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Case Report

Dural arteriovenous fistulas of the anterior condylar confluence involving the anterior condylar vein within the hypoglossal canal: Two case reports

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ABSTRACT

Background: Dural arteriovenous fistulas (DAVFs) of the anterior condylar confluence (ACC) are rare vascular lesions at the skull base, often characterized by complex venous anatomy and variable clinical presentations. Their symptoms may overlap with those of cavernous sinus (CS) DAVFs, leading to potential misdiagnosis. Advanced imaging techniques and individualized treatment approaches are essential for accurate diagnosis and effective management.

Case Description: We present two cases of ACC DAVFs successfully treated with transvenous embolization (TVE). The first case involved a patient with hypoglossal nerve palsy and non-specific headache. The second case presented with cranial nerve III palsy, proptosis, and diplopia due to retrograde venous drainage into the CS, along with hypoglossal nerve symptoms, including tongue stiffness and difficulty speaking. Both patients experienced complete resolution of symptoms following treatment.

Conclusion: ACC DAVFs are challenging to diagnose and treat due to their anatomical complexity and diverse presentations. These cases highlight the efficacy and safety of TVE as the preferred treatment, underscoring the critical role of advanced imaging and individualized management in achieving favorable outcomes.

Keywords: Anterior condylar confluence, Anterior condylar vein, Cavernous sinus dural arteriovenous fistula, Dural arteriovenous fistulas, Hypoglossal canal

INTRODUCTION

Dural arteriovenous fistulas (DAVFs) are abnormal arteriovenous connections within the dura mater involving direct communication between meningeal arteries and venous structures, such as dural venous sinuses, cortical veins, or emissary veins. Although DAVFs most commonly occur in the transverse or sigmoid sinuses, a rare and clinically significant subtype is found at the

anterior condylar confluence (ACC), which serves as a major venous hub at the skull base. ACC DAVFs are uncommon, accounting for only 1.8–3.6% of all intracranial DAVFs, yet they are notable for their complex venous anatomy and diverse drainage patterns.[2,27] These lesions frequently involve venous structures within the hypoglossal canal, such as the anterior condylar vein (ACV), and their location and vascular connections pose unique diagnostic and therapeutic challenges.[16,19]

The nomenclature of DAVFs in the region of the ACC has been challenging to standardize due to the intricate anatomy and overlapping terminology of the craniocervical junction. The complexity of the venous structures in this area has led to these fistulas being variably referred to as those involving the hypoglossal canal, ACV, ACC, inferior petrosal sinus (IPS), marginal sinus, jugular foramen, or foramen magnum.[3,5,8,15,18,21,25] This variability reflects the difficulty in precisely defining their anatomical location and venous connections. However, our cases provide clear evidence pinpointing the anatomical origin of these fistulas, confirming their localization to the ACC and their involvement with the associated venous pathways. This clarification highlights the critical importance of detailed imaging and thorough anatomical assessment in accurately diagnosing and effectively managing these lesions.[22,23]

In the present study, we report two cases of ACC DAVFs, each illustrating distinct clinical and radiographic features. Both cases were successfully treated using transvenous embolization (TVE), demonstrating the efficacy of individualized management strategies. These cases also highlight the value of advanced imaging in clarifying the anatomical site of the fistula and guiding treatment. In addition, we review the anatomy, clinical presentations, and management strategies for ACC DAVFs, contributing to a deeper understanding of these complex lesions.

CASE DESCRIPTION

Case 1

A 61-year-old female with a history of dyslipidemia presented with a 1-month history of the right-sided headache radiating to the right maxillary region. The headache was characterized by a mixed pattern of pounding and pressure sensations, with a reported intensity of 6/10. Episodes occurred 3–4 times daily, each lasting approximately 30 min. Associated symptoms included nausea and difficulty controlling the right side of her tongue, leading to impaired mastication and unintentional retention of chewed food in the right buccal cavity. The patient also reported speech difficulties and persistent hiccups throughout this period. She denied any history of diplopia, facial numbness, motor weakness, or trauma.

On physical examination, vital signs were within normal limits, and systemic examination findings were unremarkable. Neurological evaluation revealed a normal level of consciousness. Significant findings included rightward deviation of the tongue with atrophy and fasciculations, along with flaccid dysarthria [Figure 1a]. A right carotid bruit was noted. Computed tomographic angiography (CTA) and magnetic resonance imaging (MRI) of the brain revealed an enlarged right ACC and a dilated venous plexus within the right hypoglossal canal [Figures 1b-d]. In addition, poor opacification of the left sigmoid sinus was observed, raising suspicion of chronic veno-occlusive disease. Cerebral angiography demonstrated a DAVF at the right ACC, supplied by multiple feeders, including bilateral ascending pharyngeal arteries (APAs), the right sphenopalatine artery, middle meningeal artery (MMA), bilateral meningohypophyseal trunks (MHTs), and muscular branches of bilateral vertebral arteries (VAs). Venous drainage was observed through the right jugular bulb and internal jugular vein (IJV) [Figure 2].

