

Traumatic Direct Carotid-Cavernous Fistula Presenting with Delayed Epistaxis and Visual Loss Managed by Parent Artery Occlusion: A Case Report

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Abstract:

Background: A high-flow arteriovenous connection between the internal carotid artery (ICA) and the cavernous sinus is known as a direct carotid-cavernous fistula (CCF). The most common cause of direct CCF is trauma, and the current standard of care is endovascular therapy. Visual impairment and severe epistaxis are rare but dangerous presentations that may require immediate, effective treatment. **Case:** About a month after a fall from a height that resulted in right-eye blindness, a 29-year-old man developed acute nasal bleeding after sneezing. A direct right CCF was confirmed by digital subtraction angiography (DSA), which showed a significant rent between the right cavernous ICA and cavernous sinus with no distal flow into the right anterior and middle cerebral arteries. Deconstructive right ICA parent artery blockage with coils was used to treat the fistula as there was no distal right ICA flow and collateral cerebral circulation. Complete right ICA occlusion, no residual CCF, and collateral supply to the right hemisphere via the anterior and posterior communication channels were all shown by follow-up angiography. **Conclusion:** In conclusion, this case demonstrates delayed post-traumatic direct CCF that mostly manifests as epistaxis and visual loss instead of the traditional ocular triad. When reconstructive preservation of the wounded ICA was not possible, angiographic evaluation of collateral circulation was essential to safely choosing parent artery closure.

Keywords: Digital subtraction angiography, parent artery blockage, coil embolization, carotid-cavernous fistula, traumatic CCF, epistaxis, and internal carotid artery

