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Case Report

Dural arteriovenous fistulas of the occipital sinus secondary to trauma: Two case reports and a review of the literature

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ABSTRACT

Background: Intracranial dural arteriovenous fistulas (DAVFs) involving the occipital sinus (OS) are rare vascular anomalies characterized by abnormal connections between meningeal arteries and venous sinuses or cortical veins. Trauma and venous hypertension are recognized factors in the pathogenesis of DAVFs, with previous injuries to the venous sinus and subsequent angiogenic responses contributing to abnormal arteriovenous shunt formation. The OS's variable anatomy and deep midline location add unique challenges to the diagnosis and treatment of DAVFs in this region.

Case Description: We report two cases of OS DAVFs in patients with a history of remote cranial trauma. The first case describes a 36-year-old man with a 3-year history of progressive headache, recently worsening with severe headache, nausea, and vomiting. Imaging revealed a DAVF at the OS with cortical venous reflux. After an unsuccessful attempt at transarterial embolization, transvenous embolization achieved near-complete obliteration, and the patient remained asymptomatic at the 3-year follow-up. The second case involves a 54-year-old man with a history of a high fall. He initially presented with bilateral leg numbness and urinary retention, progressing to quadriparesis. Imaging demonstrated an OS DAVF with spinal venous congestion and cervical cord compression. Following an unsuccessful transarterial approach, he underwent a suboccipital craniotomy with OS ligation. Despite complete obliteration, he remained significantly disabled at the 1-year follow-up.

Conclusion: These cases highlight the role of trauma in the development of OS DAVFs and the challenges associated with their management. Successful treatment often requires a combined approach due to complex arterial feeders and venous drainage patterns. Early intervention is crucial in preventing irreversible neurological deficits caused by prolonged venous congestion, emphasizing the need for timely diagnosis and individualized treatment strategies for DAVFs involving the OS.

Keywords: Dural arteriovenous fistula, Occipital sinus, Thrombosed venous aneurysm, Transvenous embolization, Trauma-induced vascular malformation

INTRODUCTION

Intracranial dural arteriovenous fistulas (DAVFs) are rare vascular malformations involving abnormal connections between dural arteries and venous sinuses or cortical veins.[7,8] Among these, DAVFs of the occipital sinus (OS) are exceptionally rare, likely due to the small size

and variable persistence of the OS in adults.[5,17] The OS, positioned along the midline of the occipital bone within the falx cerebelli, is typically formed during early embryonic development, often regressing postnatally. However, in cases where the OS persists, it can serve as a collateral venous route, particularly if the transverse or sigmoid sinuses are hypoplastic or obstructed.[14] Persistent OS has been associated with unusual venous drainage pathways, contributing to the formation of DAVFs in some cases.[2,10]

Trauma is recognized as a possible risk factor for DAVF formation, as it can lead to dural sinus injury, venous hypertension, and subsequent angiogenesis, resulting in the formation of pathological arteriovenous connections.[16] Studies have documented that DAVFs associated with a history of trauma often involve complex drainage patterns and multiple arterial feeders, making them more challenging to treat.[9] In DAVFs involving the OS, these factors are further compounded by the anatomical variation and deep midline location of the OS, which can complicate both diagnosis and management.

In this report, we present two cases of OS DAVFs with a history of remote trauma. Both cases highlight the diagnostic and therapeutic challenges associated with OS DAVFs, particularly those with aggressive venous drainage patterns. We aim to provide insights into the pathogenesis, clinical presentation, and management of these rare lesions, emphasizing the potential role of trauma in their formation and the importance of individualized treatment strategies.

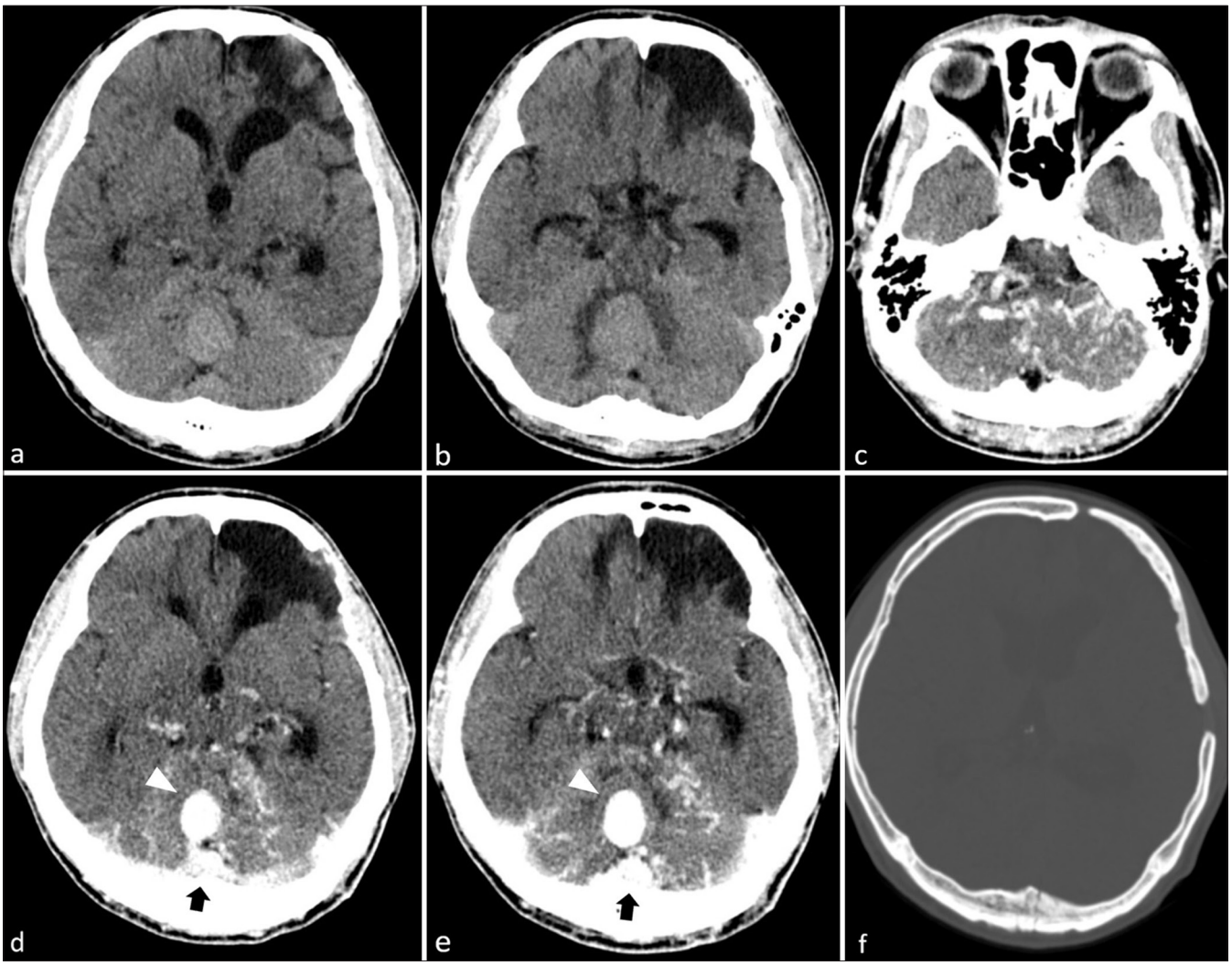

Figure 1: Axial computed tomography (CT) scans of the brain: (a and b) non-contrast images and (c-e) contrast-enhanced images reveal a large enhancing varix adjacent to the occipital sinus, along with bilateral dilated cerebellar veins. Encephalomalacic changes are observed in the left frontal lobe. (f) Axial bone-window CT scan shows evidence of a previous left frontoparietal craniotomy. Arrowheads in (d and e) indicate the enhancing varix, and arrows in (d and e) indicate the enhancing occipital sinus.