Under general anesthesia, TVE was performed through the dilated right IJV using fibered coils, achieving near-

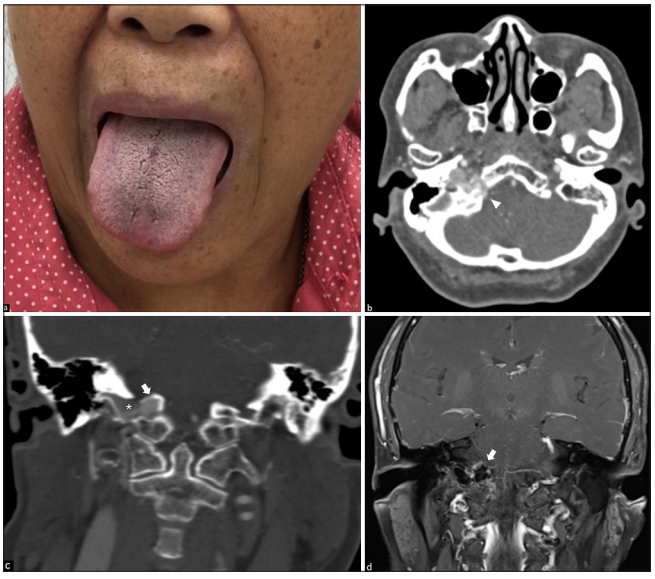

Figure 1: (a) Clinical photograph showing rightward deviation of the tongue. (b) Axial computed tomography angiography (CTA) reveals a dilated venous channel within the right hypoglossal canal (arrowhead). Coronal (c) CTA and (d) contrast-enhanced MRI demonstrating an enlarged right anterior condylar confluence (arrows) with a connection to the jugular bulb through a bridging vein (asterisk).

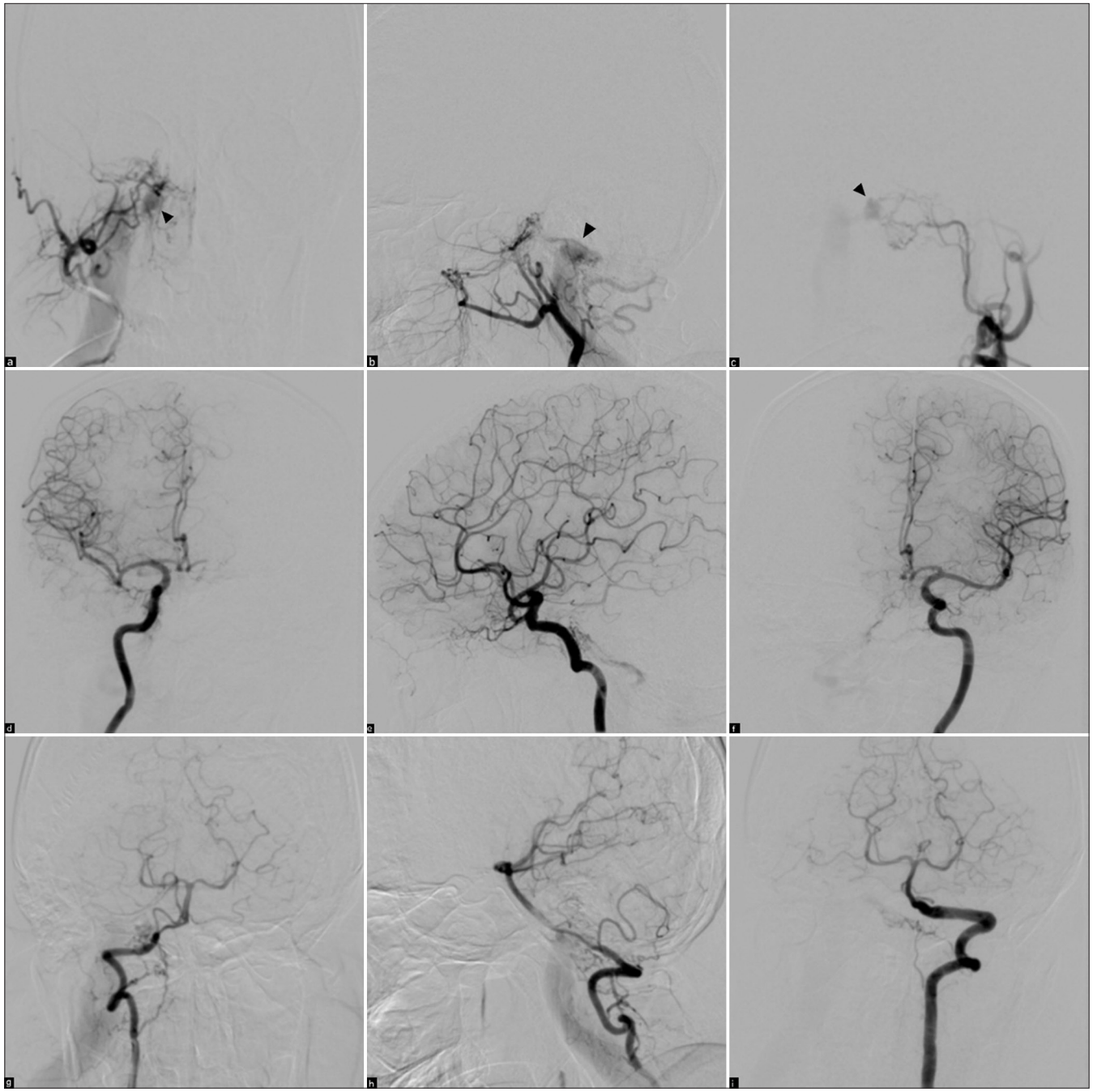

Figure 2: (a) Anteroposterior (AP) and (b) lateral views of the right external carotid artery (ECA) injection; (c) AP view of the left ECA injection; (d) AP and (e) lateral views of the right internal carotid artery (ICA) injection; (f) AP view of the left ICA injection; (g) AP and (h) lateral views of the right vertebral artery (VA) injection; and (i) AP view of the left VA injection demonstrate a dural arteriovenous fistula at the right anterior condylar confluence (ACC). Multiple feeders, including bilateral ascending pharyngeal arteries, the right sphenopalatine artery, middle meningeal artery, bilateral meningohypophyseal trunks, and muscular branches of bilateral VAs supply the fistula. Venous drainage occurs through the right jugular bulb and internal jugular vein. Arrowheads in (a), (b), and (c) highlight the dilated ACC.

complete obliteration of the fistula [Figure 3]. The patient's headache improved significantly, resolving completely within 1 month. Cerebral angiography performed 4 months post-embolization confirmed complete obliteration of the fistula [Figures 4a-h]. Gradual improvement in tongue symptoms was observed, with complete resolution achieved 2 years after the procedure. At the 5-year follow-up, the patient remained asymptomatic. Follow-up imaging, including MRI and MR angiography (MRA), demonstrated no recurrence of the fistula [Figure 4i].