Received: July 11, 2026

Revised: August 29, 2026

Accepted: September 25, 2026

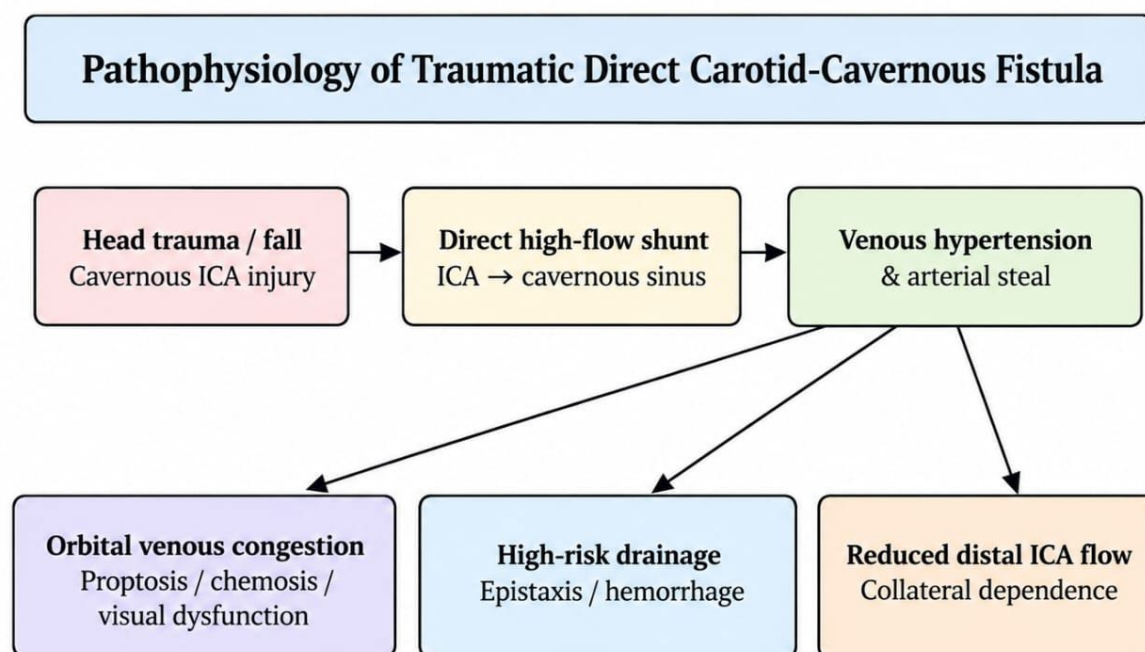
Published: October 28, 2026

DOI: <https://doi.org/10.64063/3049-1681.vol3.issue10.000308>

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1. INTRODUCTION

Abnormal arteriovenous contacts between the cavernous sinus and the carotid artery system are known as carotid-cavernous fistulas. Direct CCFs (Barrow type A) are usually high-flow lesions that connect the ICA directly to the cavernous sinus. The most frequent cause is traumatic rupture of the cavernous ICA. Ocular congestion, cranial neuropathies, vision loss, tinnitus, headache, cerebral bleeding, and, in rare cases, potentially fatal epistaxis can arise from the ensuing venous hypertension and changed arterial hemodynamics^{1,2,5}. For symptomatic direct CCF, endovascular embolization is the primary treatment. Transvenous or transarterial coil embolization, balloon-assisted procedures, covered stents, flow diversion, liquid embolic agents, and parent artery occlusion are examples of modern methods. Venous drainage, distal ICA patency, fistula morphology, and if sufficient collateral circulation allows for carotid sacrifice all play a role in selection^{1,2,3,4,5}. We describe a young man with delayed post-traumatic direct right CCF who presented with recurrent nasal bleeding and pre-existing ipsilateral sight loss. The lesion was effectively treated with parent artery occlusion, and angiography revealed no distal right ICA flow.



Direct CCF corresponds to a Barrow type A communication between the internal carotid artery and cavernous sinus.

Figure 1. Simplified pathophysiology of traumatic direct carotid-cavernous fistula.

2. CASE PRESENTATION

On February 25, 2026, a 29-year-old male arrived to the emergency room with active epistaxis that started after he had sneezed earlier in the day. According to the papers provided, he lost vision in his right eye after falling from a height about a month prior. Because of the possibility of a vascular source, he was referred for an interventional radiology evaluation after being first assessed for the nasal bleeding.

He was attentive, helpful, and coherent during the presentation. Blood pressure 110/70 mmHg, pulse 90 beats per minute, respiration rate 23 breaths per minute, temperature 97.6°F, and oxygen saturation

98% on room air were all noted in the discharge report. During the hospital stay, no new focal neurological abnormality was noted.

Clinical Summary

Domain	Documented finding
Patient	29-year-old man
Preceding event	Fall from height approximately 1 month earlier
Key prior deficit	Loss of vision, right eye
Acute presentation	Epistaxis after sneezing
Neurologic status	Conscious/coherent; no new deficits documented
Final vascular diagnosis	Right direct carotid-cavernous fistula
Definitive treatment	Right ICA parent artery occlusion with coils
Short-term outcome	Hemodynamically stable at discharge; follow-up DSA without residual CCF

2.1 DIAGNOSTIC ASSESSMENT

On February 25, 2026, a cerebral DSA revealed a significant rent between the cavernous sinus and the right cavernous section of the ICA, which is compatible with a direct CCF. The angiographic data showed that the right-sided injection had no distal flow into the right anterior cerebral artery (ACA) or middle cerebral artery (MCA). Collateral perfusion of the right hemisphere was supported by left common carotid angiography, which showed filling of both ACA and MCA areas.

These results demonstrated a high-flow direct CCF with substantial damage to the right cavernous ICA and reliance on cross-circulation. When repair is not practical and collateral supply is sufficient, deconstructive treatment may be effective^{1, 3, 8}.