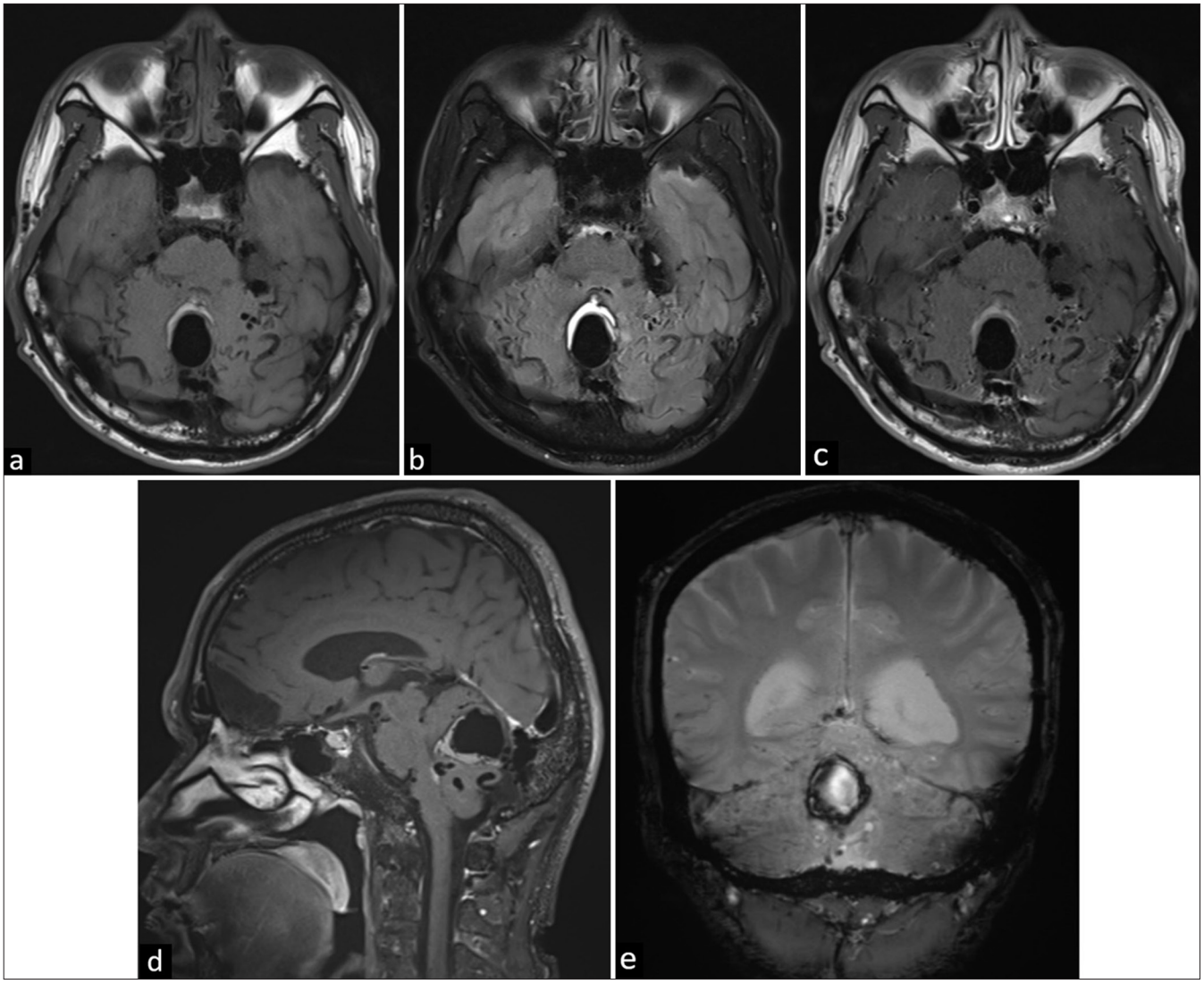

Figure 2: Magnetic resonance imaging of the brain obtained 2 weeks after symptom onset. (a) Axial T1-weighted, (b) axial fluid-attenuated inversion recovery, (c) axial T1-weighted with contrast, (d) sagittal T1-weighted with contrast, and (e) coronal T2* weighted gradient echo images reveal a large venous pouch with heterogeneous signal intensity in the anteroinferior aspect, likely indicating a partially thrombosed aneurysm. This large aneurysm is situated anterior to the occipital sinus. Bilateral dilated cerebellar veins are also observed. The asterisk (*) in T2-weighted gradient echo (GRE) imaging is used to denote the sequence type, a convention to describe the imaging technique.

CASE DESCRIPTION

Case 1

A 36-year-old man presented with a progressive headache persisting over 3 years, which had intensified over the 2 weeks before admission, accompanied by severe headache, nausea, and vomiting. His medical history was notable for a severe head injury 12 years earlier, which had required a left frontal craniotomy. Initially evaluated at a local hospital, a computed tomography (CT) scan of his brain revealed abnormal vascular structures in the posterior fossa [Figure 1], prompting referral to our institute for further assessment and management.

On neurological examination, the patient exhibited truncal ataxia and impaired coordination on finger-to-nose testing bilaterally, though he showed no motor weakness. He was alert oriented, and the remainder of his clinical examination was unremarkable.

Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) of the brain, performed 2 weeks after symptom onset, revealed a large venous pouch with heterogeneous signal intensity in the anteroinferior region, suggestive of a partially thrombosed aneurysm. This aneurysm was located anterior to the OS, and bilateral dilated cerebellar veins were also observed. These findings were consistent with a DAVF of the OS with malignant cortical venous reflux [Figures 2 and 3].

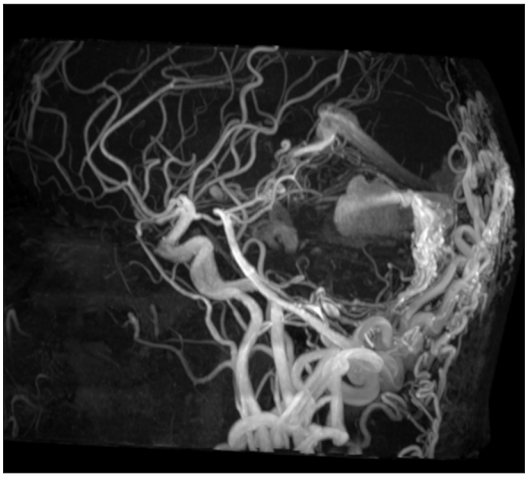

Figure 3: Sagittal maximum intensity projection image from contrast-enhanced magnetic resonance angiography demonstrates multiple dilated and tortuous vessels around the midline occipital bone, along with a large venous varix.

Cerebral angiography further delineated the DAVF at the OS, with arterial supply originating from multiple branches, including the bilateral occipital arteries (OAs), middle meningeal arteries (MMAs), posterior inferior cerebellar arteries (PICAs), meningohypophyseal trunks, ascending pharyngeal arteries, and posterior meningeal arteries (PMAs). The DAVF drained into a large venous varix located anterior to the OS, with further drainage into cerebellar veins and subsequently into the anterior spinal vein, corresponding to a Cognard type V DAVF classification. The caudal portion of the OS was occluded [Figures 4 and 5].

Under general anesthesia, transarterial embolization (TAE) of the fistula was attempted through the dilated left PICA but was unsuccessful. Consequently, transvenous embolization (TVE) was performed through the right transverse sinus using fibered coils, achieving nearly complete obliteration of the fistula [Figure 6]. The patient demonstrated marked clinical improvement following the procedure and was able to resume work.

At the 3-year follow-up, the patient remained asymptomatic. Follow-up imaging, including MRI, MRA, and cerebral angiography, confirmed complete obliteration of the OS DAVF with no signs of recurrence [Figure 7].

