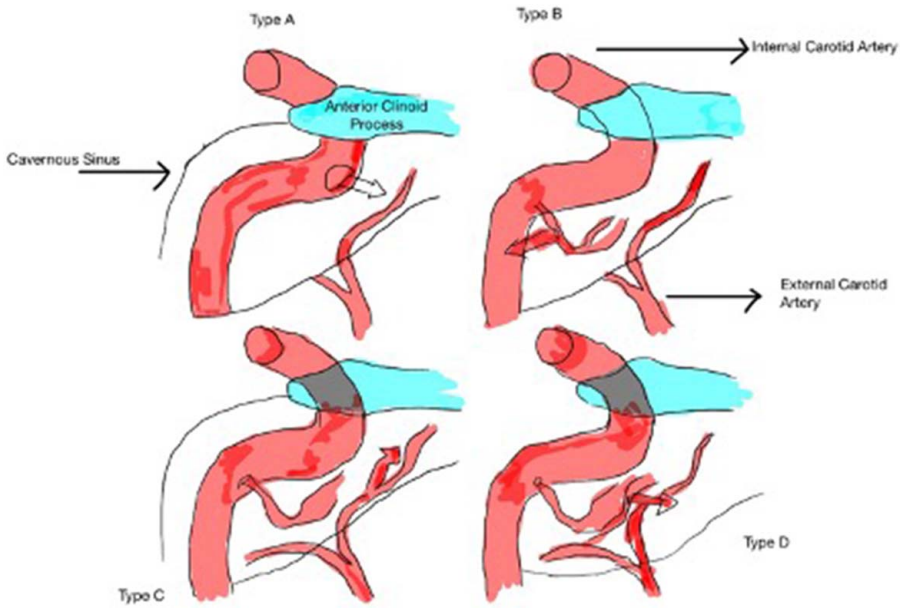

Figure 9. Classification of carotid-cavernous fistula.

Table 1

Barrow classifications for types of carotid-cavernous fistula.

Type	Direct vs. indirect	Description
A	Direct	Direct connection between ICA and cavernous sinus.
B	Direct	Connection between dural branches of ICA and cavernous sinus
C	Indirect	Connection between dural branches of ECA and cavernous sinus
D	Indirect	Connection between dural branches of both ICA and ECA and cavernous sinus

ECA, external carotid artery; ICA, internal carotid artery.

chosen for traumatic lesions because the single artery supply is large enough to provide microcatheter access to the feeder and cavernous sinus. The trans-venous technique is only used when the fistula fails or recurs and arterial access to the site of the fistula cannot be achieved[47].

The tiny meningeal branches supplying the fistula are distally catheterized using trans-arterial methods (Fig. 11). The microcatheter tip should be positioned as close to the point of fistulous communication as is practical when inserting the super selective microcatheter. In order to occlude the fistulous connections and penetrate the cavernous sinus, liquid embolic agents (n-BCA, EVOH) are injected under fluoroscopic supervision once a suitable microcatheter position has been attained. Coils and particle agents have been utilized, but by themselves, they are unable to permanently occlude the fistula[5].

Venous route

Via inferior petrosal sinus

Because the IPS is directly connected to the internal jugular vein (IJV) through the petro-occipital fissure, access to the CS through the ipsilateral IPS is typically favored. It is typically viable to maneuver a microcatheter into the anterior region of the CS to approach the junction between the SOV and CS after securely inserting it into the posterior part of the CS.

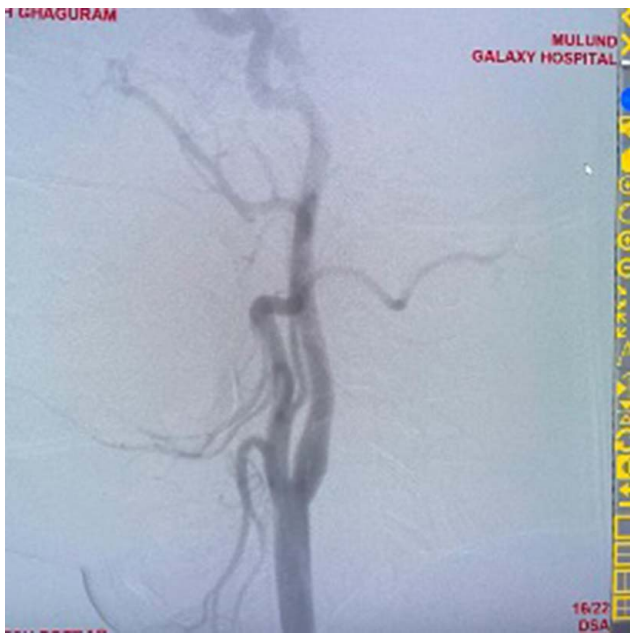

Figure 10. Carotid cavernous fistula on digital subtraction angiography.

Via superior ophthalmic vein

The attempt to approach by SOV can be taken into consideration if the ipsilateral or contralateral IPS approach fails. There are typically two ways to access the CS through the SOV: a direct percutaneous SOV approach and a trans-femoral SOV technique using the facial vein[48].

Other trans-venous approaches

Other accesses may also be taken into consideration if all of the aforementioned methods prove unsuccessful. The frontal vein, superficial temporal vein, and facial vein have all been directly cannulated, according to some publications[49]. Rare cases of direct cannulation of the superficial middle cerebral vein following endovascular and open craniotomy surgery have been documented[50].

Prognosis

Although ocular symptoms subside quickly after successful treatment, patients may experience a brief increase in symptoms as a result of thrombus spreading across the cavernous sinus and into the SOV. With trans-arterial embolization, gamma knife radiosurgery, or conservative treatment, patients may experience what is known as the "paradoxical worsening phenomena." Although troubling to the patient, such symptoms typically go away on their own with time. Inflammation linked to sinus thrombosis may be reduced with a brief course of corticosteroids[29,51].

Conclusion

A carotid-cavernous fistula is a rare entity and often misdiagnosed due to its overlapping symptoms with modalities such

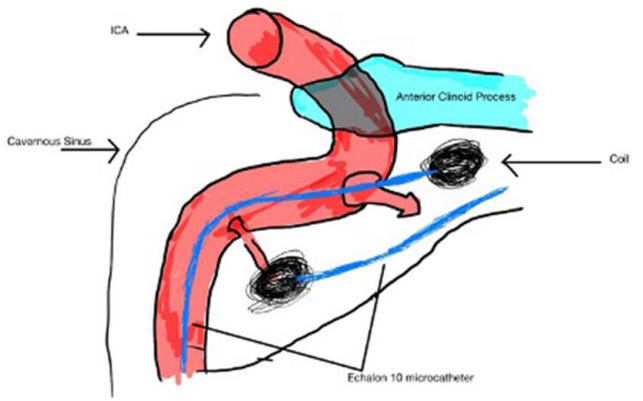

Figure 11. Trans arterial embolization of carotid-cavernous sinus fistula.

as cavernous sinus thrombosis. In order to diagnose and treat such CCF situations, a thorough approach is necessary. For the diagnosis and categorization of CCF, cerebral angiography continues to be the gold standard. The primary line of treatment is still trans-venous endovascular therapy. The trans-arterial technique is another excellent choice with the development of emboli agents. After receiving neuro-vascular treatment for CCF, there is a higher success rate and full symptom remission.

Ethical approval

Not applicable.

Consent

Consent was obtained by the patient in this study. The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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All authors have equal contribution.

Conflicts of interest disclosure

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