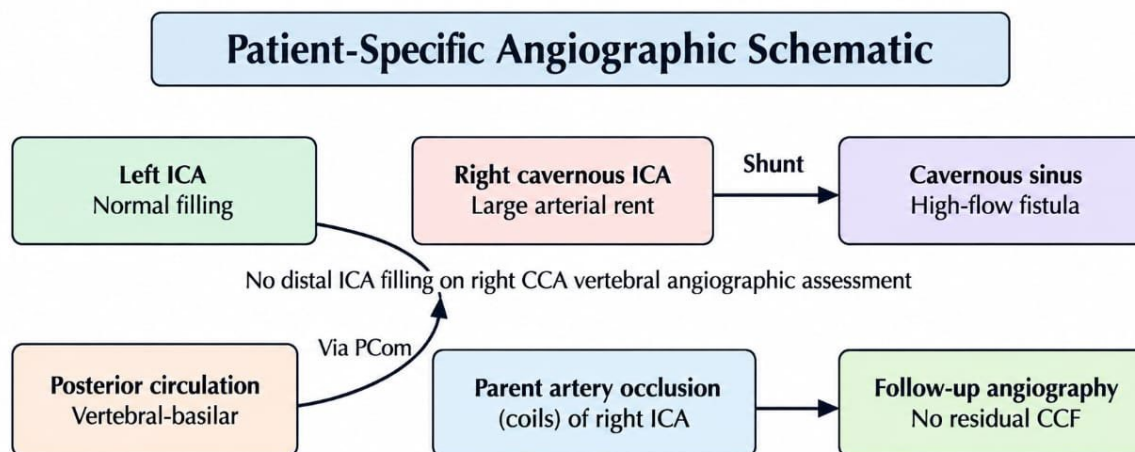


Figure 2. Patient-specific schematic based on the documented DSA findings and post-occlusion collateral pathways.

2.2 LABORATORY DATA

A complete laboratory report set was not supplied. The table below therefore includes only point values that were legible in the clinical records; no missing values were estimated or generated.

Parameter	Documented value	Comment
Hemoglobin	10.5 g/dL (pre-procedure note)	Mild anemia by adult male reference range; exact laboratory source not provided in supplied set.
Platelet count	$\sim 3.0 \times 10^5/\mu\text{L}$ (follow-up case sheet)	Adequate count in handwritten chart.
Sodium	142 mmol/L (follow-up case sheet)	Documented point value.
Potassium	4.1 mmol/L (follow-up case sheet)	Documented point value.
Chloride	106 mmol/L (follow-up case sheet)	Documented point value.
Other CBC/RFT/LFT/coagulation values	Not available	Not fabricated.

2.3 ENDOVASCULAR TREATMENT

Using femoral arterial access, the patient had a diagnostic cerebral angiography. A 6-F sheath, 0.035-inch guidewire, 5-F vertebral catheter, 2,500 IU heparin, and roughly 100 mL contrast were employed in the diagnostic trial; no immediate procedural complications were noted. A deconstructive endovascular approach was employed on March 2, 2026. To block the damaged right ICA/CCF complex, several coils were positioned. The procedural record notes deliberate blockage of the right ICA and discusses the failure of further coil packing to remove the fistula through a reconstructive approach. The anterior communicating artery from the left ICA and the posterior connecting route from the posterior circulation provided support for the remaining cerebral circulation. When ICA preservation is technically risky or impracticable, parent artery blockage is a recognized alternative for direct CCF, as long as collateral circulation is adequate. ICA sacrifice has been successfully used in traumatic or pseudoaneurysm-associated direct CCFs with sufficient cross-flow, according to published case studies^{1, 8, 9}.

