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From Mystery to Cure: A Rare Case of Low Back Pain

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CASE REPORT

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ABSTRACT

Low back pain (LBP) is one of the most common complaints in outpatient neurology and orthopedic clinics, with a wide range of differential diagnoses. Spinal dural arteriovenous fistulas (AVFs), although rare, are a critical yet often overlooked cause of myelopathy and should be considered in atypical presentations. We report the case of a 57-year-old woman with a history of hypertension and hypothyroidism who presented with gradual-onset LBP radiating to both lower limbs. Initially attributed to degenerative spinal disease, her condition rapidly progressed over 4–5 days to include bilateral lower limb weakness, sensory disturbances, and bladder and bowel dysfunction. Neurological examination revealed hypertonia, motor power of 2/5 in both lower limbs, exaggerated deep tendon reflexes, and bilateral extensor plantar responses. Lumbar spine MRI showed only mild degenerative changes without cord compression. Given the rapid neurological decline, spinal angiography was performed and revealed a dural AVF arising from the right L4 lumbar artery. The patient underwent endovascular embolization with N-butyl cyanoacrylate (NBCA), resulting in prompt improvement of autonomic symptoms and gradual motor recovery. At follow-up, she was pain-free and neurologically intact. Lumbar dural AVFs are rare and can mimic more common degenerative spine conditions, delaying appropriate diagnosis and treatment. This case underscores the importance of considering vascular etiologies in patients with rapidly progressive myelopathy and highlights the role of spinal angiography in diagnosis. Early intervention with embolization can lead to excellent clinical outcomes.

KEYWORDS

spinal dural arteriovenous fistula, low back pain, myelopathy, lumbar avf, endovascular embolization, spinal angiography, neurological deficits

INTRODUCTION

LBP is among the most frequently encountered complaints in outpatient departments globally, especially among the elderly. According to a 2021 global study by Xu et al., the age-standardized prevalence rate of LBP in adults aged 55 years and older was 18,282.8 per 100,000 population [1]. The increasing incidence of LBP is primarily driven by population aging and growth, posing a significant burden on healthcare systems worldwide. In clinical practice, the common causes of LBP in individuals aged 50 years and above include degenerative disc disease, facet joint osteoarthritis, spinal stenosis, osteoporotic compression fractures, spondylolisthesis, sacroiliac joint dysfunction, myofascial pain syndrome, and less frequently, tumors or infections. Chronic illnesses such as ankylosing spondylitis and prior spinal surgeries also contribute to the differential diagnosis of LBP presenting in our outpatient departments. Despite the predominance of these etiologies, the underlying cause of LBP is not always one of the possibilities mentioned above. The most common cause of spinal vascular malformation is a spinal dural AVF, which is an abnormal connection between spinal arteries and veins within the spinal dura mater. The incidence of spinal dural AVF is reported to be around 5–10 cases per million people per year [2]. They are very rare and, therefore, are easily missed or underdiagnosed. An article by Krings et al. mentioned that around 80% of spinal dural AVFs are mostly located between the T6 and L2 levels, with sacral and higher cervical involvement seen in 4% and 2% of cases, respectively [3]. AVFs involving the lumbar arteries are exceptionally rare, and there are limited data on the exact incidence of lumbar artery AVFs alone. Lumbar artery AVFs are typically associated with pseudoaneurysms or develop secondary to trauma, surgical intervention, or catheter-based procedures; however, our case is an example that this may not always be true [4]. The usual presentation of a spinal dural AVF is progressive spastic paraparesis and sensory loss with sphincter dysfunction, often with radicular pain, and LBP is reported as the initial presenting symptom in around 33% of cases [5]. Here, we report a case of a middle-aged woman who initially presented with gradual-onset LBP, followed by progressive bilateral lower limb weakness and bladder and bowel dysfunction, and was ultimately diagnosed with a spinal dural AVF of the right L4 lumbar artery.

