Cognard Type V Dural Arteriovenous Fistula: A Case Report

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DISCLOSURES
None.

CONSENT
Yes.

HUMAN AND ANIMAL RIGHTS
None

ABSTRACT

Dural arteriovenous fistula with perimedullary venous drainage (Cognard type V) is a rare intracranial vascular lesion that may cause progressive myelopathy due to venous hypertension and is frequently misdiagnosed because of nonspecific clinical and magnetic resonance imaging (MRI) findings. We report an adult patient presenting with progressive neurological deficits at the cervicomedullary junction. MRI showed diffuse T2-Weighted hyperintensity and swelling without diffusion restriction; subtle perimedullary flow voids were initially overlooked. The patient received corticosteroids for presumed myelitis, with subsequent deterioration. Digital subtraction angiography revealed a dural arteriovenous fistula supplied by intracranial dural branches with perimedullary venous drainage. Owing to unfavorable anatomy for endovascular therapy, microsurgical disconnection was performed. Therefore, early recognition and angiographic confirmation are essential to avoid delayed diagnosis and irreversible neurological injury.

CASE REPORT

BACKGROUND

Dural arteriovenous fistula accounts for approximately 10–15% of intracranial vascular malformations [1], with a peak incidence between 50 and 60 years of age and an equal gender distribution [2]. The etiology of this type of cerebrovascular malformation is primarily idiopathic. However, a history of cranial surgery, traumatic brain injury, infection, and dural venous sinus thrombosis have been suggested as associated risk factors.

Morphological classification of dural arteriovenous fistula is of great clinical importance. Several classification systems have been proposed by different authors, among which the Cognard classification is most widely applied in clinical practice due to its strong correlation with prognosis and therapeutic strategy. This system categorizes dAVFs into five types based on venous drainage patterns. Among these, Cognard type V dural arteriovenous fistula, characterized by drainage into the spinal perimedullary venous system, is extremely rare, accounting for approximately 5.9% of all dural arteriovenous fistulas [3]. Clinical presentation is highly variable and often overlaps with other conditions (such as inflammatory or infectious diseases) in both clinical and imaging features, requiring meticulous evaluation. Digital subtraction angiography (DSA) remains the gold standard for diagnosis [4]; however, it is an invasive procedure and is typically performed only when this diagnosis is suspected. Therefore, recognition of suggestive signs on MRI is crucial to avoid diagnostic delay, as early detection and accurate diagnosis directly affect treatment outcomes and prognosis. The primary goal of treatment is complete obliteration of the arteriovenous shunt, which may be achieved by endovascular intervention, surgery, or stereotactic radiosurgery [5].

We report a clinical case of a 62-year-old male patient admitted with dizziness, nausea, sphincter dysfunction, and progressive weakness of both lower extremities. The patient was diagnosed with a dural arteriovenous fistula with perimedullary venous drainage at the cervical level (Cognard type V) and had previously undergone multiple courses of conservative medical treatment without improvement. Neurological deficits progressed to quadriplegia, and the patient developed acute respiratory failure that was life-threatening. Emergency microsurgical intervention was performed to occlude the fistula using vascular clips, in combination with intensive medical treatment and rehabilitation, resulting in gradual clinical recovery. Through this case, we emphasize the importance of early diagnosis and the pivotal role of definitive treatment modalities in patients with dural arteriovenous fistula with perimedullary venous drainage.

CASE REPORT

A 62-year-old male patient with no significant past medical history was admitted to the Stroke Center with complaints of dizziness and unsteady gait. On clinical examination, the patient was alert, afebrile, with a Glasgow Coma Scale score of 15. Pupillary light reflex was positive. No focal neurological deficits, sensory disturbances, or sphincter dysfunction were noted at admission.

Laboratory evaluation, including complete blood count and serum biochemistry, was within normal limits (glucose: 6.0 mmol/L, urea: 5.2 mmol/L, high-sensitivity CRP: 0.3 mg/L). Non-contrast MRI of the brain and cervical spine demonstrated enlargement and hyperintensity of the medulla oblongata and upper cervical spinal cord at the C1–C2 level on T2-weighted, FLAIR, and STIR images, without definite diffusion restriction on DWI/ADC (Figure 1).

Cerebrospinal fluid (CSF) analysis revealed a small number of red blood cells (0.002 T/L) and white blood cells (0.006 G/L), with mildly elevated protein (0.54 g/L). Multiplex real-time PCR of CSF was positive for Enterovirus and negative for Mycobacterium tuberculosis. Gram stain and culture of CSF were negative, excluding bacterial and fungal infections.

Serologic tests to detect specific autoantibodies, including anti-dsDNA, anticardiolipin IgM/IgG, anti-β2 glycoprotein IgM/IgG, and cANCA/pANCA were within normal range. Autoimmune encephalitis antibodies (NMDAR, LGI1, AMPAR1/2, GABAB, DPPX, CASPR2) were not detected in CSF.