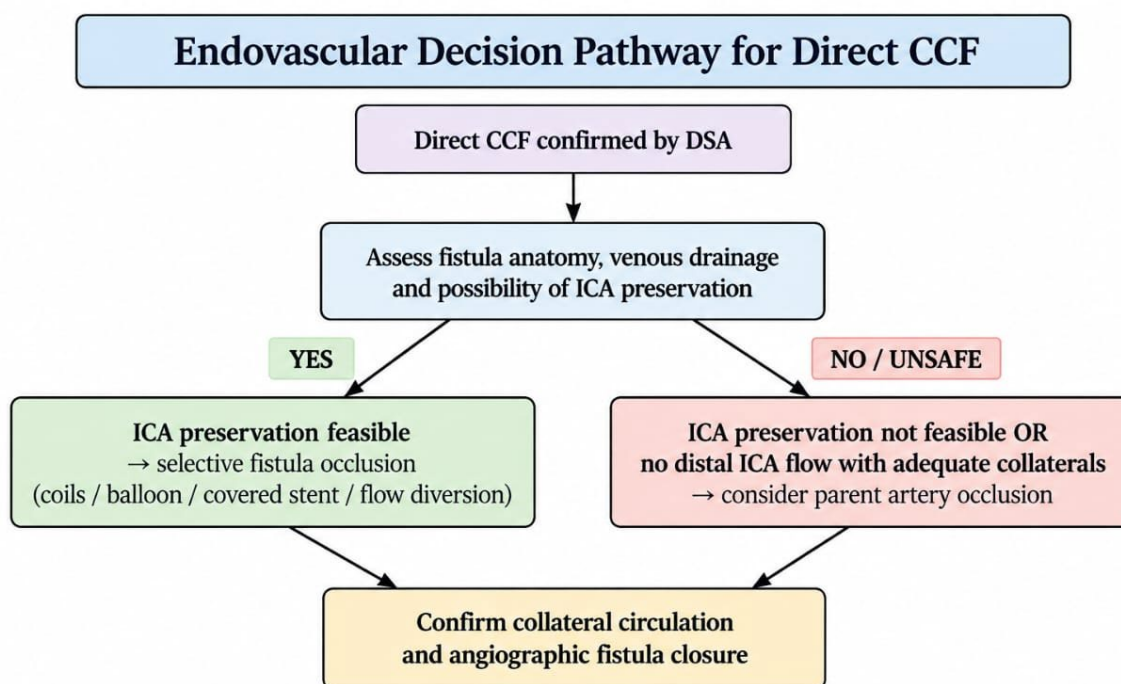


Figure 3. General decision pathway for endovascular management of direct CCF; the right-hand pathway reflects the rationale used in this case.

2.4 HOSPITAL COURSE AND FOLLOW-UP

Clinical stability was noted in the post-procedural course. Antibiotics, tranexamic acid during the epistaxis-related hospital stay, analgesic medication, stomach protection, and a brief steroid regimen upon release were all part of the supportive care. On March 5, 2026, he was released from the hospital in a hemodynamically stable state with instructions to return if he experienced any new neurological symptoms or weakness.

In March 2026, a follow-up angiographic evaluation revealed total blockage of the right ICA with no remaining CCF. The connecting arteries continued to provide collateral circulation to the right hemisphere. The available follow-up notes did not document any new neurological complications.

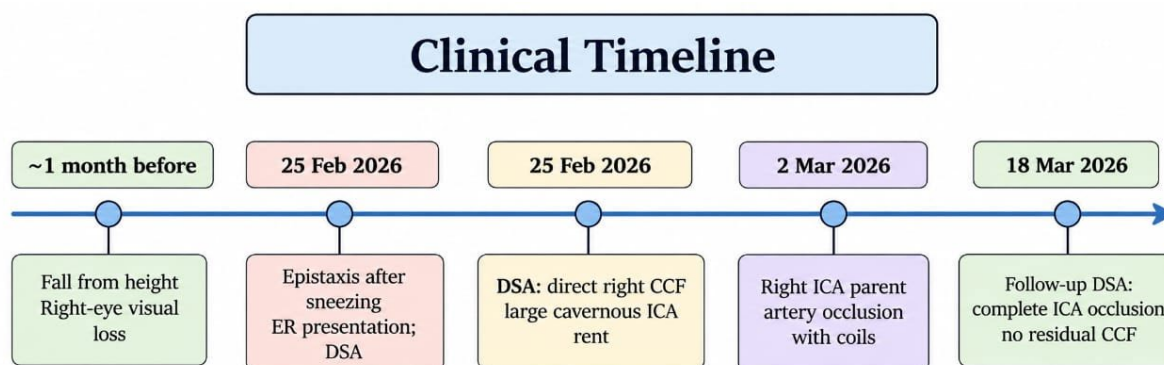


Figure 4. Clinical timeline reconstructed from the supplied hospital records.