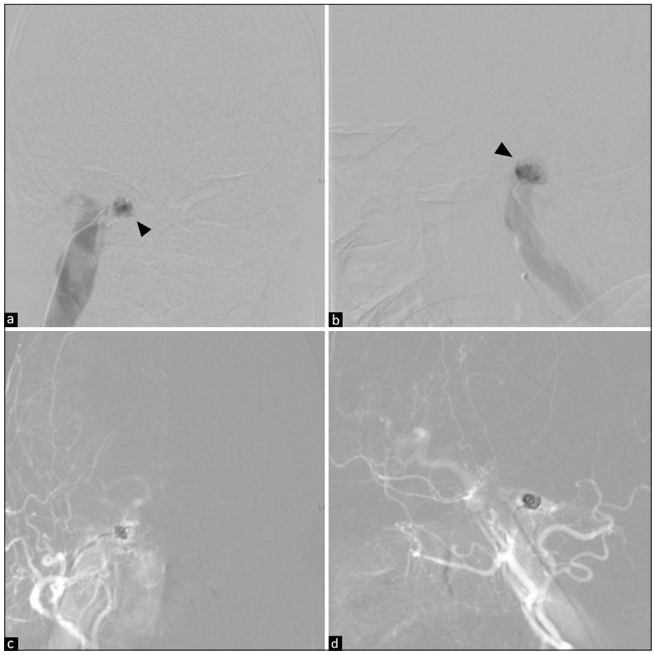

Figure 3: (a) Anteroposterior (AP) and (b) lateral views of venography performed via microcatheter injection clearly demonstrate the enlarged right anterior condylar confluence (arrowheads). (c) AP and (d) lateral views of the right external carotid artery injection show fibered coil placement within the venous pouch under road-mapping guidance.

Case 2

A 59-year-old woman with a history of hypertension initially presented to a local hospital with a 1-week history of left eye ptosis and diplopia. She also reported persistent tinnitus in the left ear, which had begun 2 weeks before her presentation. The patient denied any history of recent trauma. A contrast-enhanced computed tomography scan of the brain performed at the local hospital demonstrated dilation of the left cavernous sinus (CS) [Figure 5a]. Based on these findings, a provisional diagnosis of CSDAVF was made, and the patient was referred to our institute for further evaluation and management.

On admission to our hospital, additional symptoms were identified, including tongue stiffness and difficulty with speech. On physical examination, mild proptosis of the left eye was noted, accompanied by a cranial bruit in the left periorbital region and the left posterior auricular area. Fasciculations were observed on the left side of the tongue [Figure 5b].

The ophthalmologic evaluation revealed pupils measuring 3 mm bilaterally, with the left pupil demonstrating non-reactivity to light (both direct and consensual responses). Complete ptosis of the left eye was observed, and extraocular movements were restricted in upward, downward, and medial gaze, consistent with complete cranial nerve (CN) III palsy. These findings underscored the need for targeted imaging and further management to confirm the diagnosis and guide treatment.

Cranial CTA revealed a dilated left ACC and venous plexus within the left hypoglossal canal [Figures 5c and d]. Cerebral angiography confirmed a DAVF at the left ACC, supplied by multiple feeders, including bilateral APAs, the left sphenopalatine artery, MMA, posterior auricular artery, occipital artery (OA), bilateral MHTs, and muscular branches of the left VA. Venous drainage was observed downward through the left jugular bulb and IJV, with additional drainage into the suboccipital venous plexus. Upward drainage extended into the left IPS, with subsequent drainage to the left CS and left-sided cortical veins [Figure 6].

Under general anesthesia, TVE was performed through the dilated left IJV using fibered coils, achieving near-complete obliteration of the fistula [Figure 7]. Cerebral angiography performed 4 months post-embolization confirmed complete obliteration of the fistula [Figures 8a-h]. The patient's symptoms gradually improved, with complete recovery achieved 1 year after the procedure. At the 3-year follow-up, the patient remained asymptomatic. Follow-up imaging, including MRI and MRA, demonstrated no recurrence of the fistula [Figure 8i].

DISCUSSION

Anatomy of the ACC

The ACC is a critical venous structure located extracranially at the opening of the hypoglossal canal at the skull base. This small but significant venous confluent, typically measuring around 5 mm in diameter, serves as a central hub for venous drainage, connecting the IJV, posterior fossa dural venous sinuses, and vertebral venous plexus (VVP).[8,21] Positioned anterior to the jugular foramen and medial to the IJV, the ACC facilitates multidirectional venous flow through its intricate connections, which include tributaries from the basilar plexus, marginal sinus and emissary veins such as the lateral and posterior condylar veins.[7,21]

The ACC is closely associated with the ACV, which traverses the hypoglossal canal alongside the hypoglossal nerve and branches of the APA.[19,24] This venous plexus provides critical links between the ACC and the anterior internal VVP. In addition, the ACC connects to the IPS through the inferior petro-occipital vein, offering an indirect route to the CS.[21,24] Its communication with the VVP through the ACV and lateral condylar veins further emphasizes its role in facilitating cerebral venous outflow, particularly