Case 2

A 54-year-old man with a history of a high fall from a tree 10 years prior presented to a local hospital 5 months before admission, reporting bilateral leg numbness without motor weakness. He also experienced urinary retention and constipation. Evaluation by a urologist, including cystoscopy, indicated bladder atony, leading to the insertion of a Foley catheter. Three months before admission, the patient developed progressive quadriparesis, with muscle strength graded 4 in the upper extremities and 1–2 in the lower extremities, rendering him unable to walk. A cervical spine MRI conducted at the local hospital raised suspicion of a spinal cord tumor, resulting in a referral to our institute [Figure 8].

On admission, a CT scan of the brain revealed an old cerebral contusion in the bilateral anterior and inferior frontal lobes, as well as a linear fracture in the left posterior fossa [Figure 9]. MRA identified a DAVF of the OS, with drainage into both the anterior and posterior spinal veins, causing venous congestion of the cervical cord [Figure 10]. Cerebral angiography, along with maximum intensity projection-reformatted images from angiographic CT and 3D reconstruction, clearly demonstrated an OS DAVF fed by the meningeal branch of the left PICA, with drainage into the anterior and posterior spinal veins, corresponding to a Cognard type V DAVF classification. The cranial portion of the OS was occluded [Figures 11 and 12].

An initial attempt at TAE through the left PICA was unsuccessful due to the vessel's small and tortuous origin. The patient subsequently underwent a suboccipital craniotomy, during which the PICA feeder was coagulated, and the OS was ligated.

Following surgery, the patient developed a pseudomeningocele, which did not resolve with spinal drainage and required placement of a right frontal ventriculoperitoneal shunt. Post-procedural angiography confirmed complete obliteration of the OS DAVF, and a follow-up MRI demonstrated a reduction in cervical cord congestion [Figures 13 and 14]. However, at the 1-year follow-up, the patient showed only minimal clinical improvement and remained significantly disabled.

DISCUSSION

Anatomy of the OS

The OS is the smallest of the dural venous sinuses, located along the midline of the occipital bone and positioned between the layers of the dura mater within the falx cerebelli. The OS typically originates near the torcular Herophili, which serves as a confluence point for major venous structures, including the superior sagittal sinus, straight sinus, and transverse sinuses.[3,16] From the torcular Herophili, the OS descends within the attached edge of the falx cerebelli toward the foramen magnum, eventually draining into either the marginal sinus, vertebral venous plexus, or sigmoid sinus.[11,14]

The OS demonstrates significant anatomical variability, both in its size and in its drainage pathways. It is frequently

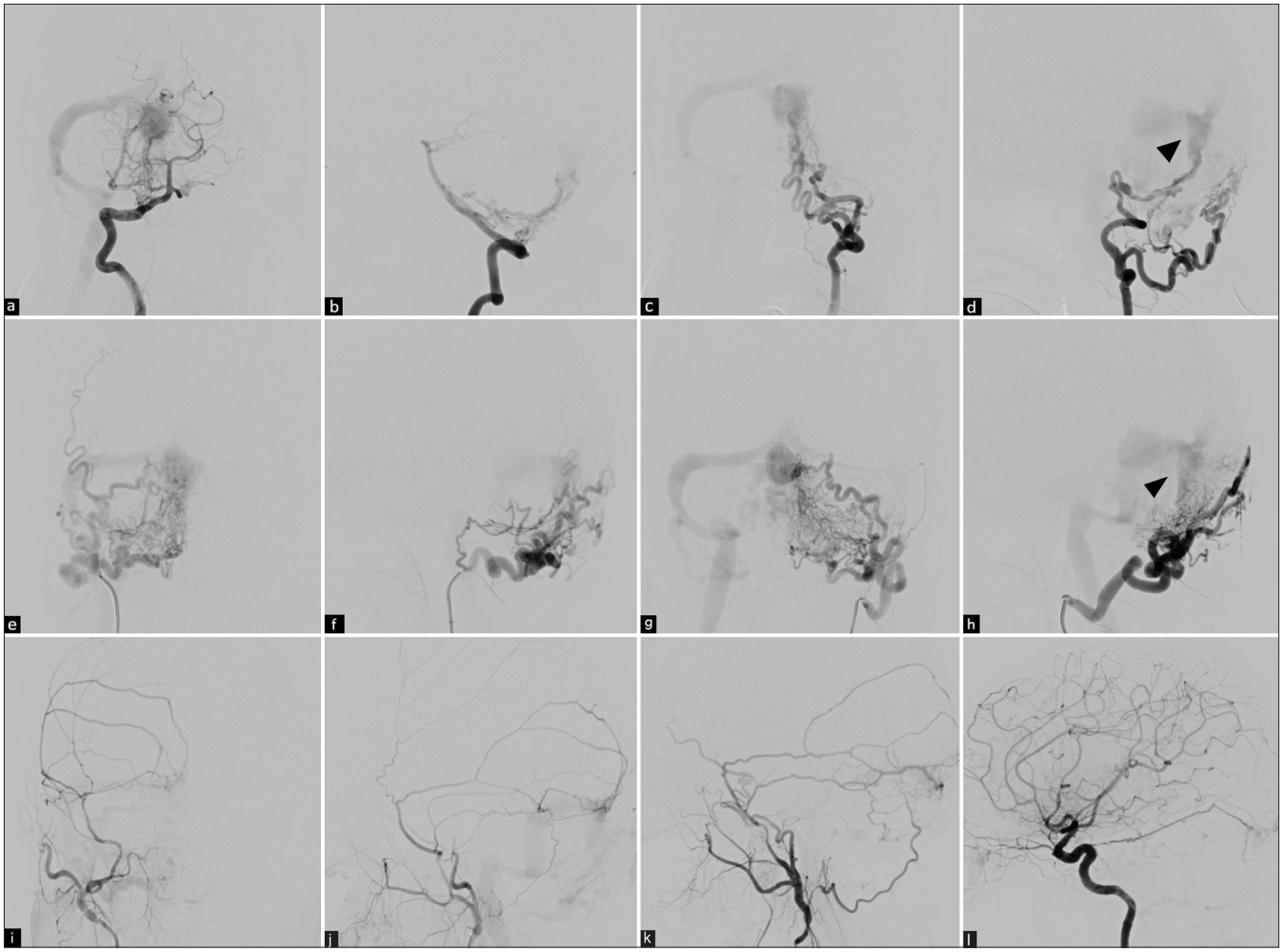

Figure 4: Cerebral angiography demonstrating multiple arterial injections to reveal the dural arteriovenous fistula at the occipital sinus. (a) Anteroposterior (AP) and (b) lateral views of the right vertebral artery (VA) injection, (c) AP and (d) lateral views of the left VA injection, (e) AP and (f) lateral views of the right occipital artery (OA) injection, (g) AP and (h) lateral views of the left OA injection, (i) AP and (j) lateral views of the right external carotid artery (ECA) injection, and lateral view of the left (k) ECA and (l) internal carotid artery injection reveal a dural arteriovenous fistula at the occipital sinus. Numerous branches, including bilateral occipital arteries, middle meningeal arteries, a dural branch of posterior inferior cerebellar arteries, and the left meningohypophyseal trunk, feed this fistula. Arrowheads in (d and f) indicate the occipital sinus.

hypoplastic or absent in adults, as it typically regresses after infancy. However, in cases where the OS remains patent into adulthood, it may serve as an alternative venous drainage route, particularly if the transverse or sigmoid sinuses are hypoplastic or obstructed.[14] Hyperplastic OS variants have been reported, with studies indicating that these enlarged OS structures can alter the venous architecture of surrounding structures, such as reducing the incidence of torcular Herophili fenestrations and influencing transverse sinus symmetry.[19]