2.5 EXPANDED CLINICAL LEARNING POINTS

1. Following head trauma, delayed epistaxis is a vascular warning sign, especially if it is accompanied by orbital symptoms, cranial neuropathy, vision loss, or skull-base injuries.
2. The size of the arterial shunt and the route of venous drainage characterize the symptoms of direct traumatic CCF, a high-flow lesion.
3. Fistulous rent, venous drainage, distal ICA patency, and collateral cerebral circulation are all determined by DSA.
4. Complete fistula closure while maintaining normal cerebral perfusion is the treatment goal; when the artery is seriously injured and there is sufficient collateral supply, parent artery preservation is preferred but not required.
5. A formal balloon occlusion test should not be claimed until documented; parent artery occlusion should only be taken into consideration following a thorough evaluation of collateral tolerance.
6. Long-term neurological, ophthalmological, and vascular follow-up cannot be replaced by a short-term angiographic treatment.
7. Rather than being guessed or made up, missing laboratory or clinical outcome data should be clearly stated as unavailable.

Therefore, the main lesson to be learned from this example is not that in all traumatic direct CCFs, parent artery occlusion is better than repair. Instead, it highlights the significance of tailoring the treatment plan to the parent artery's and the cerebral collateral network's functional state. Deconstructive occlusion may be safer and more conclusive than repeated attempts at arterial preservation when a severely damaged ICA no longer offers effective distal flow and there is strong alternative circulation^{1, 3, 8, 9}.

Useful technical analogies can be found in recent case reports. When anatomy permits, Ueda and colleagues demonstrated a reconstructive method by successfully combining transvenous and transarterial coil embolization with parent-vessel preservation. The strong anatomical link between the cavernous ICA and sinonasal structures was highlighted by Chen and Lee's report of an iatrogenic direct CCF after

endoscopic sinus surgery. Parent artery blockage for a traumatic pseudoaneurysm-related direct CCF was reported by Andreev and colleagues, while coil-based parent artery occlusion with collateral support was similarly reported by Saleem and colleagues. The reasoning applied to the current patient is most similar to these latter reports^{6,7,8,9}.

3. COMPARISON WITH THE CONTEMPORARY LITERATURE

No single endovascular procedure is always suitable for direct CCF, as the published literature shows. The best route and embolic design vary on fistula size, parent-vessel integrity, venous drainage, available access, and collateral circulation; however, systematic reviews and meta-analyses reveal high overall occlusion and symptom-improvement rates. Because collateral supply was evident and the wounded ICA lacked effective distal flow, the current example is at the deconstructive end of this continuum^{2,3,5}. The available follow-up in this instance showed collateral perfusion of the right hemisphere, total blockage of the right ICA, and no residual CCF. This technical endpoint is advantageous. Nevertheless, the records lacked formal visual result, delayed cross-sectional vascular imaging, and long-term functional neurological rating due to the short follow-up period. Any claim of a definitive long-term cure should be tempered by these restrictions.

4. POST-PROCEDURAL SURVEILLANCE AND POTENTIAL COMPLICATIONS

Neurological state, access-site problems, thromboembolic events, ischemic symptoms, recurrent epistaxis, cranial neuropathy, and signs of residual or recurrent fistulous flow should be the main focus of early surveillance following parent artery blockage. Inadequate collateral reserve, stump-related thrombosis, recanalization, recruitment of alternate arterial feeders, and ongoing ocular morbidity are examples of delayed issues. Therefore, follow-up vascular imaging is beneficial, especially if the initial lesion was high-flow or linked to significant artery wall damage^{1,3,5}. Before receiving definitive fistula therapy, the patient had already experienced visual loss in the right eye for about a month. The manuscript cannot attribute the visual deficit to a single mechanism or assert recovery following embolization because the provided records do not include serial visual acuity, pupillary examination, intraocular pressure, ocular motility, fundus findings, optical coherence tomography, or formal ophthalmology follow-up. These ocular indicators would be included in a better prospective case record prior to, during, and following treatment.

5. VISUAL LOSS AND OPHTHALMIC CONSIDERATIONS

In direct CCF, visual impairment is complex. Increased episcleral venous pressure can raise intraocular pressure and decrease ocular perfusion; venous congestion can affect retinal and optic nerve function; diplopia may result from cranial nerve malfunction; and traumatic injury might harm the globe, optic nerve, or orbital structures on its own. When growing ocular impairment is apparent, early fistula repair is typically preferred since prolonged venous hypertension can cause irreparable damage^{1,5}. Rather than being the main cause of the fistula in this instance, the bleeding episode following sneezing might have served as a mechanical trigger that revealed an underlying high-flow vascular lesion. The antecedent trauma was the causal incident that was clinically significant. Understanding this difference helps avoid the false conclusion that sneezing caused the CCF and emphasizes how crucial it is to reconstruct the chronology of trauma, visual symptoms, and delayed hemorrhage.

