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Case Report

Determining the best endovascular approach: a case report of transvenous coiling for direct carotid cavernous fistula

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ABSTRACT

Direct carotid-cavernous fistula (CCF) is an abnormal high-flow arteriovenous connection between the cavernous segment of the internal carotid artery (ICA) and the cavernous sinus (CS), most commonly resulting from trauma. The high-flow nature of the fistula leads to arterialized venous hypertension within the CS and retrograde cortical venous reflux, which clinically manifests as proptosis, chemosis, and ocular bruit. Transarterial embolization (TAE) is often the preferred treatment for direct CCF because it typically involves a single, direct arterial feeder. However, it carries the risk of distal embolization into the arterial or venous system. This case reported a traumatic direct CCF successfully managed via transvenous embolization (TVE). A 48-year old male presented with typical symptoms of CCF following trauma. Its angioarchitecture included a single feeding artery from the left C4 segment of the ICA, a fistulous point in the left posterior CS, and venous drainage via the left inferior petrosal sinus (IPS) with venous engorgement of the left superior ophthalmic vein (SOV). The transfemoral TVE approach via the IPS was selected because it provides the shortest and most direct route to the posterior CS. Embolization was performed via three detachable coils deployed at the fistula site, resulting in complete obliteration of the fistula. While TVE avoids the potential complications associated with distal arterial embolization, it does carry a risk of venous congestion and, in rare cases, technical rupture of pial veins. Nonetheless, in the treatment of direct CCF, TVE has demonstrated efficacy comparable to that of TAE in achieving significant flow reduction and, in many cases, complete obliteration of the fistula.

Keywords: Direct carotid cavernous fistula, Direct cavernous dural arteriovenous fistula, Inferior petrosal sinus, Transvenous approach, Transvenous coiling

INTRODUCTION

Dural arteriovenous fistula (DAVF) is an uncommon cerebrovascular disease characterized by an active abnormal direct arteriovenous connection within the dural leaflet without intervening in the capillaries. Carotid cavernous fistula (CCF) represents the second most common subtype of DAVF and involves feeding arteries from either or both the ICA or the external carotid artery (ECA) or their respective branches and draining veins toward the CS.[1,2] This may lead to significant morbidity, including proptosis and diplopia, until permanent vision loss occurs. High-flow CCFs may also increase the risk for seizures and intracerebral hemorrhage.[1,3]

Challenges in managing this disease arise from the choice of the best treatment for CCF. The treatment choice for CCF was suggested on the basis of its hemodynamic-angioarchitecture profile. Low-flow CCFs may spontaneously resolve with conservative management, except if retrograde flow or cortical venous reflux occurs. On the other hand, high-flow CCFs require more fistula closure.[4,5] Multiple treatment modalities, including endovascular TAE, endovascular TVE, surgical approaches, stereotactic radiosurgery, or combined approaches, have been proposed, but there are still no evidence-based guidelines regarding the best approach and when to initiate these procedures.[4,6]

The approach using TAE is commonly utilized in the treatment of direct high-flow CCFs, as these lesions typically involve a single arterial feeder resulting from a tear in the ICA communicating with the CS. However, several considerations may reduce the preference for TAE in certain cases. These include the potential risk of embolic material migrating into distal arterial or venous structures, which can result in unintended ischemic or hemorrhagic complications.[2,7]

The use of TVE via various venous access routes has been widely used for the treatment of indirect low-flow CCF with consistently favorable outcomes, with TVE via the infeIPS being the most favorable approach.[1,2,4,6,8,9] This report presents case in which direct CCF was successfully managed with TVE via multiple detachable coils. In cases where TAE is technically challenging or contraindicated, TVE may serve as an effective alternative for direct CCF, offering comparable efficacy in achieving flow reduction and in some cases, complete fistula obliteration.

CASE REPORT

A 48-year-old male was admitted due to a progressively worsening headache for one month. She previously experienced a traffic accident three months prior. Following the event, the patient developed left eye swelling, recurrent episodes of left-sided pulsating headache, and left-sided tinnitus. There was also mild memory impairment that did not interfere with her activities of daily living. Physical examination revealed proptosis, chemosis, ptosis, tenderness, and conjunctival hyperemia (Figure 1) in the patient's left eye, with a visual acuity of 2/60, binocular diplopia, and left extraocular muscle restriction. Consciousness was normal, and the other physical and neurological findings were uneventful.