Based on these findings, the initial diagnosis by radiologists and neurologists was Enterovirus-induced medullary and upper cervical myelitis. The patient was treated with intravenous methylprednisolone (Solu-Medrol) 1 g daily for 5 days and received prophylaxis for deep vein thrombosis. After four days of treatment, the patient developed progressive bilateral lower extremity weakness with muscle strength decreasing to 1/5, accompanied by sphincter dysfunction, without sensory impairment. MR spectroscopy of the brain lesion demonstrated a lipid/lactate peak without increased choline metabolism, excluding neoplastic pathology. Contrast-enhanced MRI of the cervical spine showed no increase in lesion size compared with prior imaging, with subtle enhancement on T1-weighted post-contrast images. Additionally, dilated, tortuous vascular structures were noted along the anterior surface of the medulla and upper cervical spinal cord, raising suspicion of a spinal vascular malformation (Figure 2). The patient was subsequently referred for cerebral DSA for further evaluation and possible intervention.

DSA of bilateral internal and external carotid arteries and vertebral arteries revealed a dural arteriovenous fistula supplied by bilateral meningohypophyseal branches of the internal carotid arteries, with a fistulous point at the right paraclival region and drainage into the anterior spinal venous system, consistent with Cognard type V (Figure 2).

Endovascular access was attempted via the posterior-inferior branch of the right meningohypophyseal trunk using a 1.5Fr Marathon microcatheter and a 0.008” Hybrid guidewire. Navigation was technically challenging due to the small caliber and acute angulation of the vessel. Balloon protection of the right internal carotid artery using a Hyperglide balloon was attempted; however, retrograde flow into intracranial branches persisted (Figure 3). Given the high risk of embolic material migration into intracranial circulation, the procedure was terminated without embolization. After multidisciplinary discussion, conservative medical management and rehabilitation were selected.

After two weeks of conservative treatment, neurological status worsened with progression to quadriparesis (upper extremity strength 3/5, lower extremity strength 1/5). The patient acutely developed dyspnea, cyanosis, low-grade fever, hypotension (70/40mmHg), bilateral pulmonary crackles, and oxygen saturation of 89%. He was transferred to the Emergency Center and diagnosed with acute respiratory failure due to pneumonia, with suspected septic shock, quadriplegia, and medullary–cervical spinal cord injury secondary to dural arteriovenous fistula (Cognard type V). The patient required sedation, mechanical ventilation, vasopressor support, and high-dose broad-spectrum antibiotics.

Laboratory tests showed leukocytosis (23.61 G/L), neutrophilia (81.9%), and elevated procalcitonin (0.31 ng/mL). Arterial blood gas analysis revealed metabolic alkalosis with elevated pH (7.496), increased standard bicarbonate (28.8 mmol/L), and hypoxemia (pO_2 58.8 mmHg). Sputum and bronchial lavage cultures were positive for Klebsiella aerogenes (+++), while fungal and tuberculosis tests were negative. CSF examination remained negative for bacterial and fungal infection. Chest X-ray and non-contrast chest CT demonstrated consolidation and atelectasis predominantly involving the right

upper lobe. Follow-up non-contrast cervical spine MRI showed progression of medullary and cervical spinal cord edema extending to the C5 level on T2-weighted and STIR images (Figure 4).

Emergency microsurgical intervention was performed, exposing the right paramedian clival fistula and clipping the fistulous connection (Figure 5), combined with intensive medical treatment for pneumonia. Postoperatively, upper limb strength improved to 4/5, lower limb strength 1/5, respiratory failure resolved, and SpO_2 99%. Follow-up CTA and DSA showed no residual malformation. Non-contrast cervical spine MRI showed reduction of edema and no more tortuous dilated vessels anterior to the medulla (Figure 6). The patient was transferred to rehabilitation.

DISCUSSION

Cognard type V dural arteriovenous fistula (dAVF) with perimedullary venous drainage represents a particularly aggressive subtype characterized by direct shunting of arterial blood from dural branches - most commonly meningeal, occipital, or vertebral arteries - into the spinal perimedullary venous system, bypassing the normal capillary network to drain into intracranial venous sinuses [6]. These direct arteriovenous connections lead to progressive venous dilatation and elevated spinal venous pressure, resulting in decreased spinal cord perfusion secondary to a reduced arteriovenous pressure gradient and direct compression of arterial feeders by the engorged venous system [6]. These pathophysiological changes impair microcirculatory perfusion, culminating in spinal cord edema, intramedullary hemorrhage and ischemia, and ultimately progressive myelopathy. Clinically, the disease course is most often subacute or chronic, manifesting as gradually progressive quadriparesis, sensory disturbances, and sphincter dysfunction. However, acute deterioration may occur in the setting of venous thrombosis, intramedullary hemorrhage, or acute ischemic spinal cord injury [7].