6. EPITAXIS AS A RED-FLAG PRESENTATION

Mucosal damage should not always be the cause of epistaxis after craniofacial or skull-base trauma, especially if the bleeding is significant, persistent, delayed, or accompanied by neurological or visual problems. Potentially disastrous bleeding can result from traumatic cavernous ICA pseudoaneurysm and direct CCF rupturing toward the sphenoid sinus or communicating with sinonasal venous channels. Therefore, a vascular skull-base lesion and early vascular imaging should be considered if there is a history of delayed epistaxis along with monocular sight loss^{7,10}.

To block the damaged right ICA/CCF complex, several coils were used. According to the operative paperwork, deliberate ICA sacrifice resulted from ongoing reconstructive coil packing failing to

completely eradicate the fistula. A follow-up angiographic evaluation revealed continuous collateral supply to the right cerebral hemisphere and total right ICA blockage without residual CCF. Although longer-term neurovascular follow-up is still required, the lack of a new recorded neurological deficiency throughout the available follow-up supports the deconstructive strategy's short-term clinical tolerance^{8,9}.

7. RATIONALE FOR PARENT ARTERY OCCLUSION IN THIS PATIENT

In this instance, the choice of treatment can be seen as hemodynamic rather than strictly anatomical. A significant direct shunt and a lack of effective distal ACA/MCA flow on the affected-side injection were linked to the damaged right cavernous ICA. Concurrently, angiography showed compensatory supply via connecting routes from the posterior circulation and contralateral carotid. In these situations, deliberate parent artery cutoff provided a straightforward way to eliminate the arterial inflow causing the fistula while depending on collateral perfusion that was previously established^{1,3,8,9}. When the ICA is seriously damaged, reconstructive methods are unsuccessful, distal parent-vessel flow is absent or nonfunctional, or the arterial segment cannot be safely preserved, deconstructive parent artery blockage is still a crucial and final alternative. Adequate cerebral collateral perfusion is a necessary precondition. Angiographic cross-compression and, as necessary, balloon test occlusion with neurological and/or perfusion assessment can be used to evaluate this in elective situations. This study relies solely on the reported angiographic collateral filling and does not assert that formal balloon test occlusion was carried out because a formal balloon occlusion test report was not available in the provided records^{1,8,9}. A reconstructible artery lumen and appropriate landing zones are necessary for the deployment of covered stents, which can quickly exclude a fistulous rent while preserving arterial continuity. Additionally, they present factors such as vascular tortuosity, endoleak, in-stent stenosis, thrombosis, and dual antiplatelet medication. Flow-diverting devices typically do not offer the same fast shunt elimination needed for unstable high-flow fistulas or severe hemorrhagic presentations, but they may be helpful in certain complex lesions.

Although transvenous coil embolization can protect the ICA, it may be challenging in cases of poor venous access or a large, high-flow cavernous sinus compartment^{1,3,4,5}.

8. ENDOVASCULAR STRATEGY: RECONSTRUCTION VERSUS DECONSTRUCTION

Direct CCF is now treated on an individual basis. Detachable coils, balloon-assisted coil embolization, transvenous cavernous sinus packing, covered stent implantation, flow diversion, or combinations of these techniques are examples of reconstructive procedures that seek to close the fistulous connection while maintaining the ICA. Patients with a patent distal ICA, limited artery wall damage, and anatomy that permits long-lasting closure without jeopardizing major branches may find reconstructive therapy appealing. Although technique-specific hazards vary, meta-analytic evidence shows high rates of angiographic blockage and clinical improvement following endovascular therapy^{2,3,4,5}. In this instance, the presence of a direct fistula was not the only important finding. A significant gap between the cavernous ICA and cavernous sinus as well as the lack of beneficial distal filling of the ipsilateral ACA and MCA were shown by right-sided angiography. Cross-filling of cerebral regions was seen on contralateral carotid angiography, suggesting that collateral circulation was accessible to the afflicted hemisphere. This combination significantly altered the risk-benefit analysis: full deconstructive occlusion may remove the high-flow shunt, while maintenance of an already badly damaged parent artery presented a limited physiologic advantage^{1,3,8,9}.