CASE DETAILS

A 57-year-old woman with a history of hypertension and hypothyroidism presented with a gradual onset of LBP radiating to both lower limbs for several days. The pain was dull, aching, and progressively

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worsening. She had no history of trauma, recent surgical procedures, or vaccinations. There was no fever, headache, or a band-like sensation in her back, and no recent history of travel was noted. She reported associated paresthesia and numbness in both feet. During her initial outpatient department visit, her neurological examination was unremarkable. She was started on neuropathic pain medication, and an MRI scan was planned. However, within 4–5 days, she returned with a new development of progressive weakness in both lower limbs, followed by urinary retention and bowel dysfunction. On repeat neurological examination, her upper limb tone was normal, but she had spasticity in both lower limbs. Motor power was reduced to 2/5 in her lower limbs but was 5/5 in her upper limbs. Deep tendon reflexes were exaggerated, and plantar responses were bilaterally extensor. A urinary catheter was inserted due to retention. Based on the clinical findings, a spinal cord pathology was evident. A clinical diagnosis of non-traumatic myelopathy was considered, and appropriate investigations were initiated. Laboratory evaluation revealed a hemoglobin of 12.6 g/dL, with normal renal, liver, and electrolyte panels. Vitamin B12 was elevated (1440 pg/mL), folate was 7.8 ng/mL, and vitamin D was 32 ng/mL. MRI of the lumbosacral spine showed mild degenerative changes and an L4–L5 disc bulge without significant thecal sac indentation. An altered signal intensity was noted in the lumbar spinal cord region on the T2 image. (Figure 1) Autoimmune markers, including Antinuclear Antibodies (ANA) and Anti-Neutrophil Cytoplasmic Antibodies (ANCA), were negative, and C-Reactive protein (CRP) was mildly elevated at 1.03 mg/dL. Based on these findings, we deduced that the cause of the myelopathy was neither metabolic nor degenerative. A demyelinating pathology was considered less likely due to the patient's age. However, in view of the rapid neurological progression and the mild cord hyperintensity on imaging, a spinal vascular malformation was suspected.

Further Investigation

Due to rapid neurological decline, without any further delay, spinal angiography was performed via femoral access. Angiography identified a spinal dural AVF originating from the right L4 lumbar artery with an enlarged intradural draining vein [Figure 2].

Therapeutic Intervention

Procedure

Procedure was done under general anesthesia. A 5 Fr short sheath was placed in the right femoral artery. A 4 Fr cobra diagnostic catheter was advanced over a guide wire. The L4 lumbar radicular artery of the right side was catheterized. Angiogram was taken on different planes. Radiculomedullary artery was accessed using a marathon microcatheter. Angiogram revealed the origin of the dural AVF. Microcatheter was advanced over the chikai 10 wire. 30% NBCA was injected slowly to occlude the fistulous point. (Post surgical images as shown in Figure 3)

Post-Procedure Care

Supportive care, physiotherapy, and oral anticoagulation with apixaban.

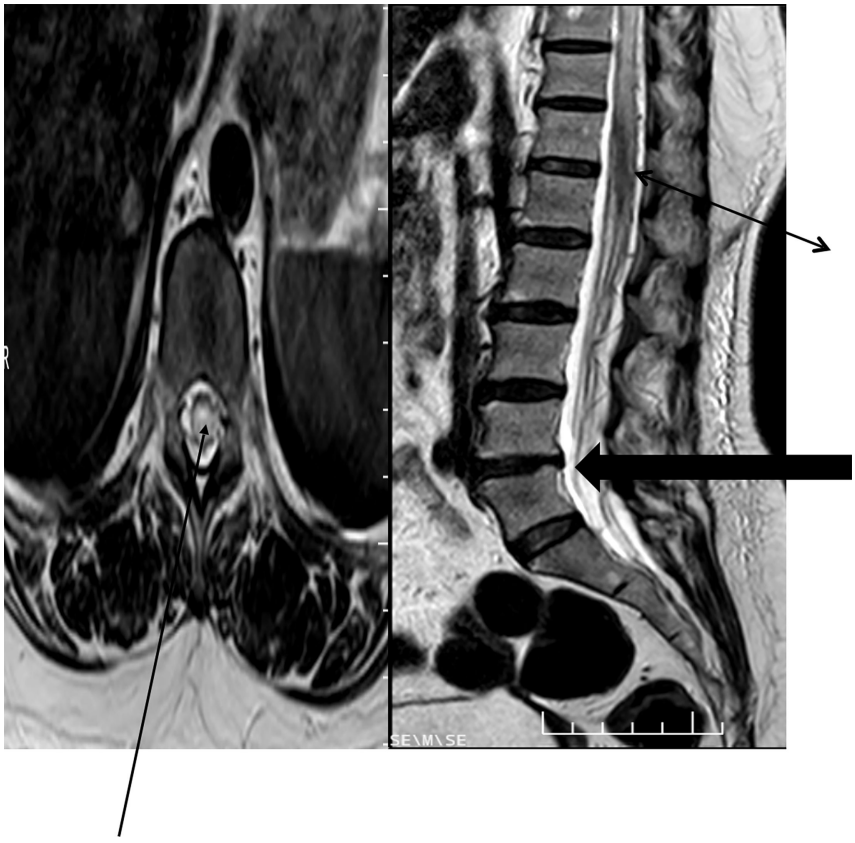

FIGURE 1. T2 sagittal and axial images of MRI lumbosacral spine showing degenerative changes with L4–L5 prolapsed intervertebral disc (marked with broad black arrow) and hyperintensity in the lumbar cord region (marked with narrow black arrow) with flow voids (marked with double-headed black arrow).