From an embryological perspective, the OS is derived from the posterior dural venous plexus during fetal development. This plexus contributes to the formation of the transverse and sigmoid sinuses, with the OS developing as a median channel between them. Over time, the OS often regresses as other venous pathways, such as the transverse and sigmoid sinuses, become the primary drainage routes. However, in cases of developmental or post-traumatic venous anomalies, the OS may persist, playing a compensatory role in cranial venous drainage.[14,16]

Due to its deep midline location and connections to both intracranial and spinal venous structures, the OS is clinically significant in certain pathologies, such as DAVFs and other venous malformations. Persistent or hyperplastic OSs can complicate surgical approaches in the posterior cranial fossa, where venous bleeding risks are heightened. Moreover, the OS is occasionally involved in conditions such as venous hypertension and dural AVFs, where its drainage patterns

into spinal or marginal sinuses may contribute to atypical presentations, including spinal cord venous congestion and progressive myelopathy.[2,18]

Literature review

Our literature review compiled demographic data (i.e., gender and age), clinical presentation, history of trauma, Cognard classification, arterial supply and venous drainage of the fistulas, presence of venous aneurysms, associated venous sinus occlusions, treatment approach, and neurological outcomes for patients with OS DAVFs. This review included a total of eight cases, with our series contributing two cases [Table 1].[2,5,10,15,17,18]

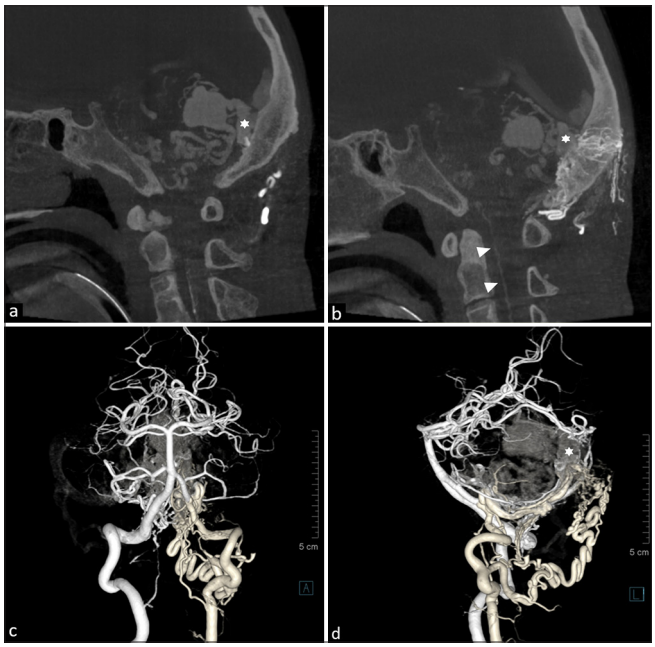

Figure 5: (a and b) Sagittal maximum intensity projection-reformatted images of angiographic computed tomography of the left vertebral artery (VA), (c) coronal, and (d) sagittal views of 3D reconstruction images fused from the right and left VAs clearly demonstrate the occipital sinus dural arteriovenous fistula draining into a large venous varix, cerebellar veins, and the anterior spinal vein. Asterisks in (a, b, and d) indicate the occipital sinus, and arrowheads in (b) indicate the anterior spinal vein.

Of the patients reviewed, 3 (37.5%) were male and 5 (62.5%) were female, with a median age of 52.5 years (range: 15–81 years). Presenting symptoms included hemorrhage in 2 cases (25%) and non-hemorrhagic symptoms in 6 cases (75%), such as tinnitus, nausea and vomiting, progressive myelopathy, headache, and/or cerebellar symptoms. Only our 2 cases (25%) had a documented history of trauma.

In terms of Cognard classification, one case was type I, two cases were type IIa+b, two cases were type II, and three cases were type V, indicating that 7 cases (87.5%) were classified as malignant types with cortical venous reflux. Arterial supply was most commonly bilateral, sourced from dural branches of the MMA, OA, PMA, ascending pharyngeal artery, and/or PICA. Venous varices were observed in 4 cases (50%), and venous sinus occlusion of either the OS or transverse sinus was reported in 4 cases (50%).

Treatment outcomes varied: 3 cases (37.5%) achieved successful occlusion with TVE, 2 cases (25%) with TAE, 2 cases (25%) with a combination of TAE and TVE, and 1 case (12.5%) with surgery alone. Neurological outcomes indicated good recovery in 5 cases (62.5%), incomplete recovery in 2 cases (25%), and poor outcome in 1 case (12.5%).

Pathogenesis of DAVF at the OS

The pathogenesis of DAVFs, particularly those involving the OS, remains complex and not fully understood. A primary factor contributing to DAVF development is venous hypertension, often resulting from cerebral venous thrombosis or sinus occlusion. This increased venous pressure can lead to the formation of abnormal arteriovenous shunts between meningeal arteries and nearby venous structures, facilitating DAVF development.[6,12] Animal studies reinforce the role of venous hypertension in DAVF

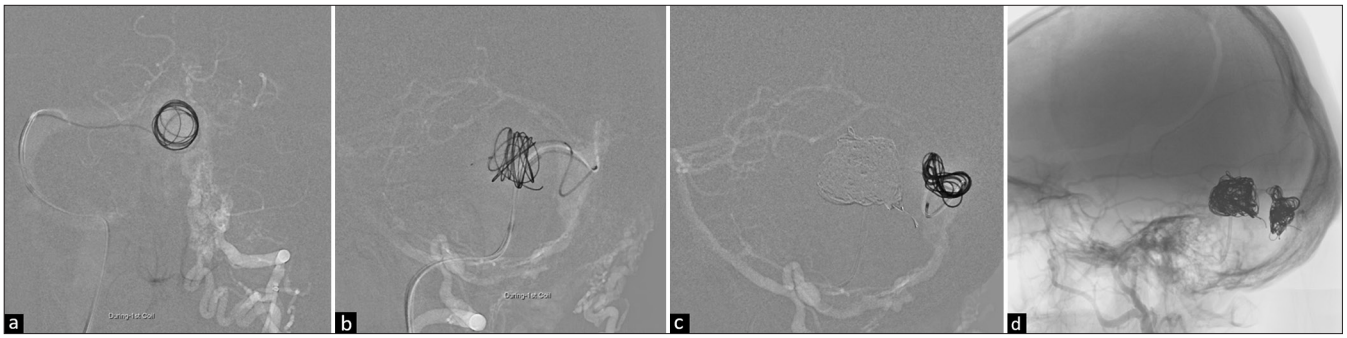

Figure 6: (a and b) Fibered coil placement within the large venous pouch under road mapping guidance. (c) Coil deployment in the occipital sinus. (d) The lateral view of an unsubtracted image shows dense coiling within the venous pouch and occipital sinus.

Table 1: Literature review of dural arteriovenous fistula involving the occipital sinus.