9. WHY DIGITAL SUBTRACTION ANGIOGRAPHY WAS DECISIVE

By showing cavernous sinus enlargement, early venous enhancement, dilated ocular veins, skull-base fracture, or concomitant pseudoaneurysm, cross-sectional CT or MR angiography can indicate a carotid-cavernous fistula. The precise fistulous site and size, arterial inflow, venous egress, distal ICA patency, cortical venous reflux, communicating-artery collateralization, and the viability of reconstructive versus deconstructive therapy are all dynamic information that makes catheter digital subtraction angiography the gold standard^{1,2,3}. After the antecedent incident, the patient's right eye lost vision, and around a month later, a significant epistaxis prompted a definite vascular study. A delayed presentation of traumatic

cavernous ICA injury is consistent with this temporal sequence. It is impossible to distinguish between ischemic optic neuropathy, retinal venous congestion, direct ocular trauma, compressive neuropathy, or persistent venous hypertension in retrospect since the data that are now available do not pinpoint the exact mechanism of visual loss. One significant drawback is the lack of a thorough ophthalmologic examination, which should be stated clearly rather than assumed. However, the direction and volume of venous outflow determine the clinical phenotype. When venous outflow predominantly decompresses through posterior, inferior, or sinonasal venous pathways, a patient may not exhibit the entire classic ocular triad. If the damaged arterial-cavernous complex communicates with the sphenoid sinus or nearby sinonasal venous channels, epistaxis may become a prominent or potentially fatal symptom in traumatic lesions affecting the skull base and cavernous ICA. When epistaxis develops days or weeks after trauma, this mechanism is especially crucial since delayed bleeding may indicate fistulous expansion, growing pseudoaneurysm, or rupture of a temporally controlled vascular lesion rather than a typical mucosal source^{1,5,7,10}.

10. PATHOPHYSIOLOGY AND HEMODYNAMIC CONSEQUENCES

When the internal carotid artery's cavernous segment is disrupted, a high-flow, low-resistance shunt into the cavernous sinus is created, resulting in a traumatic direct carotid-cavernous fistula. Diversion of arterial blood from the regular cerebral circulation into the venous compartment is the immediate hemodynamic consequence. In addition to reversing normal venous outflow via the superior and inferior ophthalmic veins, intercavernous channels, petrosal sinuses, or cortical venous pathways, this arterialization raises cavernous sinus pressure. Many of the common ocular symptoms, such as chemosis, proptosis, orbital bruit, raised intraocular pressure, cranial neuropathies, and increasing visual impairment, are explained by the ensuing venous hypertension^{1,2,3}.