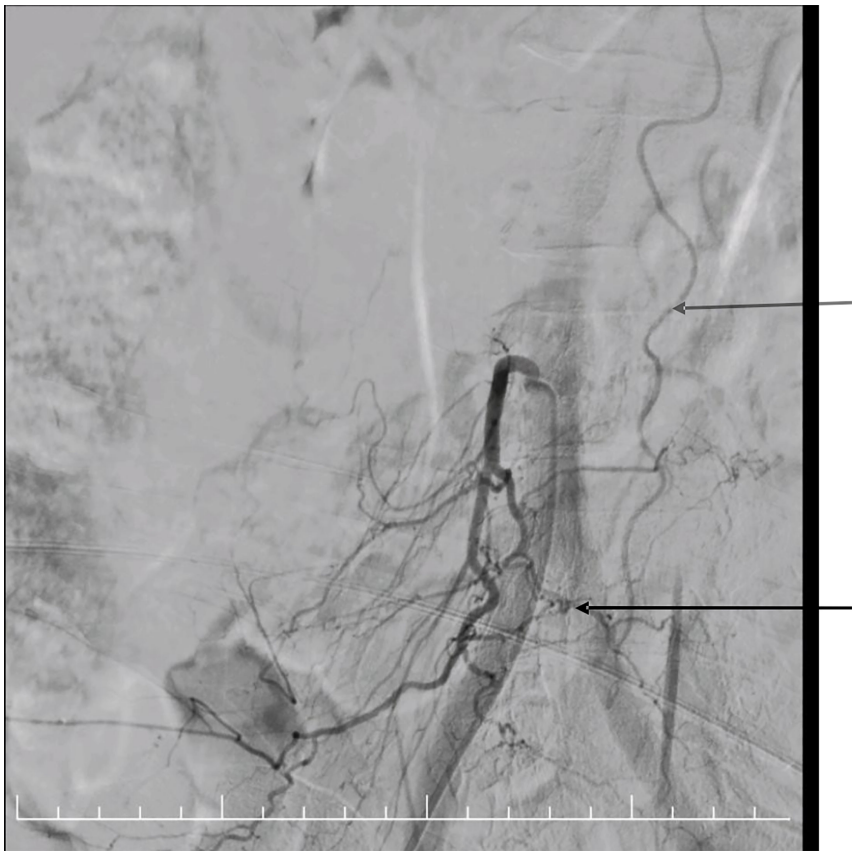

FIGURE 2. Spinal angiography image showing right L4 lumbar artery dural arteriovenous (marked in black arrow) fistula with enlarged vein inside the spinal cord (marked with grey arrow)

Follow-up and Outcomes

Short-Term
Immediate improvement in bladder and bowel function; regained motor strength.

Two-Week Follow-Up
Ambulatory without assistance; no residual neurological deficits; no pain.

Long-Term Plan
Continued outpatient monitoring and rehabilitation as needed.

Patient Perspective
The patient expressed gratitude for the rapid diagnosis after several days of uncertainty and was pleased with the quick improvement in mobility and independence. She emphasized the importance of not ignoring worsening symptoms.

DISCUSSION
Myelopathy can arise from a wide spectrum of etiologies, including infectious, traumatic, autoimmune, metabolic, neoplastic, hereditary-degenerative, and vascular causes. Clinical evaluation, combined with detailed neurological examination, targeted imaging, and cerebrospinal fluid (CSF) analysis, often helps narrow down the differential diagnosis. Among these, vascular malformations, though rare, can present as rapidly progressive myelopathy and pose a diagnostic challenge, especially in the absence of significant findings on routine imaging. In our case, the patient was ultimately diagnosed with a spinal dural AVF originating from the right L4 lumbar artery, a rare entity both in location and presentation.