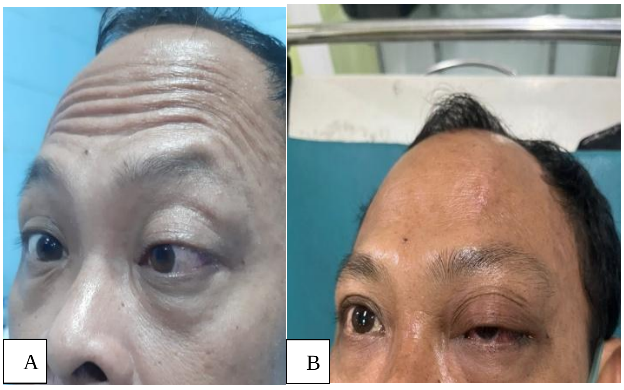

Figure 1 (A and B): Clinical presentation of the patient.

A brain CT scan revealed significant left eye proptosis with an enlarged left SOV (Figure 2). Digital subtraction angiography (DSA) confirmed the presence of a type A CCF with a feeding artery directly from the left cavernous (C4) segment of the ICA and draining vein to the CS with left SOV vein engorgement.

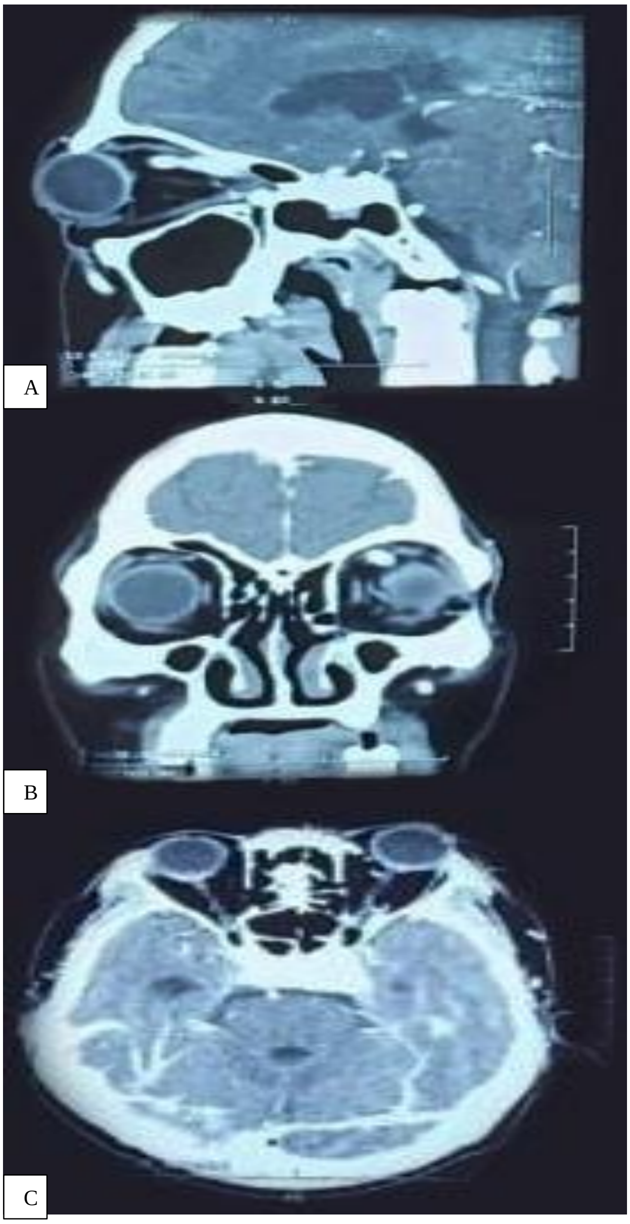

Figure 2 (A-C): Brain CT scan of the patient.

Owing to the favorable visualization of the left IPS, TVE at the IPS was selected. Transarterial and transvenous access were obtained via the right femoral artery and vein, respectively, via 6F femoral sheaths. An ENVOY 6Fr (Codman Neuro, Raynham, MA) guiding catheter was placed at the left cervical ICA for angiography and roadmaps, whereas another similar guiding catheter was positioned at the left internal jugular vein (IJV) for embolization. An excelsior microcatheter (Stryker Neurovascular, Fremont, CA) was then navigated from the left IJV through the left IPS to reach the posterior CS. Three target XL 360 detachable coils (Stryker Neurovascular) with dimensions of 20 mm×50 cm, 7 mm×30 cm, and 5 mm×20 cm were then deployed as proximal to the fistule as possible with caution to preserve the left SOV. Post-TVE DSA revealed complete CCF occlusion and reduced flow to the left SOV.