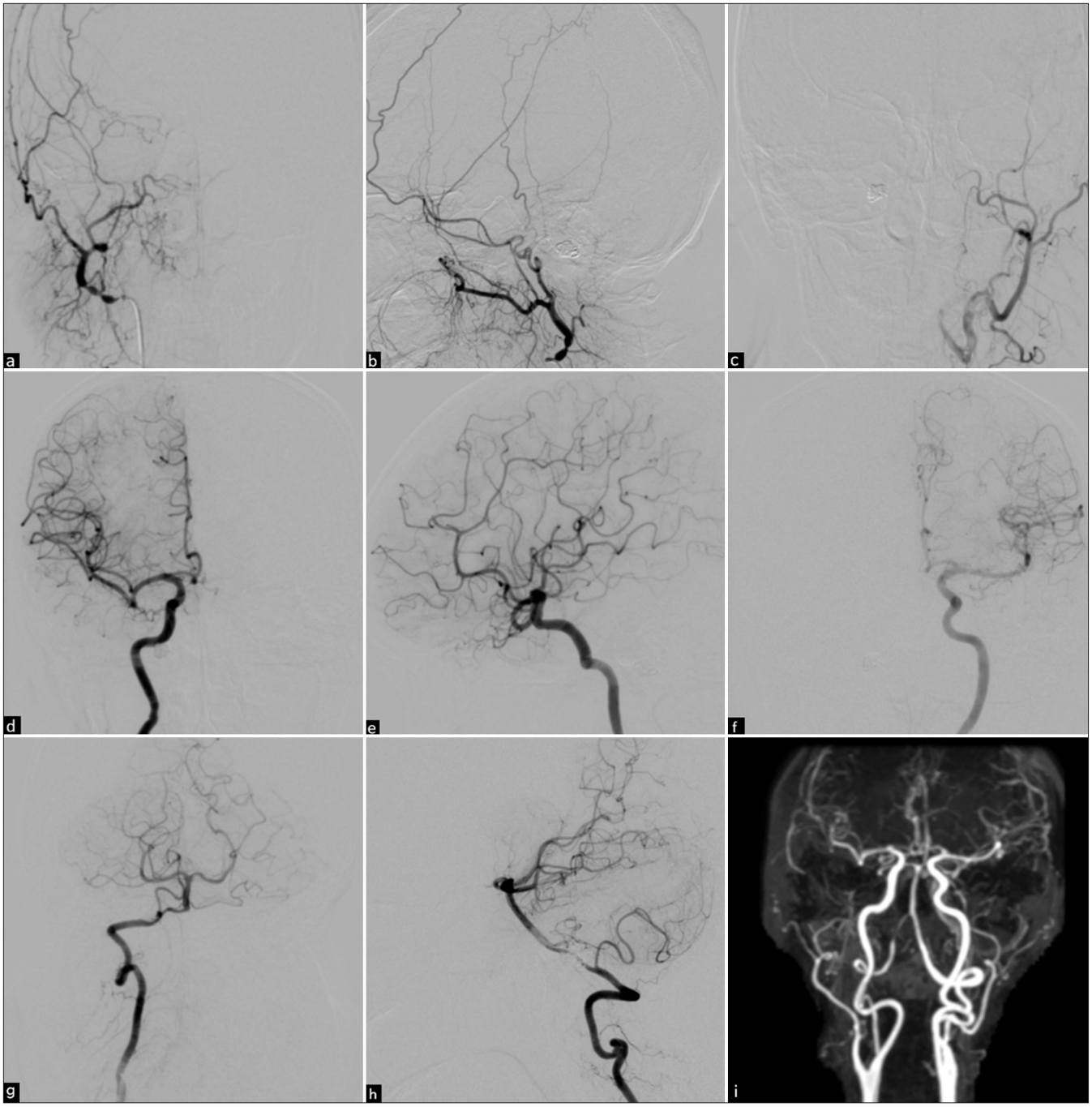

Figure 4: Cerebral angiography performed 4 months after embolization. (a) Anteroposterior (AP) and (b) lateral views of the right external carotid artery (ECA) injection; (c) AP view of the left ECA injection; (d) AP and (e) lateral views of the right internal carotid artery (ICA) injection; and (f) AP view of the left ICA injection; (g) AP and (h) lateral views of the right vertebral artery injection confirm complete obliteration of the fistula. (i) Contrast-enhanced magnetic resonance angiography obtained 5 years after embolization demonstrates no recurrence of the fistula.

during changes in body position. This complex network underscores the ACC's importance as a key anatomical structure in the craniocervical junction and its relevance in the diagnosis and management of venous pathologies, including DAVFs.[7,19]

Blood supply in ACC DAVFs

The feeders and drainage patterns in ACC DAVFs reflect the complex vascular anatomy of the skull base. The arterial supply of ACC DAVFs typically originates from branches