Understanding the regional vascular anatomy is essential for recognizing and managing such conditions. The lumbar arteries typically consist of four paired vessels that arise from the posterolateral aspect of the abdominal aorta at vertebral levels L1–L4. These arteries supply the vertebral bodies, paraspinal muscles, dura, and nerve roots, and form an extensive network of anastomoses with adjacent segmental vessels. Each lumbar artery runs laterally behind the psoas major muscle and divides into anterior and posterior central branches. It also gives off a spinal branch that runs through the intervertebral foramen and then divides into a radiculomeningeal artery (which supplies the dura mater and nerve roots) and a radiculomedullary artery (which may not be present at every level). The radiculomedullary artery follows the nerve root to the spinal cord surface. These radiculomedullary arteries reinforce the longitudinal spinal arteries: one anterior spinal artery and two posterior spinal arteries. The most important of these feeders is the artery of Adamkiewicz, which usually arises from a left posterior branch of a lumbar artery, most often between T9 and L2 [6,7].

The venous drainage of the spinal cord is equally complex. The intrinsic veins of the spinal cord include the anterior and posterior spinal veins, which run along the cord surface, and the radicular veins, which drain into veins accompanying the nerve roots. The venous drainage of the spinal cord consists of an internal and an external venous plexus. The internal vertebral venous

plexus, also known as the epidural venous plexus, is located inside the spinal canal within the epidural space. It receives blood from the spinal veins via the radicular veins and communicates freely between the anterior and posterior plexuses. The external vertebral venous plexus lies outside the vertebrae, surrounding the spine both anteriorly and posteriorly. It communicates with the internal plexus via intervertebral veins. This anatomical configuration allows for bidirectional flow and may predispose to venous congestion when pathological arteriovenous communications occur. Blood from the lumbar veins flows into the inferior vena cava via the radicular veins and the internal and external venous plexuses. Some lumbar veins ascend and communicate with the right azygos and left hemiazygos veins, providing a link to the superior vena cava (SVC). They also communicate with the iliolumbar and sacral veins [8,9].

Spinal vascular malformations are classified into several types: dural AVFs, intramedullary arteriovenous malformations (AVMs), and cavernous malformations. Among these, dural AVFs are the most common, accounting for approximately 70% of spinal vascular lesions [10,11]. These lesions typically involve an abnormal communication between a radiculomeningeal artery and a radicular vein on the dural surface. The pathological retrograde drainage into perimedullary veins leads to venous hypertension, cord edema, reduced perfusion, and ultimately ischemia of the spinal cord. This is believed to be the main mechanism underlying the neurological deficits observed in these patients [8,9].

Clinically, spinal dural AVFs often present with progressive, stepwise neurological deterioration, including sensory disturbances, motor weakness, and sphincter dysfunction. However, a minority of cases, like ours, may present acutely or with stroke-like symptoms. Thus, one needs to have a high clinical suspicion for spinal vascular malformations. Whenever a patient presents to us with above-mentioned symptoms and the cause cannot be attributed to a specific pathology, one needs to think about underlying vascular pathology. Routine MRI may reveal nonspecific findings or even appear normal. Certain MRI findings like T2 hyperintensity, spinal cord edema or swelling, and serpentine flow voids (which actually represent dilated perimedullary veins) on the dorsal cord surface in the absence of any other pathology should raise a high index of suspicion and without any further delay, evaluation with spinal angiography should be performed, which remains the diagnostic gold standard. Thus, we need to keep vascular pathology as a differential and go ahead with angiography once clinical suspicion arises.

Our patient presented with LBP and rapidly progressing lower limb weakness over a few days, accompanied by bladder and bowel involvement. Her initial imaging showed nonspecific T2 hyperintensity and flow voids in the lumbar region. Without delay, a spinal angiography was performed, which revealed a dural AVF arising from the right L4 lumbar artery. Our case is unique because it is one of the first reported cases of a lumbar fistula arising from the right L4 lumbar artery. Most spinal dural AVFs are reported on the left side. The reason for the left-sided predominance is the higher prevalence of left-sided radiculomedullary arteries, especially the artery of Adamkiewicz, which is found in 70–80% of people. These arteries have a higher flow capacity and larger caliber than their right-sided counterparts, making them more likely to develop abnormal arteriovenous shunts. Additionally, an embryological persistent left-sided dominance in the thoracolumbar region creates more potential sites for fistula formation [12].