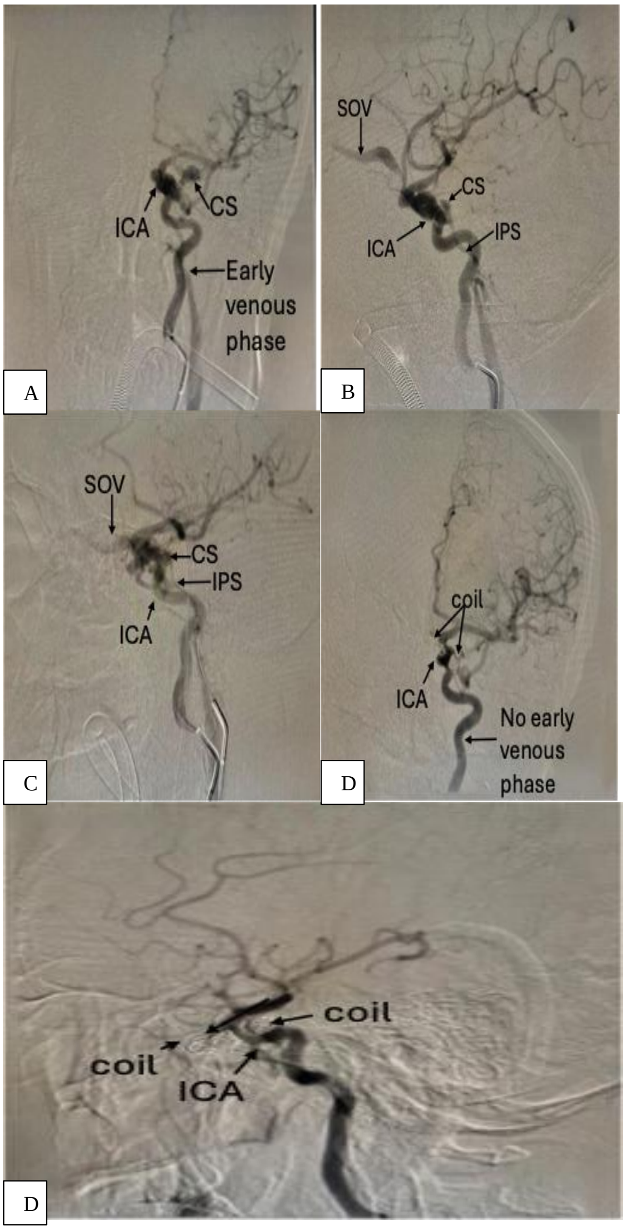

Figure 3 (A-E): Digital subtraction angiography of the left ICA. The pretreatment phase of the anterioposterior (A) and lateral (B) views revealed an abnormal connection between the left cavernous segment of the ICA and the CS during the arterial phase. The IPS and engorged SOV are also visible (B). During the treatment phase (C), double transfemoral access was deployed to the carotid artery and IPS. Transarterial access was used as a roadmap, and transvenous access was used via internal jugular vein and IPS to deploy several coils. Posttreatment anteroposterior (D) and lateral (E) angiographic views demonstrated successful coil deployment at the fistula site, with resolution of SOV engorgement.

No procedural complications were detected. The headache, proptosis, chemosis, hyperemia, and ptosis completely resolved within two to three weeks. Double vision and tinnitus persisted but did not progress.

DISCUSSION

Direct CCF is a high-flow vascular anomaly that necessitates prompt intervention because of its low likelihood of spontaneous closure. It carries significant risks, including hemodynamic instability, permanent visual impairment, cranial neuropathies, elevated intraocular and intracranial pressures, and the potential for intracerebral hemorrhage. Although numerous studies have demonstrated substantial symptomatic improvement with endovascular treatment (EVT), no standardized guidelines have been established for the optimal management of direct CCF. The present study provides further evidence supporting the efficacy of TVE in achieving meaningful clinical improvement in patients with direct CCF. Moreover, TVE was demonstrated to be a safe procedure with no documented intraprocedural or postprocedural complications, as provided in this case.