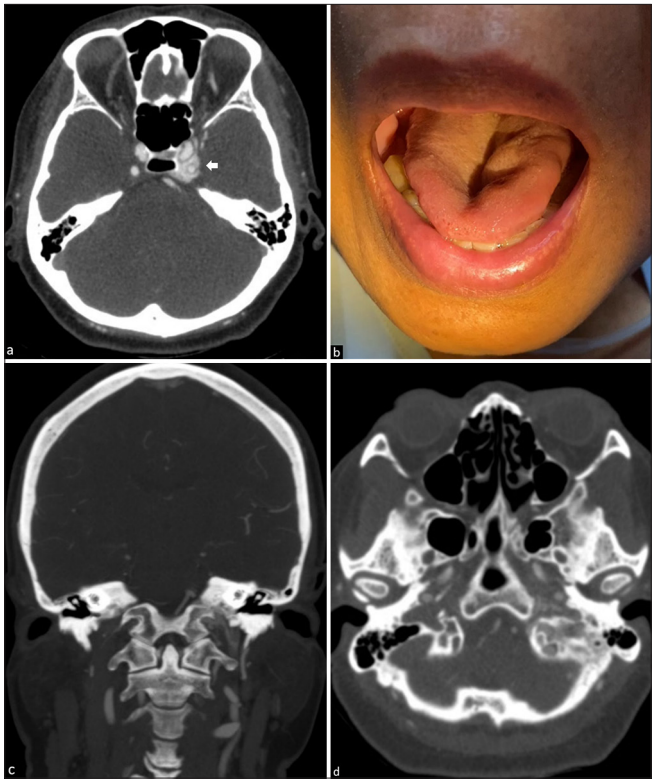

Figure 5: (a) Axial contrast-enhanced computed tomography (CT) scan revealing dilation of the left cavernous sinus (arrow). (b) Clinical photograph showing fasciculations on the left side of the tongue. (c) Coronal and (d) axial CT angiography demonstrate a dilated left anterior condylar confluence and venous plexus within the left hypoglossal canal.

of the external carotid artery, with the APA being the most consistent feeder. Other contributing arteries may include the OA, MMA, and posterior auricular artery. In some cases, feeders from the internal carotid artery (ICA), particularly the MHT, and branches from the VA also supply the fistula. These arteries converge at or near the ACC, forming abnormal arteriovenous connections.[16,23,25]

Venous drainage and clinical implications of ACC DAVFs

The clinical presentation of DAVFs involving the ACC varies widely, reflecting the region's complex venous anatomy and diverse drainage patterns.[4] Anterograde drainage to the IJV or VVP frequently results in pulse-synchronous tinnitus, as observed in one of our cases, where the patient presented with right-sided tinnitus and associated hypoglossal nerve palsy. Conversely, retrograde drainage into the IPS and CS can lead to orbital symptoms such as diplopia, proptosis, and chemosis, mimicking CSDAVFs.[13,14] In our second case, the patient exhibited left eye proptosis, diplopia, and CN III palsy caused by venous congestion in the CS. Persistent tinnitus, in this case, underscored the overlap of symptoms arising from multiple venous drainage patterns.

The symptomatology of ACC DAVFs may evolve due to changes in venous drainage patterns caused by venous stenosis, thrombosis, or high-flow vasculopathy. For instance, a patient presenting with pulsatile tinnitus might later develop ocular symptoms or hypoglossal palsy as the drainage shifts retrograde into the CS.[23,25]

Rare complications of ACC DAVFs include congestive myelopathy, which results from venous hypertension affecting the anterior spinal vein.[1] Limb weakness, urinary disturbances, and spinal cord edema characterize this condition. Non-specific symptoms, such as headache and otalgia, have also been reported in some cases. In one of our cases, the patient presented with a non-specific headache in addition to more specific symptoms, highlighting the overlapping and variable clinical presentations of ACC DAVFs.

Hypoglossal nerve palsy, though uncommon, may occur due to irritation or compression of the nerve adjacent to the fistula site. Clinical studies indicate that hypoglossal nerve palsy is observed in approximately 11.7% of patients with ACC DAVFs.[19,25] In our two cases, venous drainage patterns were closely associated with CN XII dysfunction, leading to tongue-related symptoms such as stiffness, fasciculations, and difficulty speaking. These symptoms are likely attributable to venous congestion within or near the hypoglossal canal. The venous plexus surrounding the ACC, combined with its proximity to the hypoglossal canal, predisposes the hypoglossal nerve to compression or ischemic injury.

Intracranial hemorrhage, though infrequent (5%), represents a severe complication, particularly in cases with perimedullary venous drainage. Posterior fossa subarachnoid or cerebellar hemorrhage may arise from significant venous hypertension, necessitating immediate intervention.[8,25]

Possible pathogenesis of ACC DAVFs

The pathogenesis of ACC DAVFs remains unclear, although recent studies have provided insights into potential mechanisms. ACC DAVFs are hypothesized to originate as acquired lesions influenced by factors such as trauma, venous outflow obstruction, or thrombosis, which may result in venous hypertension. Venous hypertension, often secondary to conditions such as IJV stenosis, thrombosis, or compression, disrupts the normal flow dynamics at the ACC. This leads to the rerouting of venous drainage pathways, frequently into collateral channels such as the VVP or IPS. Prolonged venous hypertension can induce ischemia and activate angiogenic factors, including vascular endothelial growth factor and basic fibroblast growth factor. These factors promote abnormal neovascularization and the formation of pathological arteriovenous connections.[10,20,28]