First-line diagnostic modalities are usually CT and MRI. CT initially helps exclude conditions such as stroke or brain tumors that may produce clinical symptoms similar to dural arteriovenous fistula. MRI has advantages in detecting spinal cord - brainstem edema caused by perimedullary venous congestion, demonstrated as diffuse spinal cord hyperintensity not following an arterial vascular territory. The Flow Void sign on sagittal T2-Weighted images and the appearance of tortuous dilated vessels on contrast-enhanced MRI may serve as reliable indicators of vascular malformations [8].

Returning to our clinical case, unfortunately, the flow void sign on sagittal T2-Weighted images was overlooked on the initial cervical spine MRI. According to Haryu S et al., this sign is detected in only approximately 36.6% of cases [9]. Meanwhile, on contrast-enhanced images, perimedullary vessels may be more easily visualized, with a detection rate of about 76%[10]. Detection of the flow void sign is not always straightforward, and initial misdiagnosis is common [11-13], due to both subjective and objective factors. Perimedullary veins, particularly the anterior spinal veins, are small-caliber vessels that run tortuously along the pia mater and lie within the anterior median fissure - a relatively deep groove on the anterior surface of the spinal cord - thus on MRI, they may be mistaken for nerve roots, normal vessels, cerebrospinal fluid flow, or motion artifacts. Other unfavorable factors include indirect imaging features of cranial dural arteriovenous fistula with perimedullary venous drainage, such as spinal cord hyperintensity similar to more common conditions (transverse myelitis, ADEM, NMOSD…) (Table 1), together with initially vague clinical manifestations and CSF biochemical-PCR findings suggestive of viral infection, which further complicate diagnostic orientation. These misinterpretations led to corticosteroid therapy in our patient. Notably, corticosteroid treatment may worsen clinical symptoms, as previously reported. According to Nasr DM et al., up to 53.8% of patients receiving intravenous methylprednisolone experienced acute symptom deterioration after steroid administration [14]. One proposed mechanism is that high-dose corticosteroids contribute to volume expansion and venous congestion, increase venous pressure, reduce the arteriovenous pressure gradient, and aggravate spinal cord hypoxia and edema[15]. Therefore, although extremely rare, neurologists should consider this disease as a differential diagnosis, especially in cases that do not respond or worsen after corticosteroid therapy [16].

Respiratory failure symptoms in Cognard type V dural arteriovenous fistula are rare and, to date, have mostly been reported in isolated case reports [11,17,18]. Although there is limited literature on the exact mechanism, we propose the following anatomical - physiological explanations: first, the medulla oblongata contains the dorsal respiratory group - responsible for basic inspiratory rhythm generation, the ventral respiratory group - playing a crucial role in forced expiration regulation, and the nucleus of the vagus nerve - an important mediator in maintaining rhythmic respiratory center activity and the coordination between inspiration and expiration. Second, the high cervical spinal cord at levels C3-C5 contains the motor neurons of the phrenic nerve, which innervates the diaphragm - the primary muscle of respiration. For these reasons, venous hypertension - induced injury in the medulla and upper cervical cord leads to edema, dysfunction, and reduced perfusion of anatomical structures essential for respiratory rhythm and neuromuscular function, resulting in bradypnea, irregular breathing, reduced tidal volume, and predisposing to infection, pneumonia, and respiratory failure.

Based on the aforementioned pathophysiological mechanisms, the most important therapeutic goal in Cognard type V dural arteriovenous fistula is complete elimination of the arteriovenous shunt. Various definitive treatment modalities have been previously described, including endovascular intervention, surgery, and radiosurgery, whereas conservative/ medical therapy is only supportive. Endovascular intervention

is considered the standard and first-line treatment, aiming to occlude the fistulous tract via arterial or venous routes. The choice of approach depends on each specific case. Transarterial microcatheter access with embolization using agents such as Onyx (EVOH) is often considered first-line therapy [19], particularly suitable for malformations with 1–2 main arterial feeders when the microcatheter can reach close to the fistulous point. Limitations include difficulty accessing tortuous and sharply angled feeding pedicles, with the risk of reflux into intracranial arterial branches or recurrence due to incomplete embolization. An unsuccessful transarterial intervention was reported by Aiuxut Lorenzo S et al., in which collateral feeders developed after the first embolization, leading to venous congestion and deterioration of spinal venous drainage with recurrence of symptoms [5]. A transvenous approach to the fistulous point may be considered in such cases; however, several limitations exist. Perimedullary veins are often difficult to access, and venous sinuses are frequently stenosed or occluded due to the underlying pathological process. Therefore, although transvenous embolization has shown promising results in selected cases, it is feasible only in a limited number of carefully selected patients.