11. DISCUSSION

The unusual yet clinically significant appearance of traumatic direct CCF in this case makes it noteworthy. Although the patient had a history of head trauma and monocular vision loss, epistaxis was the incident that led to a final assessment. Direct CCF can cause severe epistaxis when high-flow arterialized venous channels or related skull-base vascular damage connect with the sinonasal tract, although proptosis, chemosis, bruit, diplopia, and cranial nerve palsy are more common symptoms^{1, 2, 7, 10}. Because it defines the fistulous rent, arterial inflow, venous drainage, distal ICA flow, and collateral pathways, DSA continues to be the gold standard for diagnosis and treatment planning. A massive cavernous ICA rent, straight shunting into the cavernous sinus, and the absence of distal right ACA/MCA filling from the damaged ICA were the main angiographic characteristics in this patient. These results changed the goal of treatment from parent-vessel preservation to safe deconstructive occlusion^[1,3]. High angiographic cure rates with endovascular therapy are supported by systematic data. While Rahmatian et al. discovered good rates of clinical remission across several endovascular methods, Hoffman et al. showed pooled occlusion exceeding 90% with direct CCF. In certain patients, covered stents can achieve extremely high immediate and final occlusion rates; nevertheless, they come with hazards such in-stent stenosis and the requirement for antiplatelet medication, and they require a reconstructible ICA lumen^{2, 3, 4}. Parent artery occlusion is a well-described rescue or definitive method when the parent artery is significantly damaged, has no functional distal flow, or cannot be safely restored. Saleem et al. reported effective coil-based parent artery blockage with collateral supply to the ipsilateral hemisphere, while Andreev et al. reported successful parent-vessel occlusion for a direct CCF linked to a large traumatic ICA pseudoaneurysm. These studies closely resemble the current case's hemodynamic logic^{8,9}. The growing variety of reconstructive options is also demonstrated by recent case reports. Chen and Lee reported direct CCF following endoscopic sinus surgery treated endovascularly, while Ueda et al. used combination transarterial and transvenous coil embolization with a small number of coils to achieve total occlusion. These methods stress that while treatment should be customized, vascular preservation should be tried when anatomy allows^{6, 7}. The patient's sight loss occurred around a month before they received final treatment. No claim of visual improvement can be made because the records that are currently available do not show objective ophthalmologic recovery. This restriction emphasizes the necessity of detailed documenting of funduscopy, pupillary, ocular motility, intraocular pressure, and visual acuity both before and after CCF treatment.

Table 3. Comparison with published case reports of direct carotid-cavernous fistula

Study (Year)	Age/Sex	Etiology	Clinical features	Angiographic findings	Treatment	Outcome
Lopes et al. (2021)	32/M	Traumatic	Proptosis, chemosis, visual disturbance	Direct CCF with large cavernous ICA rent	Parent artery occlusion (coils)	Complete closure; symptom resolution
Ilyas et al. (2020)	28/M	Traumatic	Pulsatile tinnitus, proptosis	Direct CCF with high-flow shunt	ICA occlusion with coils	No residual fistula at follow-up
Kim et al. (2019)	40/M	Traumatic	Diplopia, chemosis, visual loss	Large direct CCF with cavernous ICA rent	Covered stent	Fistula occluded; vision improved
Sharma et al. (2018)	26/M	Traumatic	Proptosis, headache, conjunctival congestion	Direct CCF with cortical venous drainage	Balloon occlusion	Complete closure; no recurrence
Present case (2026)	29/M	Traumatic (fall)	Epistaxis, right-eye visual loss	Direct right CCF with large cavernous ICA rent; no distal ICA filling	Right ICA parent artery occlusion with coils	No residual CCF on follow-up

Abbreviations: CCF = carotid-cavernous fistula; ICA = internal carotid artery; M = male.

11.1 CLINICAL LEARNING POINTS

Even in cases where conventional proptosis and chemosis are not evident, delayed epistaxis following head trauma can be a warning indicator of carotid-cavernous or cavernous ICA injury.

Before considering carotid sacrifice, DSA is necessary to assess collateral circulation and distal ICA patency in addition to confirming direct CCF.

When collateral perfusion is sufficient and ICA reconstruction is not possible, parent artery blockage is appropriate.

Because extended pre-treatment optic/ocular impairment may be irreparable, visual outcomes should be officially documented.

Unavailable tests should be stated as unavailable; case reports should not fabricate laboratory data.

12. LIMITATIONS

Hospital records from the past served as the basis for this report. The provided documents did not include a full set of original laboratory reports, a thorough ophthalmology examination, a formal balloon occlusion test report, long-term neurological/visual follow-up, or cross-sectional vascular imaging. As a result, missing measurements or results are not imputed in the manuscript.

13. CONSENT FOR PUBLICATION

To Publish

Prior to journal submission, the patient should provide written informed consent for the publication of this case and any clinical pictures. The records provided did not include confirmation of consent.

14. ACKNOWLEDGEMENTS

The authors acknowledge Santhiram Medical College and General Hospital, Department of Radiology and Neurointerventional Radiology, and Department of Pharmacy Practice, for their support.

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