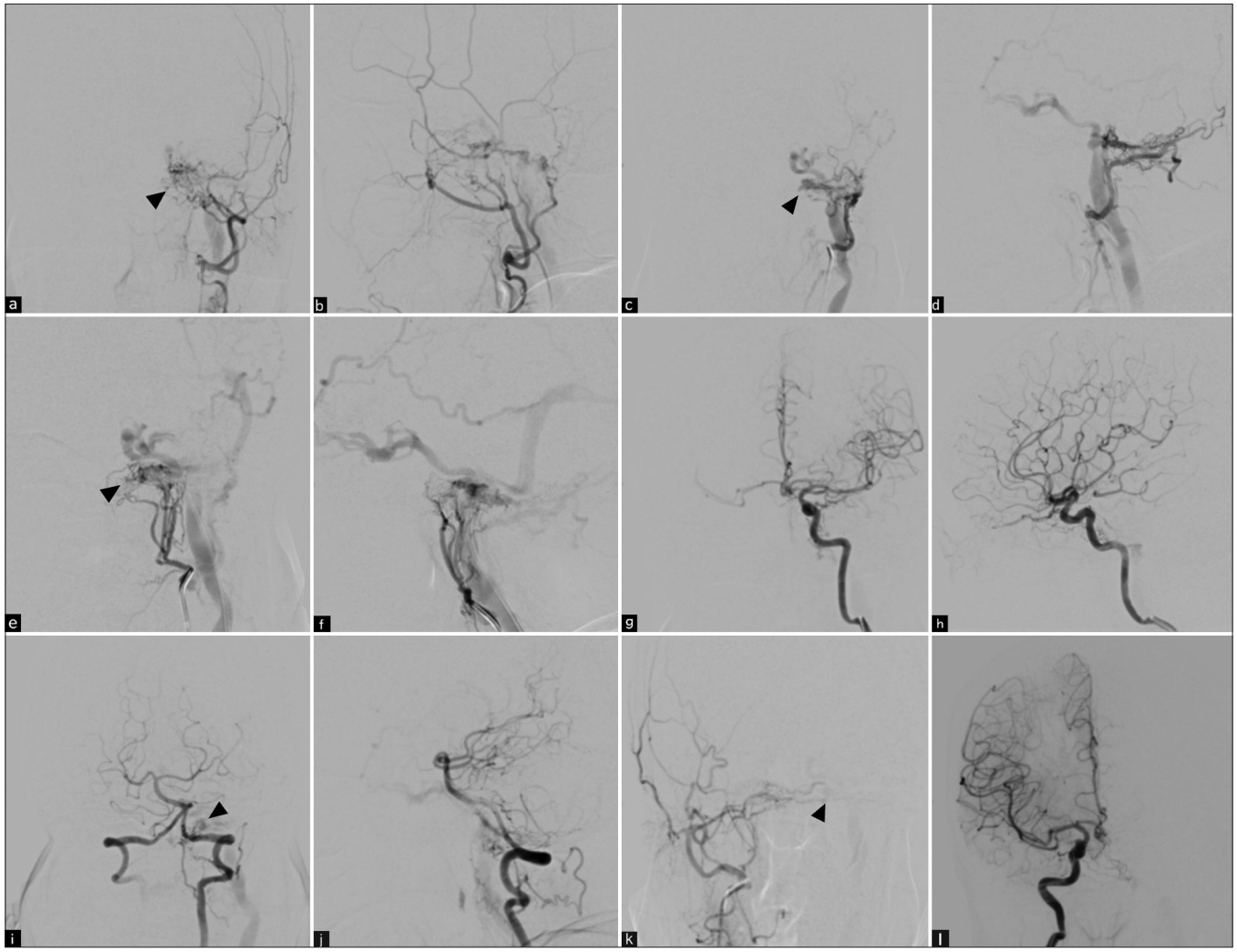

Figure 6: (a) Anteroposterior (AP) and (b) lateral views of the left external carotid artery (ECA) injection; (c) AP and (d) lateral views of the left occipital artery injection; (e) AP and (f) lateral views of the left ascending pharyngeal artery injection; (g) AP and (h) lateral views of the left internal carotid artery (ICA) injection; (i) AP and (j) lateral views of the left vertebral artery (VA) injection; (k) AP view of the right ECA; and (l) AP view of the right ICA injection demonstrate a dural arteriovenous fistula (DAVF) at the left anterior condylar confluence (ACC). The fistula is supplied by multiple feeders, including bilateral ascending pharyngeal arteries, the left sphenopalatine artery, middle meningeal artery, posterior auricular artery, occipital artery, bilateral meningohypophyseal trunks, and muscular branches of the left VA. Venous drainage occurs downward through the left jugular bulb and internal jugular vein, with additional drainage into the suboccipital venous plexus. Upward drainage is observed into the left inferior petrosal sinus, extending to the left cavernous sinus and left-sided cortical veins. Arrowheads in (a), (c), (e), (i), and (k) highlight the dilated ACC.

In our first case, the presence of chronic veno-occlusive disease in the contralateral sigmoid sinus provides an illustrative example. Such venous occlusion may induce compensatory hemodynamic changes and venous hypertension, increasing the likelihood of DAVF formation at alternative venous pathways. This phenomenon highlights how disturbed venous drainage can lead to the development of DAVFs, particularly in anatomically complex areas like the ACC. In addition, *de novo* formation of DAVFs has been documented after endovascular treatment of other lesions, further supporting the role of altered venous hemodynamics and thrombosis as critical contributors to their pathogenesis.[6,9,17]

Management strategy

Advanced imaging techniques play a critical role in the management of ACC DAVFs. Digital subtraction angiography (DSA), often complemented by angiographic CT and high-resolution CTA or MRA, is an indispensable tool for accurately mapping the venous architecture and identifying feeding arteries, drainage patterns, and critical