Surgery is considered a "salvage" solution, typically indicated when endovascular intervention fails. The advantage of microsurgery is a high cure rate, with immediate effectiveness once complete disconnection of the fistula is achieved. Several recent studies evaluating surgical outcomes in high-grade dural arteriovenous fistulas have shown very favorable results. In the report by Al-Mahfoudh R et al., the rate of complete fistula disconnection reached 100%, confirmed by digital subtraction angiography, with no patients experiencing permanent neurological deficits or symptom recurrence [20]. In another report by Cheng Qiu et al., the rate of complete fistula obliteration was 85.185%, higher than that of endovascular treatment (60%) [21]. In our case, significant clinical improvement after microsurgical intervention further demonstrates the urgent role of definitive treatment modalities in general and the importance of surgery in particular, especially from an emergency perspective.

In general, all treatment methods have certain efficacy and inherent advantages and disadvantages; therefore, treatment selection should be individualized for each case, based on specific pathological conditions, patient preference, as well as the technical facilities and expertise of the treating institution.

TEACHING POINT

Cognard type V dural arteriovenous fistula should be considered in patients with unexplained progressive myelopathy and brainstem–cervical cord edema on MRI. Subtle flow void signs and mildly dilated perimedullary veins should be meticulously evaluated, and early diagnostic confirmation with angiography, followed by definitive fistula obliteration is essential to prevent irreversible neurological injury and life-threatening complications.

QUESTIONS

Question 1: Which of the following MRI findings is most suggestive of a Cognard type V dural arteriovenous fistula?

A. Diffuse spinal cord T2 hyperintensity confined to an arterial territory

B. Ring-enhancing intramedullary lesion

C. Prominent serpiginous perimedullary flow voids on sagittal T2-weighted images (applies)

D. Restricted diffusion within the medulla

E. Isolated leptomeningeal enhancement

Explanation: Cognard type V dural arteriovenous fistulas are characterized by perimedullary venous drainage. Dilated perimedullary veins appear as serpiginous flow voids on T2-weighted imaging and represent a key diagnostic clue, whereas spinal cord edema alone is nonspecific.

[The Flow Void sign on sagittal T2W images and the appearance of tortuous dilated vessels on contrast-enhanced MRI may serve as reliable indicators of vascular malformations.]

Question 2: Cognard type V dural arteriovenous fistulas are defined by which venous drainage pattern?

A. Antegrade drainage into a dural venous sinus

B. Retrograde reflux into cortical veins only

C. Drainage into the cavernous sinus

D. Perimedullary venous drainage into the spinal venous system (applies)

E. Drainage into deep cerebral veins without spinal involvement

Explanation: The defining feature of Cognard type V lesions is venous drainage into the perimedullary spinal venous system, leading to spinal venous hypertension and progressive myelopathy. [Cognard type V dural arteriovenous fistula (dAVF) with perimedullary venous drainage represents a particularly aggressive subtype characterized by direct shunting of arterial blood from dural branches—most commonly meningeal, occipital, or vertebral arteries—into the spinal perimedullary venous system.]

Question 3: Why may corticosteroid therapy worsen neurological symptoms in patients with spinal venous hypertension due to dural arteriovenous fistula?

A. It increases arterial inflow through vasodilation

B. It causes acute arterial thrombosis

C. It increases venous congestion and reduces the arteriovenous pressure gradient (applies)

D. It induces autoimmune demyelination

E. It directly damages spinal neurons

Explanation: Corticosteroids may increase intravascular volume and venous pressure, thereby worsening venous congestion and reducing the arteriovenous pressure gradient, which can aggravate spinal cord hypoxia and edema. [One proposed mechanism is that high-dose corticosteroids contribute to volume expansion and venous congestion, increase venous pressure, reduce the arteriovenous pressure gradient, and aggravate spinal cord hypoxia and edema.]

Question 4: Which feature distinguishes Cognard type V dural arteriovenous fistula from Cognard type III or IV lesions?

A. Presence of cortical venous reflux

B. Risk of intracranial hemorrhage

C. Exclusive drainage into the perimedullary venous system (applies)

D. Association with venous sinus thrombosis

E. Need for surgical treatment

Explanation: Cognard type V lesions are uniquely defined by perimedullary venous drainage into spinal veins. Types III and IV involve cortical venous reflux without spinal venous drainage. [Cognard type V dural arteriovenous fistula (dAVF) with perimedullary venous drainage represents a particularly aggressive subtype characterized by direct shunting of arterial blood … into the spinal perimedullary venous system.]

Question 5: What approximate proportion of patients demonstrated the Flow Void sign on sagittal T2-weighted MRI?