The patient presented a male with monocular symptoms in the left eye, including visual impairment, extraocular muscle movement restriction, and the involvement of extraocular structures, such as chemosis and proptosis. These clinical findings suggested a retrobulbar pathology on the ipsilateral side with an outward mass effect. The rapid progression of symptoms following a recent traumatic event strongly pointed toward a diagnosis of direct high-flow CCF rather than alternative etiologies, including retrobulbar space-occupying lesions. Unilateral ocular involvement also has systemic causes, such as hyperthyroidism, which typically presents with bilateral ocular involvement. The classic clinical trial of CCFs consists of ocular bruit, pulsatile exophthalmos, and conjunctival chemosis.[5] Ocular complaints, especially diplopia, exophthalmos, conjunctival chemosis, and nonmigraine headache, are typically more common in CCF than in other types of DAVF.[10]

The diagnosis of a direct high-flow CCF in this case was confirmed by brain imaging, which revealed SOV engorgement and proptosis, in conjunction with DSA, demonstrating a direct fistulous connection between the C4 segment of the ICA and the CS. Compared with DSA, advanced neuroimaging modalities, including contrast-enhanced computed tomography angiography (CTA) and magnetic resonance angiography (MRA), offer sensitivities of approximately 87% and 80%, respectively, and remain the gold standard for detailed evaluation of the angioarchitecture of CCFs.[7]

Retrograde arterialized flow into the cerebral draining veins, known as cortical venous reflux, plays a central role in the symptomatology of CCF and is a key determinant in the decision for treatment.[2] CCFs with anterograde venous outflow may be discovered incidentally because of their relatively benign course. On the other hand, those with retrograde venous drainage often present with a range of clinical symptoms, including (1) subtle shunting with cortical venous drainage and resulting intracranial venous hypertension, which may manifest with cognitive impairment or parkinsonian features, and (2) high-flow shunting leading to ocular venous hypertension, with clinical signs such as chemosis, proptosis, cranial nerve palsy, and, in more severe cases, intracranial hemorrhage, focal neurological deficits, cortical vein or sinus thrombosis, or seizures.[1,3]

The decision to initiate treatment for a CCF is guided primarily by the clinical presentation, particularly the progression of symptoms, vision loss, and/or the presence of cortical venous reflux.[4] Therefore, the detailed angioarchitecture and hemodynamics of CCFs should also be assessed. Barrow et al classified CCFs into direct high-flow and indirect low-flow types on the basis of their arterial supply and hemodynamic characteristics. Type A refers to direct, high-flow CCFs resulting from direct communication between the C4 segment of the ICA and the CS. Indirect low-flow CCFs are further subdivided into type B, arising from the meningeal branches of the ICA; type C, arising from the meningeal branches of the ECA; and type D, arising from a combination of the meningeal branches of both the ICA and the ECA.[5] Direct CCF is most commonly associated with trauma, is typically symptomatic, and rarely spontaneously disappears because of its high-flow properties.[4,5] In contrast, indirect low-flow CCFs usually develop spontaneously, present with milder and more insidious symptoms, and may resolve spontaneously through thrombosis.[1,2,5] In the present case, the patient exhibited rapid progression of the classic triad of CCF symptoms consistent with a direct high-flow CCF requiring urgent intervention. The potential for irreversible functional loss, including vision impairment, restricted ocular motility, and the risk of intraocular or intracerebral hemorrhage, further underscores the need for prompt treatment.

The recognition of the CCF angioarchitecture is paramount in determining the best approach for dealing with CCF. The CS is an extradural venous channel at the bilateral parasellar compartment of the body of the sphenoid bone. It is encased by a periosteal and meningeal dura mater on all sides of its four walls, except for a single meningeal layer on its medial side. It is surrounded by the medial temporal pole and anterior clinoid process laterally, the diaphragma sella medially, and the clivus posteriorly. The important structures inside the CS include the oculomotor, trochlear, ophthalmic, and maxillary nerves on the lateral side as well as the abducens nerve and the C4 segment of the ICA on the inferolateral side. Venous inflow is from the anterolateral side, including the SOV and inferior orbital vein (IOV) anteriorly as well as the sphenoparietal (sphenobasal) sinus and superficial middle cerebral vein (SMCV) laterally. Venous outflow occurs posteroinferiorly, including posteriorly via the superior petrosal sinus (SPS), IPS, and basilar (clival) plexus to the posterior fossa, as well as inferiorly via emissary veins to the pterygoid plexus and paraspinal venous plexus. The connections between the bilateral CSs are through the intracavernous (circular) sinus.[2,4]