Lumbar artery AVFs are exceptionally rare. Most documented cases are secondary to trauma or iatrogenic injuries, such as those occurring during spinal surgery or vascular interventions. For instance, Le Viet Dung et al. described a case of lumbar artery pseudoaneurysm and AVF in a patient with prior spinal trauma and surgery. Similarly, Maleux reported a case associated with laparoscopic splenectomy. In contrast, our patient had no history of trauma or prior surgical interventions, suggesting a possible developmental etiology [13,14]

For treating spinal dural AVFs, we have two options: endovascular treatment and open surgical intervention. As endovascular intervention is less invasive, it is the preferred option over

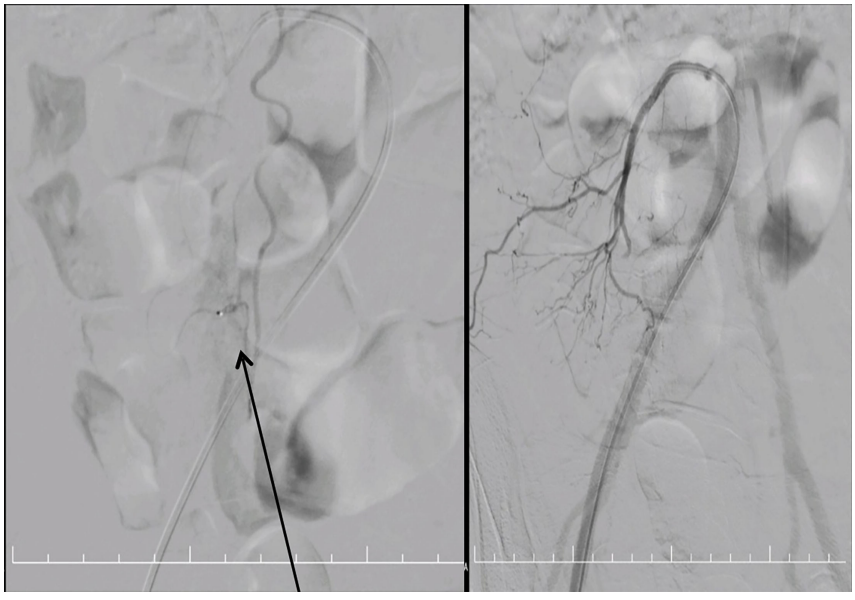

FIGURE 3. Microcatheter inserted during spinal angiography exactly localizing the arteriovenous fistula (marked with black arrow) with spinal angiography image after embolization of the spinal arteriovenous fistula with 30% N-butyl cyanoacrylate and no venous backflow/enlarged vein visible.

open surgery. The main goal of endovascular intervention is to completely block the abnormal connection between the artery and vein, which can be achieved using various embolic materials. At present, we have the following embolisation materials which can be used:

- **Liquid Embolics:** Materials like NBCA glue or Onyx. These are frequently used. NBCA polymerizes on contact with blood, acting like a glue, while Onyx is a liquid polymer that can be delivered slowly for greater precision. These liquid agents have shown to achieve a more complete and permanent blockage of the fistula.
- **Coils:** Detachable coils can be deployed to create a mechanical obstruction within the feeding artery, which can be sometimes used in combination with liquid embolics.
- **Stent Grafts:** For certain larger fistulas, a stent graft (a tube-like device with a fabric covering) can be placed to cover the fistula and redirect blood flow through the artery, effectively sealing the abnormal connection.

Talking about liquid embolic agents, we have a new agent Squid, which is similar in composition to Onyx. It is a non-adhesive, precipitating liquid agent composed of an ethylene vinyl alcohol (EVOH) copolymer dissolved in dimethyl sulfoxide (DMSO) with suspended tantalum powder for visibility under fluoroscopy. Earlier, we had Squid 18, but now we have Squid 12, which comes in a less viscous formulation and allows better penetration into smaller, more intricate vessels. This can be used in lumbar artery fistula treatment. One can guide the microcatheter to navigate through the arterial system to the feeding vessel of the fistula. Once the catheter tip is positioned as close to the fistula as possible, Squid is slowly injected. The DMSO solvent diffuses away into the bloodstream, causing the EVOH copolymer to precipitate and solidify, creating a solid cast that fills and occludes the fistula, effectively cutting off the abnormal blood flow. This "plug-and-push" technique allows the physician to control the embolization process [15,16].