Authors/ Year	Gender/ Age	Symptoms and signs	Trauma history	Cognard type	Arterial feeders	Venous drainage/ aneurysm	Sinus occlusion	Treatment	Outcome
Takagi *et al*., 2012[17]	F/51	Progressive pulsating tinnitus	N/A	I	MMA (R, L), OA (R, L)	TS (R, L)/ No	No	TVE (coils)	GR
Gupta *et al*., 2013[5]	F/35	Tinnitus on the right side since childhood. Sudden severe headache (SAH).	No	IV	OA (R, L), APA (R, L), MMA (R, L), PMA (R, L)	TS (R), CVR/Yes	OS, TS (L)	TAE (particles) TVE (coils)	GR
Tanaka *et al*., 2017[18]	M/64	Progressive gait disturbance, bladder dysfunction, venous congestion from the medulla to the upper cervical cord	N/A	V	PMA (R, L)	ASV/No	No	TAE (Onyx)	IR
Moinuddin *et al*., 2017[15]	F/81	Tinnitus and vertigo. After 2 months, she developed vomiting and became lethargic. Bilateral thalamic edema.	N/A	IIa+b	OA (R, L)	SS, CVR/ No	N/A	TAE TVE	IR
Kawauchi *et al*., 2019[10]	F/62	Headache and nausea/vomiting. Cerebellar hemorrhage.	No	IIa+b	OA (R, L), PMA (R, L)	TS (R), IHV (L), CRV/Yes	OS, TS (L)	TVE (coils)	GR
Agrawal *et al*., 2021[2]	F/15	Nausea and vomiting for 2 weeks. Hydrocephalus. Her mother had a history of hydrocephalus and brain AVM.	No	IV	N/A	Galen vein, CRV/Yes	N/A	TAE (Onyx)	GR
Current study, Case 1	M/39	A 3-year history of progressive headaches, worsening over the past two weeks with severe headache, nausea, and vomiting.	Yes	V	OA (R, L), APA (R, L), MMA (R, L), PMA (R, L), PICA (R, L)	TS (R), CRV, ASV/Yes	OS	TVE (coils)	GR
Case 2	M/54	Progressive quadriparesis, BBD.	Yes	V	PICA (L)	ASV, PSV/No	OS	Surgery	PR

APA: Ascending pharyngeal artery, ASV: Anterior spinal vein, BBD: Bowel and bladder dysfunction, CVR: Cortical venous reflux, F: Female, GR: Good recovery, IHV: Inferior hemispheric vein, IR: Incomplete recovery, L: Left, M: Male, MMA: Middle meningeal artery, N/A: Data not available, NBCA: N-butyl-2-cyanoacrylate, OA: Occipital artery, OS: Occipital sinus, PICA: Posterior inferior cerebellar artery, PMA: Posterior meningeal artery, PR: Poor result, PSV: Posterior spinal vein, R: Right, SAH: Subarachnoid hemorrhage, SS: Straight sinus, TAE: Transarterial embolization, TS: Transverse sinus, TVE: Transvenous embolization

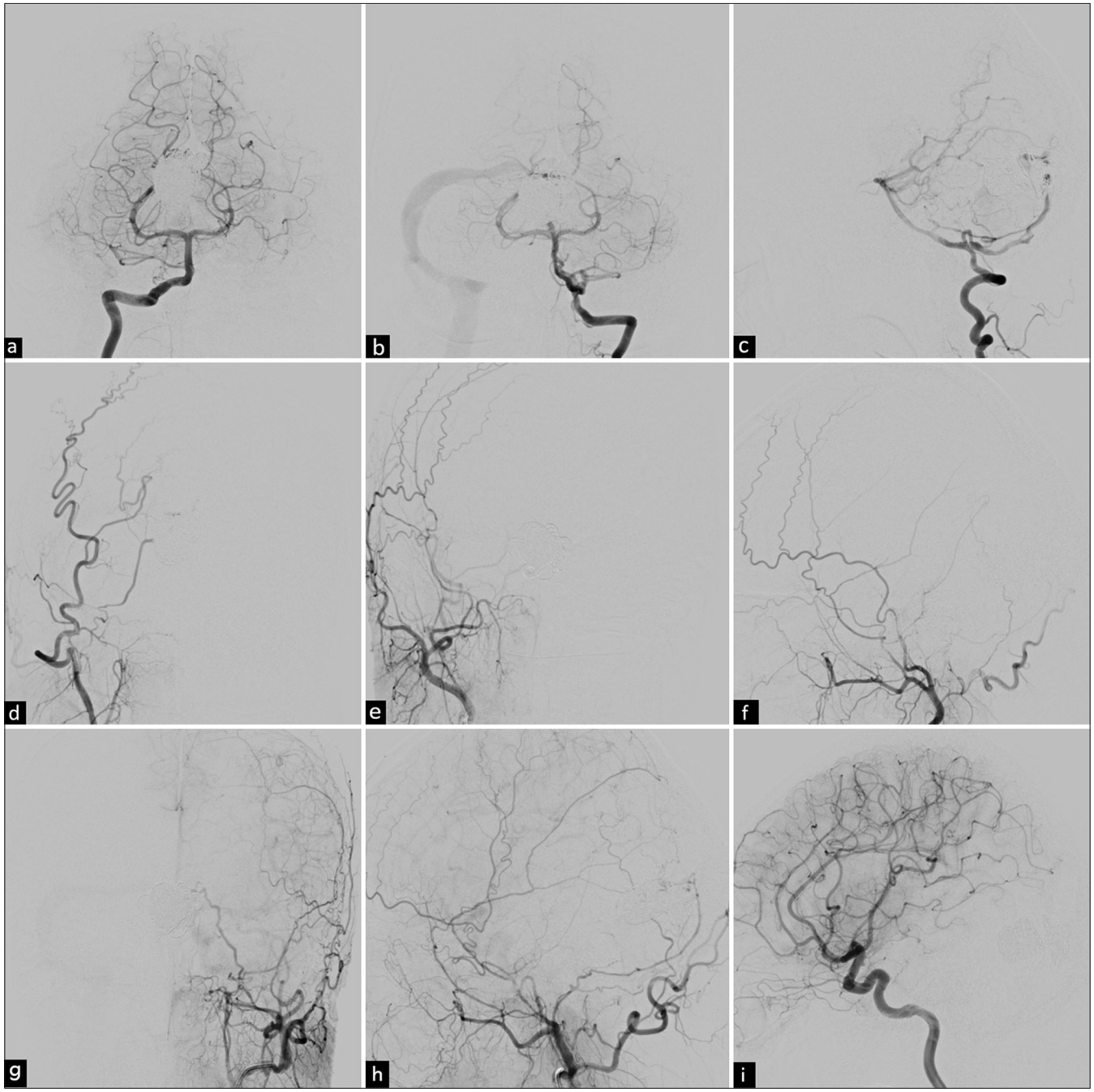

Figure 7: Cerebral angiography obtained 3 years after embolization. Anteroposterior (AP) views of the (a) right and (b) left vertebral artery (VA), (c) lateral view of the left VA, AP view of the (d) right occipital artery and (e) external carotid artery (ECA), (f) lateral view of the right ECA, (g) AP view of the left ECA, and lateral view of the left (h) ECA and (i) internal carotid artery injections confirm complete obliteration of the fistula.

pathogenesis, suggesting that elevated sinus pressures can directly stimulate angiogenesis by reducing local cerebral perfusion. This creates ischemic conditions that promote abnormal vascular connections.[13]

In the case presented by Gupta *et al*.,[5] the patient's long-standing right-sided tinnitus since childhood suggests a potential congenital origin for the DAVF at the OS. Persistent tinnitus from a young age may indicate an underlying vascular anomaly, such as a congenital DAVF, potentially formed during fetal development due to abnormal arteriovenous connections. This DAVF likely remained asymptomatic until changes in venous dynamics led to progressive symptoms,

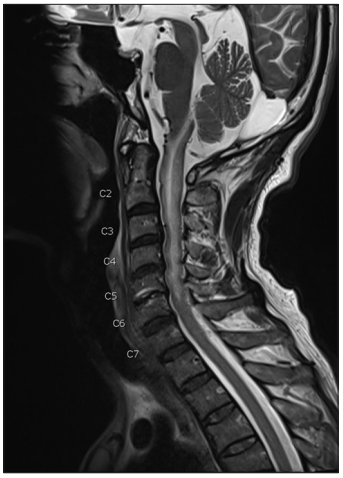

Figure 8: Sagittal T2-weighted image of the cervical spine shows an abnormal hyperintense T2 signal, indicative of venous congestion extending from the medulla to the C6 level.