A. Less than 20%

B. 20–30%

C. 30–40% (applies)

D. 40–60%

E. More than 70%

Explanation: The Flow Void sign is not consistently detected on initial MRI evaluation. Haryu S et al. reported a relatively low detection rate, explaining why early misdiagnosis is common.

[According to Haryu S et al., this sign is detected in only approximately 36.6% of cases.]

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FIGURES

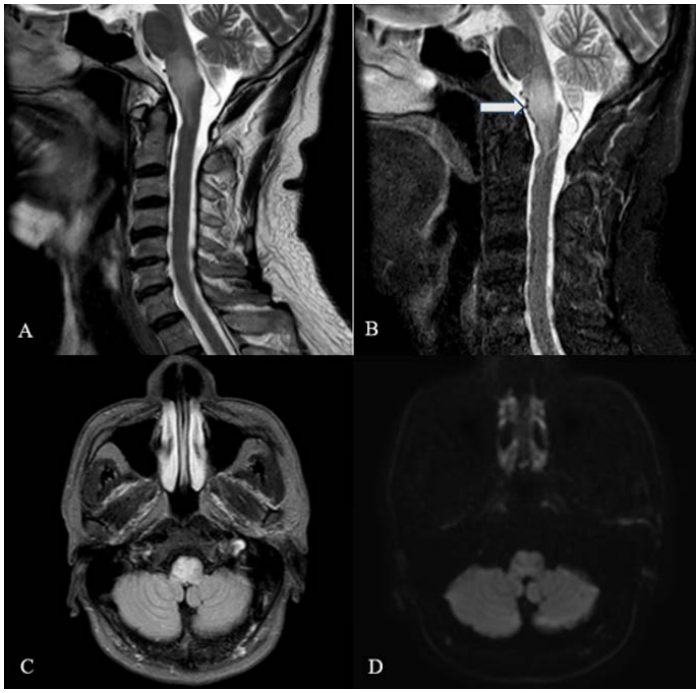

Figure 1: A 62-year-old male patient with Cognard type V dAVF: MRI findings at admission. (A), (B): Sagittal T2-Weighted (A) and STIR (B) images show a hyperintense lesion in the medulla oblongata and upper cervical spinal cord at C1-C2, with several small flow voids representing dilated vessels along the anterior surface of the medulla (large white arrows), which were initially overlooked. (C), (D): Axial FLAIR images show hyperintensity without diffusion restriction on DWI.

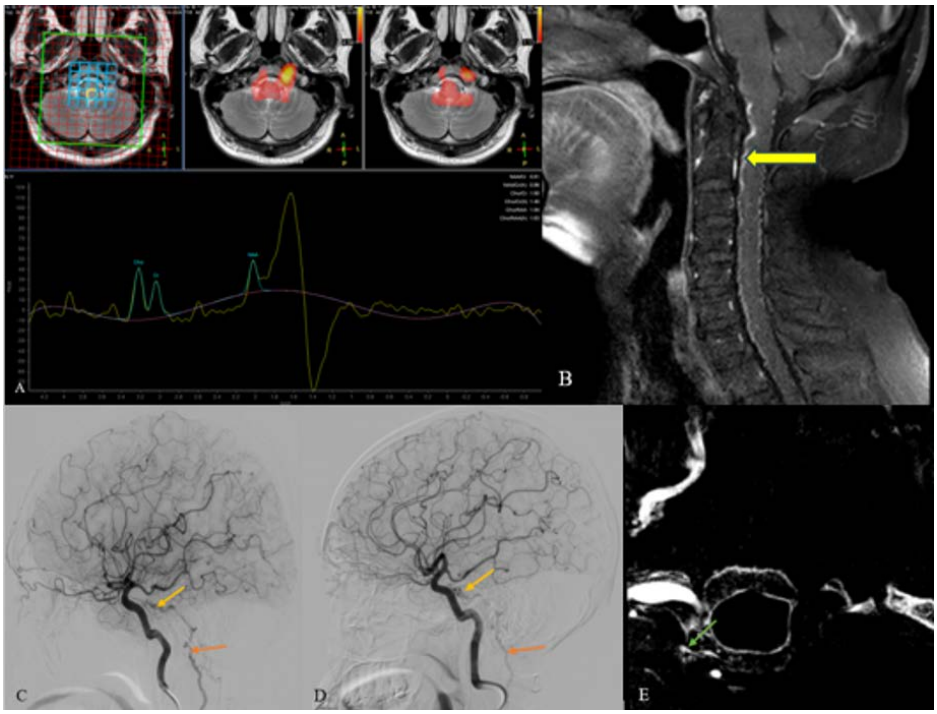

Figure 2: A 62-year-old male patient with Cognard type V dAVF: MRI and DSA findings after corticosteroid therapy. (A): MR spectroscopy showing no choline elevation, excluding tumor. (B): Sagittal T1-weighted contrast-enhanced image shows abnormal dilated tortuous vessels along the anterior surface of the medulla and upper cervical spinal cord (large yellow arrow). (C), (D): DSA of bilateral internal carotid arteries demonstrates a dural arteriovenous fistula supplied by bilateral meningeal branches of the meningohypophyseal trunk (small yellow arrows) with drainage into spinal perimedullary veins (small orange arrows). (E): Vaso-CT confirms the fistulous point adjacent to the right clivus from a meningohypophyseal branch draining into perimedullary veins (green arrow).