In CCF, the feeding arteries to the CS may arise directly from the C4 segment of the ICA or from the dural branches of either or both of the ICA, ECA or their branches, which is recognized as “dangerous anastomosis.” Significant arterial feeders include the middle meningeal artery (MMA), accessory meningeal artery (AMA), and ascending pharyngeal artery (APA), all of which originate from the ECA and may provide bilateral supply to the CS in up to 70% of cases. The dural branches of the C4 segment of the ICA, including the meningohypophyseal trunk (MHT) and inferolateral trunk (ILT), may also contribute to the arterial supply to the CCF. Except for the C4 segment of the ICA, these arteries are typically inconspicuous under normal conditions but become prominent in the presence of a CCF.[2,4] The most frequent venous drainage methods include SOV (88%), IPS (42%), and cortical venous drainage (34%).[2]

The arteriovenous connection in a DAVF, including the CCF, is called the fistula point or shunted pouch. It is a tubular or elliptical structure distinct from the main arteriovenous structure that converges multiple feeding arteries and connects them to the CS.[2] It typically involves only a portion of the anterior or posterior CS, with a predilection for the posterior compartment. Fistula points may be single or multiple and can be classified into dural, extradural, or osseous types on the basis of the location of arterial convergence.[2]

The detailed angiographic architecture of the CCF in this case revealed the left cavernous segment of the ICA with several small branches as the direct feeding artery, the left posterior CS as the fistula point, and the left IPS and the left SOV as the main draining vein. This angio-architecture corresponded to the Cognard classification of the type IIb.

Table 1: Classification of cerebral dural arteriovenous fistula[1,11]

Type	Djindjian-Merland, 1978	Borden, 1995	Cognard-Merland, 1995
I	Sinus	Sinus	Sinus, anterograde flow
II	Sinus with CVR	Sinus with CVR	IIa: sinus, retrograde flow IIb: CVR, rerograde flow IIa+b: combination
III	Direct CVD	Direct CVD	Direct CVD
IV	CVD with ectasia		CVD with ectasia
V			Spinal perimedullary drainage

CVD: cortical vein drainage; CVR: cortical vein reflux

Endovascular treatment is widely regarded as the first-line therapy for CCF, with a high cure rate and a low complication rate.[3,4,6] Various EVT techniques have been employed, including TVE, TAE, transorbital puncture, direct puncture or surgical exposure of the CS, and adjunctive radiosurgery.[3,4,6] For direct CCF, TAE aimed at closing the tear in the C4 segment of the ICA is considered the most favorable strategy.[12] This approach can be performed via detachable balloons, detachable coils, or their combinations. However, TAE was not chosen in this case because of concerns regarding the risk of inadvertent embolization into the venous system, which could lead to elevated intravenous pressure, jeopardize ocular vascular structures, and further exacerbate intraocular pressure. The approach using TVE is generally preferred over TAE in cases where the feeding artery is too small or tortuous for safe catheter navigation, when there is a heightened risk of embolic material migrating through ICA-ECA "dangerous anastomoses" that could lead to cerebral parenchymal infarction, or when the targeted feeding artery is in close proximity to the vasa nervorum, posing a risk of ischemic cranial neuropathy. A meta-analysis by Texakalidis et al revealed no statistically significant difference in outcomes between TAE and TVE in the treatment of all CCFs, including direct types.[5]

The approach using TVE may utilize several veins, including the IPS, the SOV via the angular vein, the SOV via the SMCV, the SPS via the transverse sinus, the pterygoid plexus via the maxillary vein, the inferior petrooccipital (petroclival) vein, or the contralateral CS via the intra-CS connection.[4,6] Access via IOV has also been reported, albeit rarely reported.[13] In this case, a transfemoral TVE approach through the IPS was selected because it is the shortest and most direct route to the CS. The location of the fistulous point in the posterior CS further supported the use of the IPS route, which offers better access than the SOV route-which is more suitable for anteriorly located fistulas. Additionally, the IPS was patent in this patient, facilitating straightforward catheterization and access to the CS. Alternative approaches, such as TVE via the SOV through the angular vein or SMCV, may be considered if the IPS route is not feasible.[3] These routes have demonstrated satisfactory obliteration outcomes in previous reports, although they are typically longer and more tortuous. In such cases, the "microcatheter milk" technique-where external manual manipulation over the skin is used to guide the catheter-may assist in navigating toward the fistula point. Other venous routes, including those via the SPS or pterygoid plexus, can also serve as alternative access points in cases where standard routes are unsuccessful.[4,6]