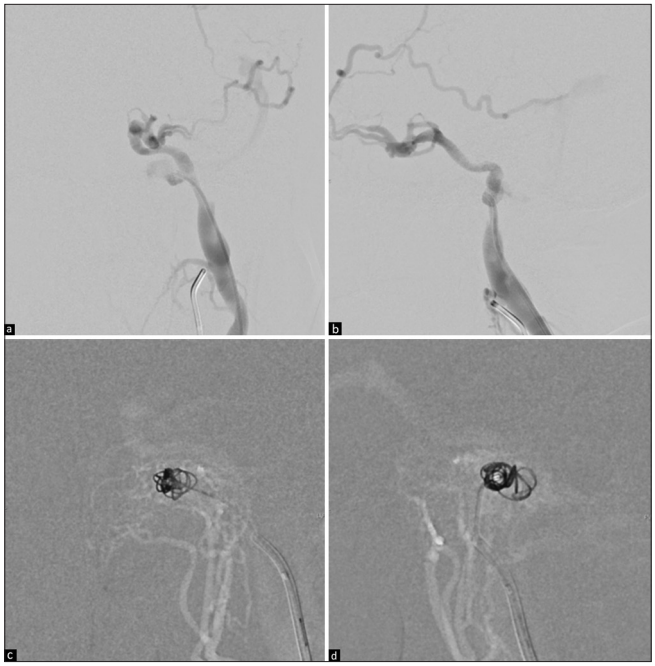

Figure 7: (a) Anteroposterior (AP) and (b) lateral views of venography performed before embolization clearly demonstrate the enlarged left anterior condylar confluence (ACC). (c) AP and (d) lateral views of the left ascending pharyngeal artery injection show fibered coil placement within the ACC under road mapping guidance.

anastomoses. These imaging modalities provide detailed visualization, enabling precise diagnosis and guiding the selection of therapeutic strategies tailored to the individual anatomy and pathology of each patient.[12,22]

Management of DAVFs involving the ACC focuses on achieving complete obliteration of the fistula while preserving normal venous drainage and minimizing complications. The choice of treatment depends on the drainage pattern, severity of symptoms, and accessibility of the fistulous point.

TVE remains the cornerstone treatment for ACC DAVFs, offering high efficacy and safety.[5,8,25] In both of our cases, we focused on targeted embolization at the ACC. In the first case, TVE was performed through a dilated IJV using fibered coils, successfully addressing the anterograde drainage pattern and resolving symptoms of pulse-synchronous tinnitus and hypoglossal nerve palsy. In the second case, TVE targeted the fistula at the ACC, addressing the retrograde venous drainage that caused CN III palsy, proptosis, diplopia, and hypoglossal nerve symptoms. Coil embolization achieved complete obliteration of the fistula in both cases, leading to full resolution of symptoms. These outcomes underscore the importance of precise catheter navigation and advanced imaging in ensuring effective treatment.

In our approach to treating ACC DAVFs, we prioritized avoiding tight packing of coils within the hypoglossal canal to minimize the risk of hypoglossal nerve compression. Instead, we utilized fibered coils, which promote progressive thrombosis to achieve complete obliteration of the fistula.[11] This technique reduces the likelihood of direct mechanical compression on the hypoglossal nerve by relying on gradual occlusion of the venous flow rather than dense packing of the coils.

Our cases support this approach, as both patients experienced complete clinical recovery following treatment without evidence of nerve-related complications. The progressive thrombosis induced by the fibered coils allowed for effective fistula closure without exerting additional pressure on surrounding neurovascular structures, including the hypoglossal nerve. This outcome highlights the importance of tailoring embolization strategies to balance effective fistula treatment with the preservation of critical neurovascular function.

Overpacking with coils within the hypoglossal canal poses a significant risk of hypoglossal nerve palsy, a complication resulting from compression of the hypoglossal nerve due to excessive coil volume.[18,23] To mitigate this risk, targeted embolization of the ACC and adjacent venous pouches is crucial. Proper planning and precise coil deployment are essential to minimize complications while achieving complete obliteration of the fistula. The potential for hypoglossal nerve palsy caused by the mass effect of coils in the ACV should always be carefully considered during treatment planning and execution.[2,22]

Transarterial embolization (TAE) serves as an adjunctive treatment in cases with multiple arterial feeders or when venous access is restricted. Although not utilized in our cases, TAE can effectively reduce arterial inflow to the fistula before definitive TVE. Liquid embolic agents, such as Onyx or N-butyl cyanoacrylate, are commonly used to diminish the fistula's supply but are generally not curative as standalone treatments.[26,29] Despite its utility, TAE carries significant risks due to the intricate vascular anatomy of ACC DAVFs. The APA, a frequent arterial feeder, supplies the vasa nervorum of CNs IX–XII, posing a risk of lower CN palsies if these branches are inadvertently embolized. In addition, anastomoses between the APA and major vessels such as the ICA or VA may allow embolic materials to migrate, increasing the risk of ischemic stroke.[2] Thus, meticulous angiographic evaluation to map these connections and precise embolization techniques are crucial to minimizing complications, underscoring the need for careful planning and execution in TAE for ACC DAVFs.

Surgical options are rarely necessary and are reserved for refractory cases or those with cortical venous reflux posing a high risk of intracranial hemorrhage. Given the success of TVE in both of our cases, surgery was not required. However, the risks associated with surgical disconnection, such as CN damage, make it a less favorable option.[5]