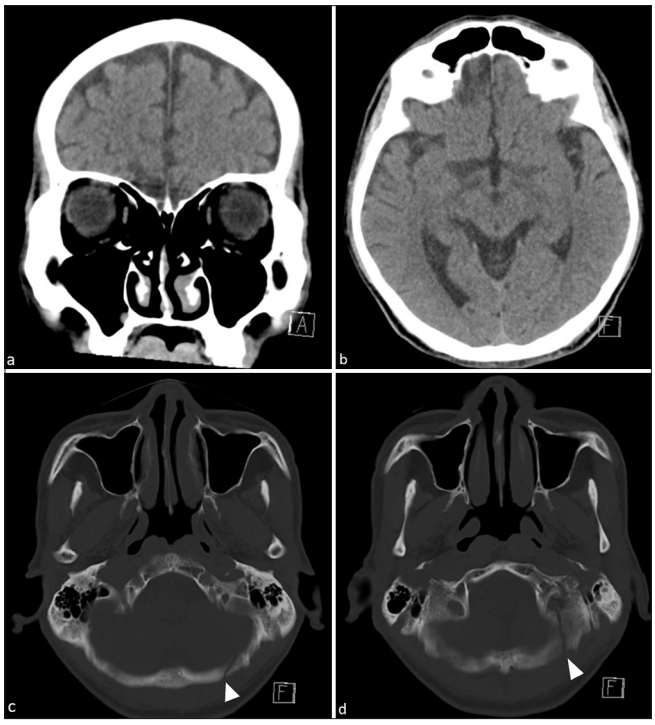

Figure 9: (a) Coronal and (b) axial non-contrast computed tomography (CT) scans of the brain show encephalomalacic changes in the bilateral anterior and inferior frontal lobes. (c and d) Axial bone-window CT scans demonstrate a linear fracture (arrowheads) at the left occipital calvarium involving the left condylar canal.

including cortical venous reflux and cerebellar venous ectasia. Similarly, the case by Agrawal *et al*.[2] supports a genetic component to DAVFs, as the patient's family history includes a mother with hydrocephalus and a brain arteriovenous malformation (AVM). Although DAVFs are typically regarded as sporadic or acquired, a family history of cerebrovascular malformations like AVMs suggests a possible genetic predisposition, which may contribute to abnormal arteriovenous connections.

Trauma is also implicated in the formation of DAVFs, as venous sinus injury can provoke inflammation, thrombosis, and neovascularization, leading to abnormal arteriovenous connections. In addition, hypercoagulable states or inflammatory conditions, such as sinus thrombosis or prolonged contraceptive use, may increase the risk of DAVFs by promoting venous stasis and vessel wall damage, which can induce DAVF formation over time.[1,4,7]

The relationship between DAVFs and prior cranial trauma is well-recognized, with trauma serving as a potential trigger for their development. In the two cases presented, the history of remote cranial trauma emphasizes its role in the pathogenesis of OS DAVFs. However, the exact mechanisms of injury remain uncertain due to the extended time interval since the trauma and the lack of detailed records from initial treatments at other hospitals.

In the first case, the patient sustained a severe head injury 12 years prior, necessitating a left frontal craniotomy. While there was no documentation of skull fractures or bleeding near the OS or foramen magnum, it is plausible that the injury resulted in increased intracranial pressure (ICP), which could have induced sinus thrombosis. Thrombosis of the OS may lead to venous hypertension, a known factor in DAVF formation. Although specific causal evidence is unavailable, the chronic progression of symptoms and delayed presentation suggest a long-standing pathological process potentially initiated by the initial trauma.

In the second case, the patient experienced a high fall 10 years before symptom onset. Although no detailed records of head injury were available, imaging at admission revealed an old cerebral contusion in the bilateral frontal lobes and a linear skull fracture in the posterior fossa. These findings indicate significant mechanical disruption during the trauma, likely affecting dural venous structures. The subsequent spinal venous congestion observed in this case underscores the possibility of trauma-induced venous hypertension and thrombosis, leading to DAVF development over time.

Severe head injuries, particularly those involving skull fractures or dural venous sinus involvement, are known to disrupt normal venous drainage pathways. Increased ICP following trauma can impair venous outflow, creating conditions conducive to sinus thrombosis. Thrombosis, in turn, may lead to venous hypertension, triggering angiogenic responses that result in the formation of abnormal arteriovenous shunts. Although direct evidence of these

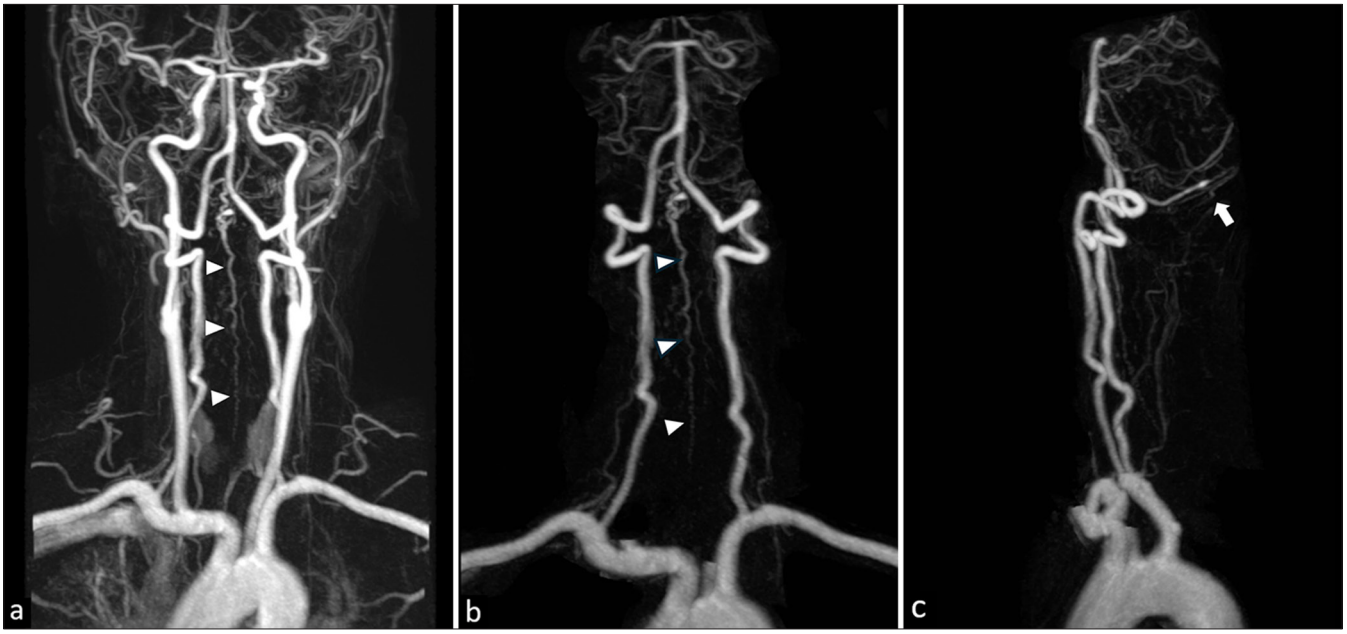

Figure 10: (a and b) Coronal and (c) sagittal maximum intensity projection images from contrast-enhanced magnetic resonance angiography demonstrate a dural arteriovenous fistula of the occipital sinus with downward drainage into dilated spinal veins. The arrow in (c) indicates the fistulous point, and arrowheads in (a) and (b) indicate the anterior spinal vein.