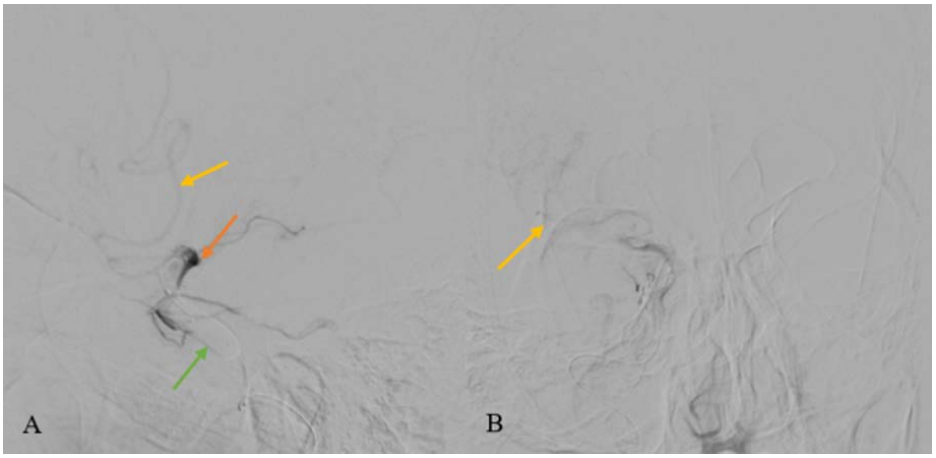

Figure 3: A 62-year-old male patient with Cognard type V dAVF: DSA images in lateral and anteroposterior projections. (A): Lateral projection shows inability of the microcatheter to reach the fistulous point due to small vessel caliber and acute angulation (small orange arrow); contrast injection reveals retrograde flow into intracranial branches (small yellow arrow) despite balloon protection (small green arrow). (B): Anteroposterior projection similarly demonstrates retrograde intracranial reflux.