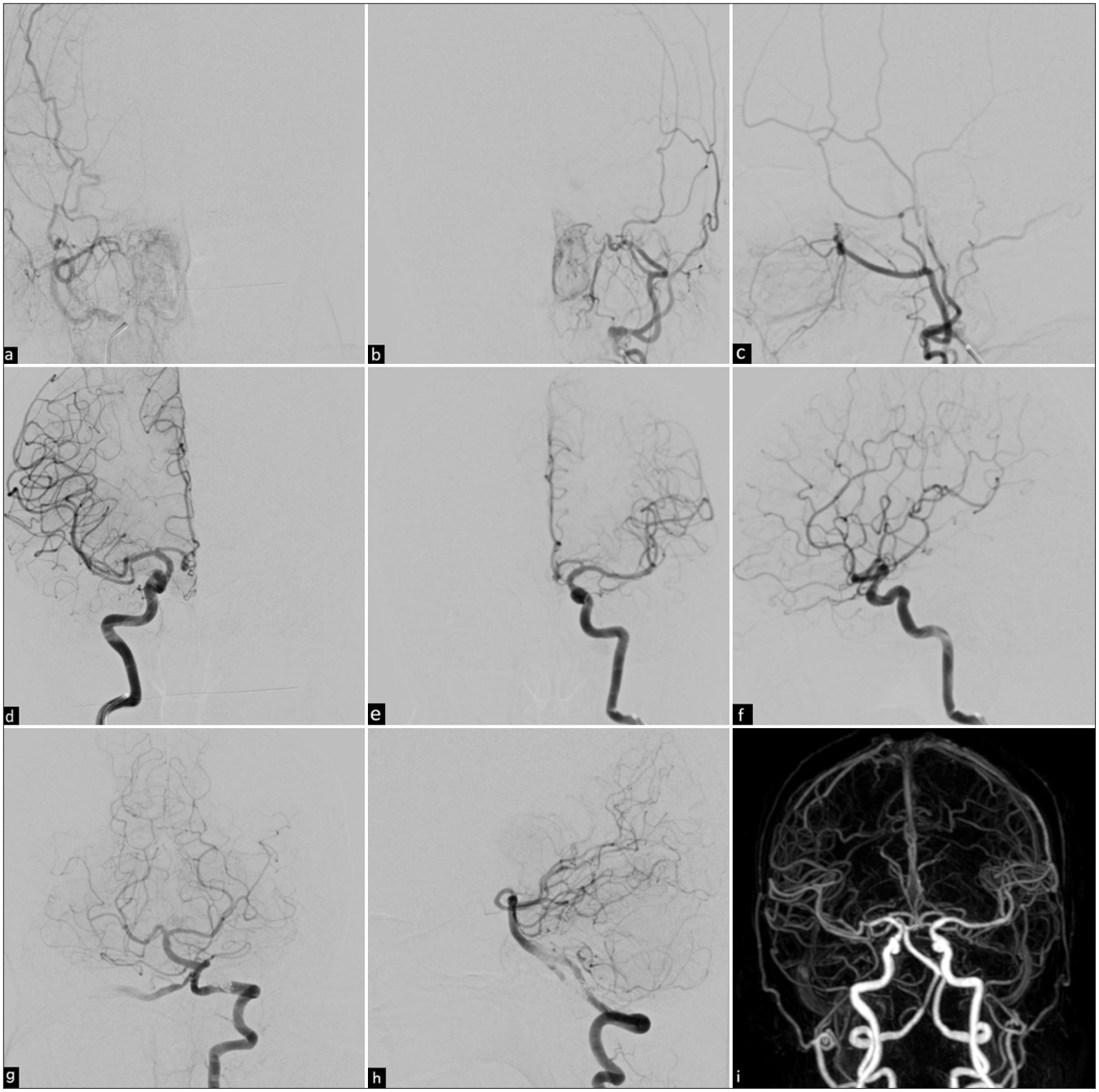

Figure 8: Cerebral angiography performed 4 months after embolization. (a) Anteroposterior (AP) view of the right external carotid artery (ECA) injection; (b) AP and (c) lateral views of the left ECA injection; (d) AP view of the right internal carotid artery (ICA) injection; (e) AP and (f) lateral views of the left ICA injection; and (g) AP and (h) lateral views of the left vertebral artery injection confirm complete obliteration of the fistula. (i) Contrast-enhanced magnetic resonance angiography obtained 3 years after embolization demonstrates no recurrence of the fistula.

The importance of post-treatment follow-up

Clinical and imaging follow-up are essential components of post-treatment care for ACC DAVFs to monitor for recurrence and ensure long-term success. Despite the high efficacy of treatments such as TVE and TAE, there remains a risk of residual or recurrent fistulas due to incomplete obliteration or revascularization over time. Recurrence may be asymptomatic initially, making routine imaging crucial for early detection.[6]

Follow-up imaging, including DSA, MRA, or CTA, should be performed at regular intervals, typically within 3–6 months

after treatment and subsequently on an annual basis. These modalities allow for detailed assessment of the treated site, identification of any residual arteriovenous shunting, and evaluation of venous drainage patterns. Clinical follow-up is equally important to detect any recurrence-related symptoms, such as headache, tinnitus, or CN dysfunction. Early detection of recurrence enables timely intervention, reducing the risk of complications such as venous hypertension, intracranial hemorrhage, or neurological deficits. By integrating routine clinical evaluation with advanced imaging, the risk of long-term complications can be minimized, ensuring optimal outcomes for patients with ACC DAVFs.

Both of our cases of ACC DAVFs were successfully treated with TVE, resulting in complete obliteration of the fistulas. Follow-up imaging, including DSA and MRA, confirmed complete obliteration and no recurrence over the long term. These outcomes highlight the efficacy of TVE and the critical importance of long-term monitoring for ensuring sustained success.

CONCLUSION

ACC DAVFs are rare but clinically significant vascular lesions at the skull base, characterized by complex feeders and diverse venous drainage patterns. These fistulas often pose diagnostic challenges due to overlapping symptoms with other skull base DAVFs, such as those involving the CS. Advanced imaging techniques, including DSA and high-resolution CT or MR angiography, are crucial for mapping venous architecture, guiding treatment strategies like TVE, and optimizing patient outcomes.

Our cases demonstrate the efficacy and safety of TVE as a definitive treatment for ACC DAVFs, achieving complete fistula obliteration with resolution of symptoms. These outcomes highlight the importance of meticulous preoperative planning, individualized treatment approaches, and targeted embolization to minimize complications, such as hypoglossal nerve palsy. Long-term follow-up with clinical evaluation and imaging is essential to monitor for recurrence, emphasizing the need for ongoing care to ensure sustained success.

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