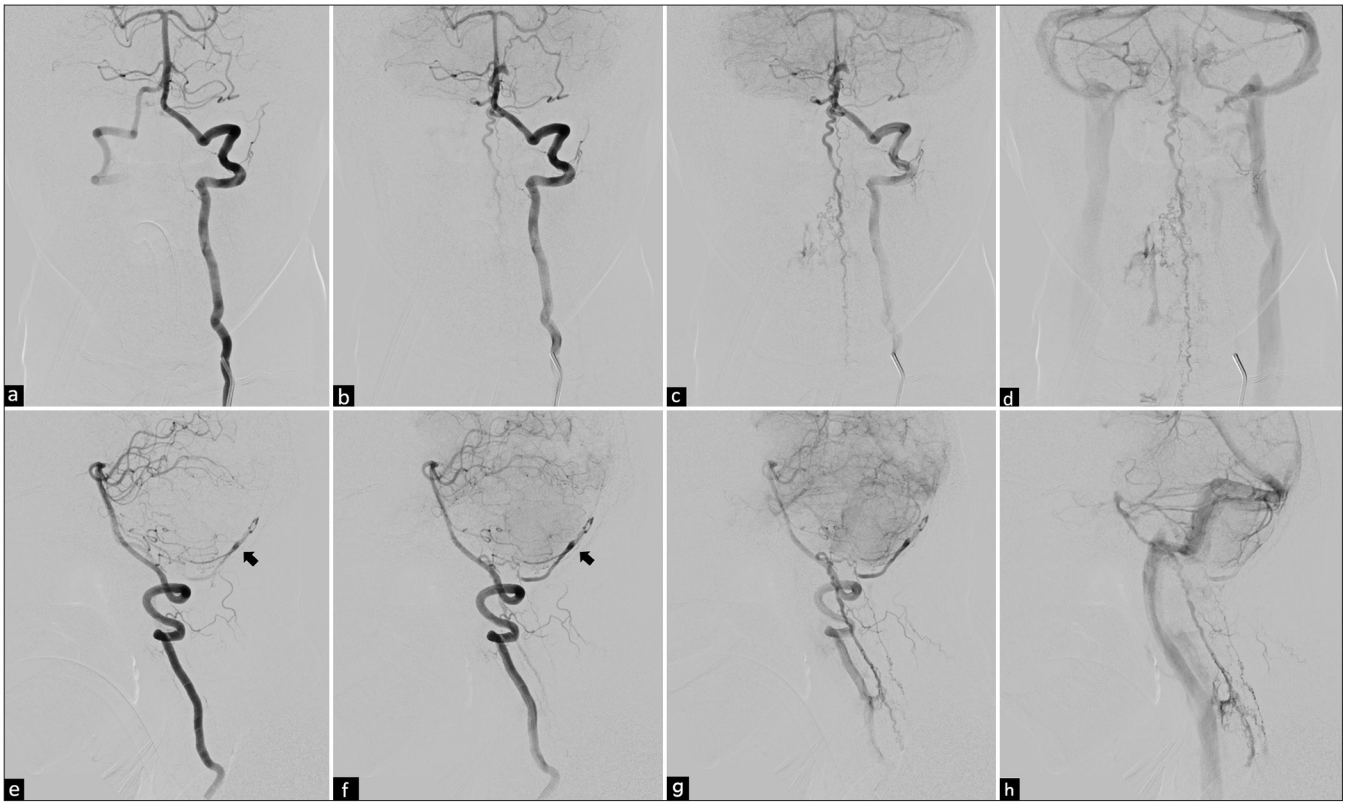

Figure 11: (a-d) Anteroposterior and (e-h) lateral views of the left vertebral artery injection from arterial to late venous phases reveal an occipital sinus dural arteriovenous fistula fed by the meningeal branch of the left posterior inferior cerebellar artery, with drainage into the anterior and posterior spinal veins. Arrows in (e and f) indicate the fistulous point.

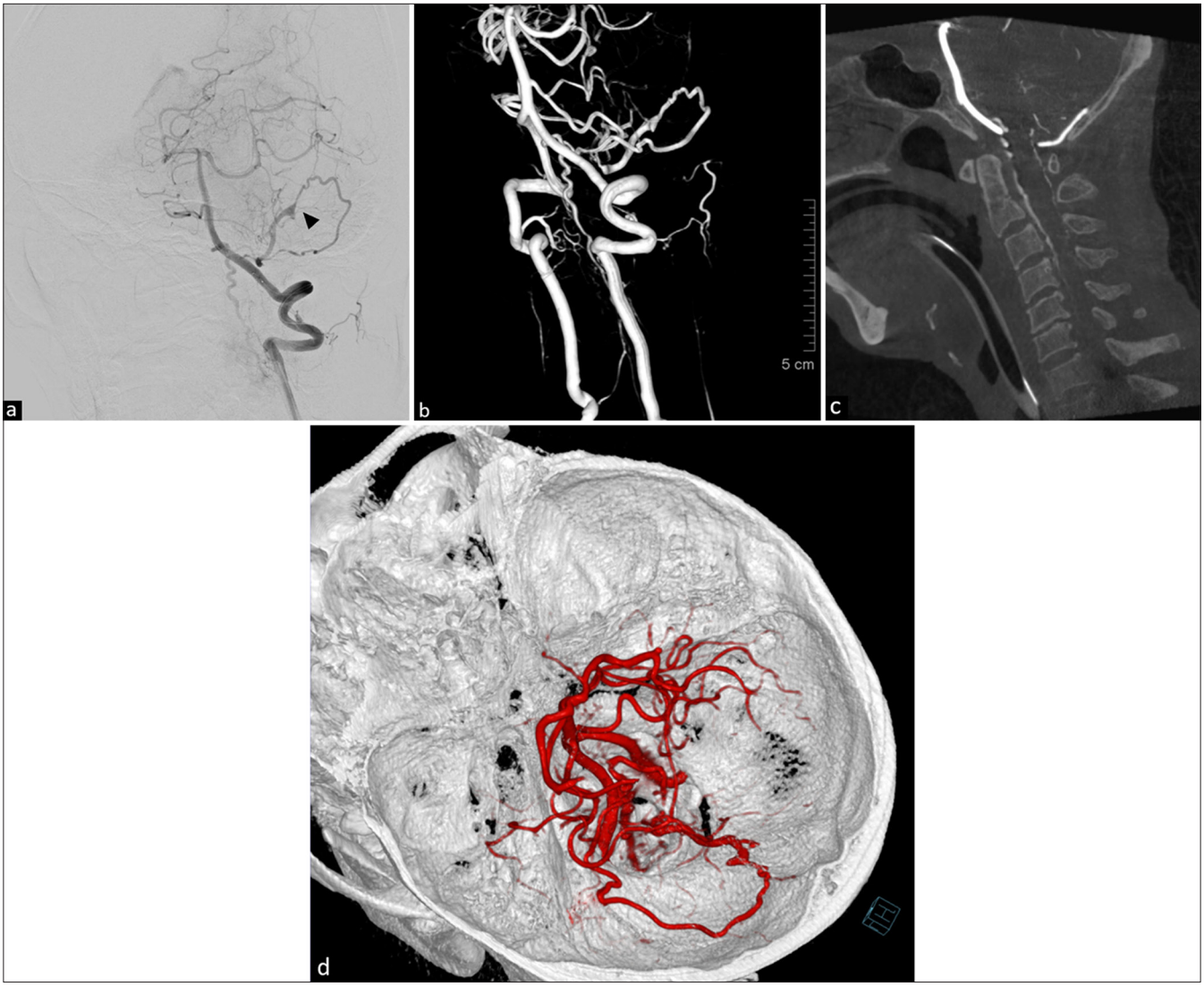

Figure 12: Oblique views of (a) the left vertebral artery injection and (b) a 3D-reconstruction image clearly demonstrate the occipital sinus dural arteriovenous fistula (arrowhead) fed by the meningeal branch of the left posterior inferior cerebellar artery. (c) Sagittal maximum intensity projection-reformatted image of angiographic computed tomography of the left vertebral artery clearly illustrates the occipital sinus, along with the anterior and posterior spinal veins. (d) The top view of a 3D reconstruction image, including body structures, also shows the fistula at the occipital sinus.