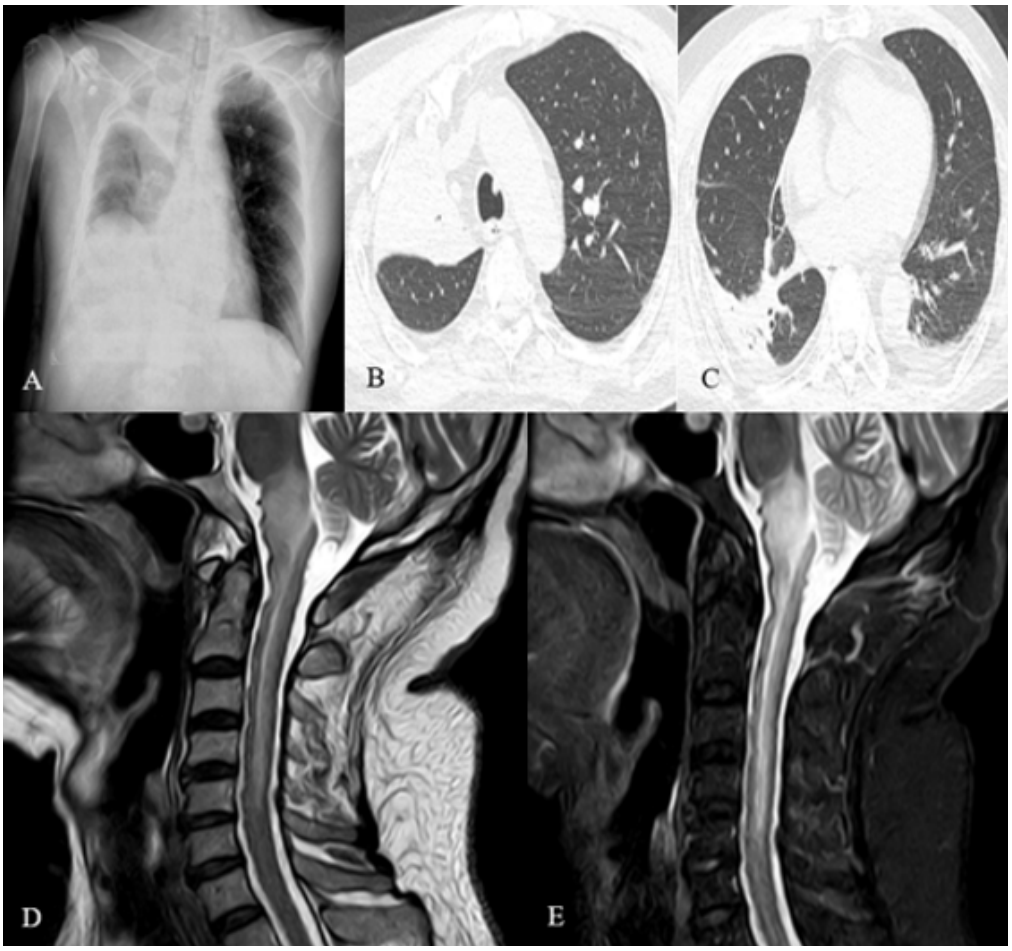

Figure 4: A 62-year-old male patient with Cognard type V dAVF: Chest imaging and follow-up MRI after the onset of respiratory failure. (A): Chest X-ray shows reduced lung volume with right upper lobe atelectasis and diffuse opacities. (B), (C): Chest CT demonstrates consolidation and atelectasis of the right lung. (D), (E): Sagittal T2-Weighted and STIR images of the cervical spine show progression of spinal cord edema extending to C5.

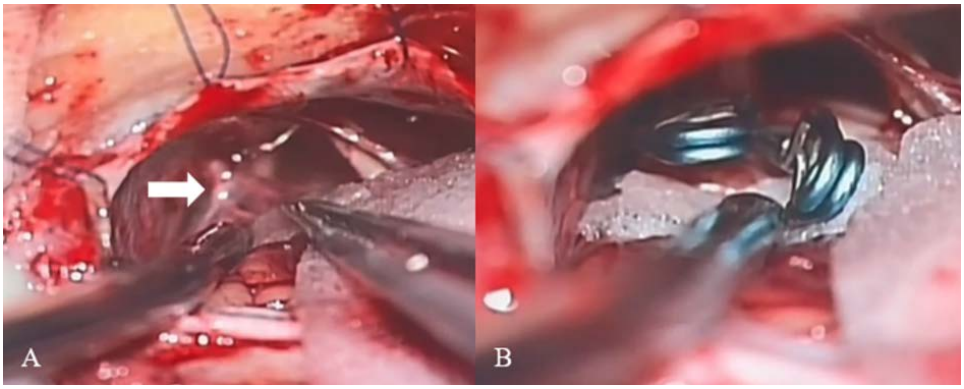

Figure 5: A 62-year-old male patient with Cognard type V dAVF: Emergency microsurgery. (A): Craniotomy exposing right paramedian clival fistula (large white arrow). (B): Obliteration with two vascular clips.