Transfemoral TVE with the IPS approach was performed via dual transfemoral access: arterial access for catheterization of the ipsilateral carotid artery to serve as a roadmap and venous access for catheterization of the ipsilateral IJV during embolization. The microcatheter was directed anteromedially at the level of the external auditory meatus and occipital bone to access the IJV-IPS junction. The IPS was subsequently selected, and a microcatheter was directed superomedially to the posterior CS.[4,6,14] Transfemoral TVE with the IPS approach was successfully performed in this case.

The embolic materials used for direct CCF may include detachable coils or liquid embolic agents.[1] In this case, coil embolization was initially performed because of the elevated risk of unintended peripheral embolization associated with liquid embolic agents when used alone. Liquid embolics may be used in conjunction with coils when complete occlusion is not achievable with coils alone. Advancements in endovascular techniques have also introduced the use of transarterial flow diverters to reconstruct the parent ICA and seal the fistulous tear. While this approach may offer comparable outcomes in terms of fistula closure, it is associated with significantly higher financial costs.[2,4,12] Another technical approach reported for TVE includes the reverse pressure cooker technique and reverse dual-lumen balloon microcatheter, which may be used to aid in the administration of liquid embolic agents.[15]

Alternative treatment modalities include direct puncture via a surgical approach and adjuvant radiosurgery.[1,2] Following the advice of endovascular treatment and the high chances of complications, the surgical approach was preserved as the first-line treatment for acute hemorrhagic CCF or second-line treatment for incomplete flow control for type III or IV CCF.[11] Direct puncture is usually performed when it is difficult for TVE or TAE to completely obliterate the CCF because of the difficulty of accessing the target vessels. Adjuvant radiosurgery, such as stereotactic gamma knife radiosurgery (SGKR), may be considered in cases where EVT results are unsatisfactory. Therapeutic doses for SGKR typically range from 20 to 50 Gy. While SGKR can achieve fistula obliteration in up to 90% of cases and symptomatic improvement in approximately 85% of cases, it is associated with a latency period before complete obliteration is achieved. This delay may increase the risk of hemorrhage, particularly in patients with retrograde cortical venous drainage.[2]

Potential complications of endovascular treatment include (2) transient complications such as facial pain, hematoma, or cranial nerve palsies; (2) technical issues including vessel injury, which may progress to intracranial hemorrhage caused by microwire or microcatheter manipulation; (3) serious events such as ischemic events due to embolic agent migration through dangerous anastomoses, distal embolism associated with high-flow fistulas, or venous infarction; and (4) rare events such as trigeminocardiac reflex-induced bradycardia and pseudoaneurysm.[2,7] Fortunately, none of these complications were observed in this case. To minimize the risk of such outcomes, careful preoperative planning, precise techniques during vascular access, and vigilant postoperative monitoring are essential.[2] The advantages of TVE include a lower risk of cranial neuropathy and a lower likelihood of inadvertent embolic agent migration than does TVA. However, TVE carries an increased risk of complications such as venous congestion and, in rare cases, technical rupture of pial veins.[15]

The primary objective in the treatment of CCF is not necessarily complete angiographic occlusion, but rather the elimination of cortical venous reflux and significant retrograde shunt flow. Although complete fistula occlusion remains the optimal therapeutic goal, the minimal acceptable outcome is the reduction of a high-flow direct CCF to a low-flow state. This is because residual low-flow CCFs have been reported to resolve spontaneously with conservative management.[2] Complete occlusion was achieved in this case.

CONCLUSION

The decision to treat CCF is guided by several factors, including its high-flow nature, symptom severity, the presence of retrograde venous drainage or cortical venous reflux, and the underlying angioarchitecture. Endovascular treatment remains the first-line treatment. TAE is typically preferred for direct CCFs because of their single arterial feeder. TVE may also be performed with comparable efficacy to TAE in achieving flow reduction and complete obliteration. It offers a reduced risk of distal embolization or inadvertent passage of embolic material through dangerous ICA-ECA anastomosis.

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