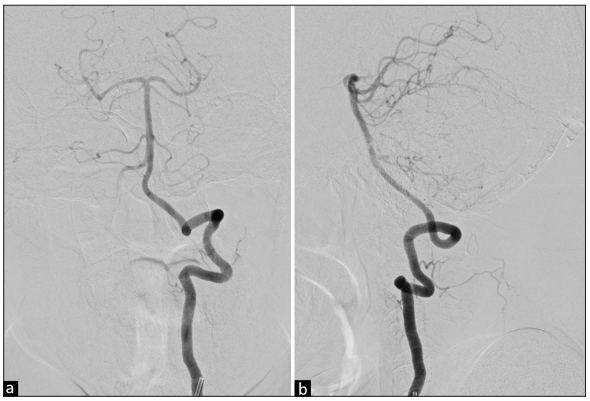

Figure 13: Cerebral angiography obtained 2 weeks post-surgery. (a) Anteroposterior and (b) lateral views of the left vertebral artery confirm complete obliteration of the fistula.

mechanisms is absent in our cases, the presence of remote cranial trauma and the anatomical location of the DAVFs align with these well-documented processes.

Management strategy

The management of DAVFs involving the OS requires a tailored approach that considers the unique anatomical and hemodynamic characteristics of each case. The primary goal is to eliminate abnormal arteriovenous shunting and relieve symptoms, particularly in high-risk DAVFs with cortical venous reflux or spinal venous drainage, which can lead to neurological deterioration.[2,5,10,15,17,18]

TVE is often the first-line treatment for OS DAVFs due to its minimally invasive nature and effectiveness in occluding

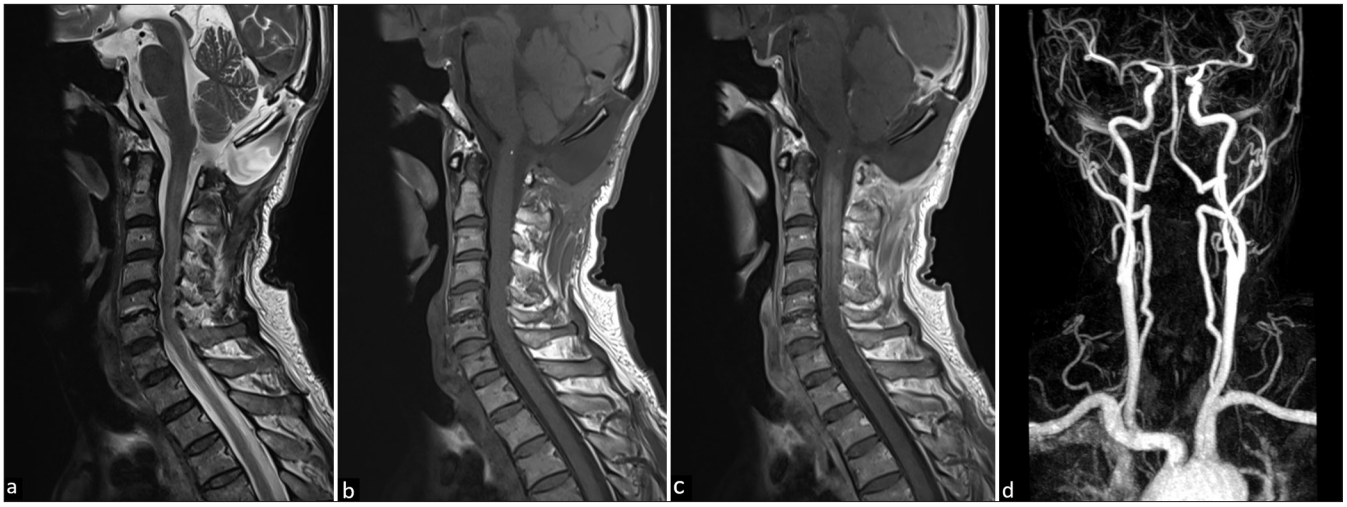

Figure 14: Magnetic resonance imaging obtained 2 months post-surgery. Sagittal images show (a) T2-weighted, (b) T1-weighted, and (c) contrast-enhanced T1-weighted sequences, revealing a significant reduction in cervical venous congestion, with residual patchy enhancement extending from C2 to C5. (d) Contrast-enhanced magnetic resonance angiography of the brain and cervical spine confirms the absence of fistula recurrence.

the venous side of the fistula. In TVE, access is typically gained through the transverse or sigmoid sinus, and coils or liquid embolic agents are deployed within the fistula to prevent blood flow. This approach is particularly successful for DAVFs with accessible venous drainage into a prominent varix or sinus.[1,3] However, TAE may also be attempted when arterial feeders are accessible, with embolic agents injected to block abnormal arteriovenous connections. TAE is often limited by small, tortuous feeders, which can make navigation challenging.[2,18]

Surgical ligation and resection may be required in cases where endovascular techniques are either unsuccessful or inaccessible, such as when arterial feeders are too small or tortuous for catheterization. Craniotomy and direct ligation of the OS can be effective in these cases, especially when high-flow DAVFs with extensive venous reflux pose a significant risk of hemorrhage or progressive myelopathy. Surgery is also a viable option for DAVFs that recur after embolization or when complications, such as hematoma, necessitate decompression.[7]

A combination of endovascular and surgical techniques is often necessary in complex cases, particularly those with multiple arterial feeders from branches of the occipital, middle meningeal, or PICAs. In such cases, an initial attempt with TAE or TVE may reduce fistula size, followed by surgical intervention to obliterate the DAVF. This combined approach minimizes the risk of incomplete treatment and optimizes patient outcomes.[5,15]

Post-procedural imaging with MRI, MRA, and cerebral angiography is essential for confirming DAVF obliteration and monitoring for recurrence. Patients with high-risk DAVFs, particularly those with cortical or spinal venous drainage, require regular follow-up due to the potential for residual or recurrent fistulas. Long-term neurological assessment is also critical to evaluate for any residual deficits or late-onset symptoms.

In summary, the management of OS DAVFs should be individualized, utilizing a combination of endovascular and surgical strategies tailored to each case's specific vascular anatomy and risk profile. Early intervention and close monitoring are crucial to prevent complications and ensure favorable outcomes.

Furthermore, while trauma and congenital factors are explored as potential contributors to OS DAVF pathogenesis, the lack of genetic and developmental data leaves questions about their roles unanswered. Future multicenter studies with standardized protocols and larger cohorts would help to address these limitations and provide more comprehensive insights into OS DAVFs.

CONCLUSION

OS DAVFs are rare and complex vascular malformations requiring careful consideration of both congenital and acquired factors, such as trauma, in their pathogenesis. The history of remote cranial trauma in both cases highlights the potential role of trauma-induced venous hypertension and thrombosis in DAVF development. While the exact mechanisms remain speculative due to limited historical data, the chronic nature of these lesions underscores the importance of recognizing trauma as a contributing factor, especially in patients with delayed symptoms. Early identification is critical, particularly in cases involving cortical or spinal venous drainage, to prevent severe neurological complications. Management demands a multidisciplinary

approach, with TVE as the preferred first-line treatment, supplemented by surgery when necessary. Regular imaging and clinical follow-up are vital for detecting recurrence or progression. Individualized treatment strategies tailored to each patient's anatomy and clinical profile are essential to improving outcomes and quality of life.

Ethical approval

The Institutional Review Board has waived the ethical approval for this study.

Declaration of patient consent

Patients' consent not required as patients' identities were not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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