We also have Scepter, which is a mini balloon catheter that is designed for temporary occlusion of blood flow and for the controlled delivery of embolic materials (like liquid glues or polymers) during procedures to treat conditions such as AVMs and dural arteriovenous fistulas (dAVFs). Its small size and balloon allow it to be navigated to distal, or more difficult to reach, target vessels. It helps prevent the reflux of embolic agents, reducing the risk of unintended embolization of healthy vessels [17,18]

Thus, the use of better embolic agents, microcatheters, and even cone-beam CT, which provides real-time 3D imaging of the vascular anatomy and helps in precisely locating the fistula point and its relationship to surrounding structures, including the vital radiculomedullary arteries, has now made endovascular intervention easier and better than open surgical methods [19].

However, these procedures involve significant anatomical and technical challenges. The unpredictable location of the feeding radiculomedullary artery, which can arise from any lumbar level or even from smaller branches, poses a risk of misidentification. For example, the artery of Adamkiewicz may originate nearby, and an inadvertent occlusion could lead to catastrophic spinal cord infarction. There is also a risk of injury to nerves and the spinal cord itself. In many cases, AVFs have multiple feeders, which can be very challenging to completely obliterate. In the case of lumbar arteries, catheter navigation is often difficult, as it was in our patient, since the arteries are very tortuous, and precise localization of the fistula can be a tedious task. Post-treatment challenges include the risk of endovascular embolization, where accidental reflux of emboli can cause unintended occlusion of normal radiculomedullary arteries. There is also a chance of fistula recurrence if the embolization is incomplete. Better patient selection: Identifying patients who are most likely to benefit from endovascular treatment is a key area of research. This includes considering the fistula's angioarchitecture, the presence of multiple feeders, and the proximity of the artery of Adamkiewicz.

In our case as well, it was challenging to negotiate the catheter in the radiculomedullary artery as it was very tortuous and locate the exact feeder, and extreme precautions and care were taken while occluding the fistula. We used NBCA as our embolic agent as it has shown really good clinical outcomes, and also it was easily available to us.

Though open surgeries have disadvantages like they are invasive, have a higher risk of injuries, and need more expertise, it has been seen that they have a higher success rate compared to endovascular treatment. A systematic review of studies on spinal dural AVFs (the most common type of spinal AVF) found a failure rate of **20%** for endovascular therapy compared to **4%** in open surgery [18].

But with advancements in the techniques, equipment, and embolic agents, the scope of endovascular intervention looks very promising. Our case is also an example of how endovascular intervention can show great results, though it is technically challenging, but timely intervention can do wonders for the patient.

We discharged our patient on apixaban (to reduce the risk of post-endovascular embolization), and the patient is currently on regular follow-up. She has now improved greatly and mobilising independently.

CONCLUSION

This case report underscores the critical importance of including rare vascular etiologies, such as a spinal dural AVF, in the differential diagnosis for patients presenting with non-specific symptoms like LBP followed by rapidly progressive myelopathy. Despite the rarity of this condition and the non-specific findings on initial MRI, our patient's swift neurological decline prompted a high index of suspicion, leading to a definitive diagnosis via spinal angiography. The successful endovascular embolization of the fistula originating from the right L4 lumbar artery resulted in a remarkable and rapid reversal of her neurological deficits. This outcome highlights that despite the technical challenges and potential risks associated with endovascular procedures in this anatomically complex region, they can be highly effective when performed promptly. Timely intervention is crucial for preventing permanent spinal cord damage and significantly improving patient outcomes. This case serves as a valuable reminder for clinicians to consider spinal angiography in cases of unexplained myelopathy, even with subtle or negative findings on conventional imaging.

Affiliations

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Patient consent

We certify that we have obtained all appropriate patient consent forms. In the form, the patient has given consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Contributors

Dr Sakshi Puri – Concept, design, definition if intellectual content, manuscript preparation and editing Dr Insha Aleena –Design, Data acquisition, manuscript preparation and editing, literature search Dr Manoj Mahata – Concept, Manuscript editing, Literature review, Data acquisition

Conflicts of interest

There are no conflicts of interest.

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We have organized the order of the visits of this patient and interpreted the results. All authors, mentioned above have approved the final manuscript as submitted and agree to be accountable for all aspects of the work. The authors have received no financial compensation for this case report.

Ethics Statement

As this submission is a single-patient case report and does not constitute a research study, it was deemed exempt from review by our institution's Institutional Review Board. This is in accordance with the established ethical guidelines for such publications:

Data Availability Statement

The data supporting the findings of this case report are not publicly available due to patient privacy and confidentiality concerns but are available from the corresponding author upon reasonable request and with appropriate ethical approvals. All data generated or analysed during the case reporting are included in this published article.

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