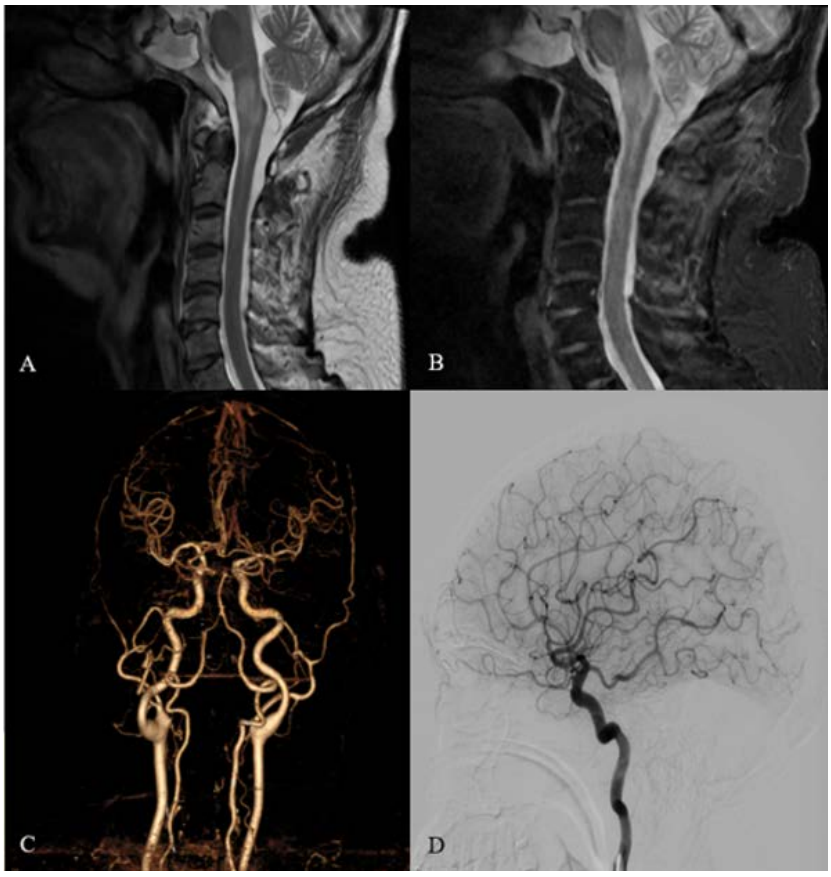

Figure 6: A 62-year-old male patient with Cognard type V dAVF: Postoperative imaging.
(A), (B): Sagittal T2-weighted and STIR images of the cervical spine demonstrate marked reduction of spinal cord edema and absence of anterior perimedullary flow voids. (C): Three-dimensional CTA showing no residual vascular malformation. (D): Follow-up DSA confirming complete obliteration of the fistula.

Table 1: Differential diagnosis of Cognard type V dural arteriovenous fistula versus other causes of longitudinally extensive spinal cord lesions on MRI

Entity	Clinical Presentation	Time Course	Cord Involvement Level	Perimedullary Dilated Veins	MRI Signal Pattern
Cognard type V DAVF	Progressive myelopathy with motor weakness, sensory deficits, and sphincter dysfunction	Subacute to chronic	Long segment	Yes	Longitudinally extensive intramedullary T2 hyperintensity with cord swelling; serpentine perimedullary flow voids
Idiopathic Acute Transverse Myelitis (ATM)	Rapidly progressive bilateral sensory and motor dysfunction with a distinct cord level	Acute	Variable; long segment (longitudinally extensive) more common than short segment	No	Occupying at least two-thirds of cross-sectional area with central cord predominance; mild cord expansion
Infectious Myelitis	Symptoms related to specific pathogen (viral, bacterial, fungal)	Acute or chronic (e.g., HIV, TB)	Nonspecific; can be long segment (e.g., TB)	No	Nonspecific diffuse T2 hyperintense signal; usually non-expansile
Multiple Sclerosis (MS)	Multiple clinical attacks; sensory and motor deficits	Acute	Short segment (spanning one to two vertebral segments)	No	Asymmetric, peripheral, wedge-shaped or round; involves <50% cross-sectional area
Acute Disseminated Encephalomyelitis (ADEM)	History of viral infection/vaccination; encephalopathy symptoms; common in children	Acute	Long segment (longer craniocaudal extent than MS); often multiple segments	No	Large cross-sectional area, ill-defined margins, centrally located
Neuromyelitis Optica Spectrum Disorder (NMOSD)	Optic neuritis, myelitis; more common in females	Acute	Long segment (traversing at least three vertebral body levels)	No	Central gray matter predominance; may show "bright spotty lesions" (T2 hyperintense similar to CSF)
Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD)	Acute myelitis (often associated with optic neuritis); may present with back pain and urinary retention; more common in children and young adults	Acute	Long segment (often ≥ 3 vertebral body levels), frequently involving the conus medullaris	No	Central, longitudinally extensive lesions with gray matter predominance; may show “H-sign”; often associated with cord swelling
Spinal Cord Ischemia	Hyperacute onset of neurological deficits	Acute (progression in less than 4 hours)	Long segment	No	Central gray matter predominance; "H-shaped" or "butterfly-shaped" pattern; restricted diffusion

KEYWORDS

Dural arteriovenous fistula, digital subtraction angiography, Cognard type V

ABBREVIATIONS

MRI = Magnetic Resonance Imaging
CT/CTA = Computed Tomography/Computed Tomography Angiography
dAVF = Dural Arterio Venous Fistula
DSA = Digital Subtraction Angiography
CRP = C-Reactive Protein
CSF = Cerebro Spinal Fluid
PCR = Polymerase Chain Reaction
anti-dsDNA = Anti–Double-Stranded DNA Antibody
cANCA/pANCA = Cytoplasmic/Perinuclear Anti-Neutrophil Cytoplasmic Antibodies
NMDAR = Anti–N-Methyl-D-Aspartate Receptor Antibody
LGI1 = Anti–Leucine-Rich Glioma-Inactivated 1 Antibody
AMPAR1/2 = Anti–A-Amino-3-Hydroxy-5-Methyl-4-Isoxazolepropionic Acid Receptor Antibody
GABAB = Anti–Gamma-Aminobutyric Acid Type B Receptor Antibody
DPPX = Anti–Dipeptidyl-Peptidase-Like Protein 6 Antibody
CASPR2 = Anti–Contactin-Associated Protein-Like 2 Antibody
ADEM = Acute Disseminated Encephalo Myelitis
NMOSD = Neuro Myelitis Optica Spectrum Disorder
EVOH = Ethylene-Vinyl Alcohol Copolymer

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