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Case Report

Unveiling the Uncommon: Vertebral Arteriovenous Fistula from Minor Blunt Trauma Revealed by Delayed Paraparesis

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Abstract

Vertebral artery arteriovenous fistulas (AVFs) are rare vascular anomalies that affect the blood supply to the spinal cord. They are divided into two groups: spontaneous and traumatic, resulting from various events such as cervical surgery, central venous catheterization, chiropractic manipulations, diagnostic cerebral angiography, percutaneous nerve blocks, radiotherapy, penetrating trauma, or, less commonly, blunt trauma. Symptoms are typically related to compression effects, manifesting as extensive myelopathy caused by spinal cord compression. Radiculopathy may also occur due to compression of nerve roots or possibly increased intracranial pressure. In cases of significant arteriovenous shunting, blood steal can occur, leading to cerebral or cerebellar ischemia.

We report the case of a 22-year-old patient with a vertebral AVF causing mass effect on the cervical spinal cord, following a late-presenting blunt trauma without bone injury, spinal cord contusion, ligamentous anomaly, or other associated lesions.

This AVF was treated endovascularly by closing the arteriovenous shunt.

Keywords: *Vertebral Arteriovenous Fistula (VAVF); Spinal Vascular Malformations; Trauma; Paraparesis*

Introduction

The vertebral arteriovenous fistula (VAVF), a rare condition that primarily affects men, accounts for 60 to 80% of all spinal vascular malformations [1,2]. Clinically, it presents with symptoms of myelopathy and radiculopathy, sometimes mistakenly confused with degenerative disc disease, myelitis, or intramedullary tumor [1,2]. They are subdivided into two types: traumatic and spontaneous. Spontaneous VAVFs are generally due to a congenital anomaly or dysplasia of the vessel wall, while traumatic VAVFs result from penetrating, blunt, or iatrogenic trauma.

Case Report

A 22-year-old young man was referred to the interventional neuroradiology department with a one-year history of tetraparesis. The patient reported headaches and neck pain, along with more recent paresthesia and numbness in the right hand following a blunt trauma 2 years ago from receiving a blow to the neck with a ball. About a week before his admission, he experienced sudden severe headaches

described as helmet-like, accompanied by uncontrollable vomiting. This symptomatology persisted for a week with progressive worsening, without any neurological or ocular manifestations. On clinical examination, the patient was conscious, well-oriented, with no motor or sensory impairment.

A CT scan revealed quadri-ventricular hydrocephalus without any identifiable intracranial process. The initial magnetic resonance imaging (MRI) of the cervico-occipital junction showed an extracranial arteriovenous fistula at the level of C1-C2 between the right vertebral artery and a suboccipital venous plexus containing dilated intracanal veins compressing the cervical spinal cord without any associated spinal cord signal abnormality. Additionally, there were intracanal venous ectasias, with the largest one located at the level of C4.

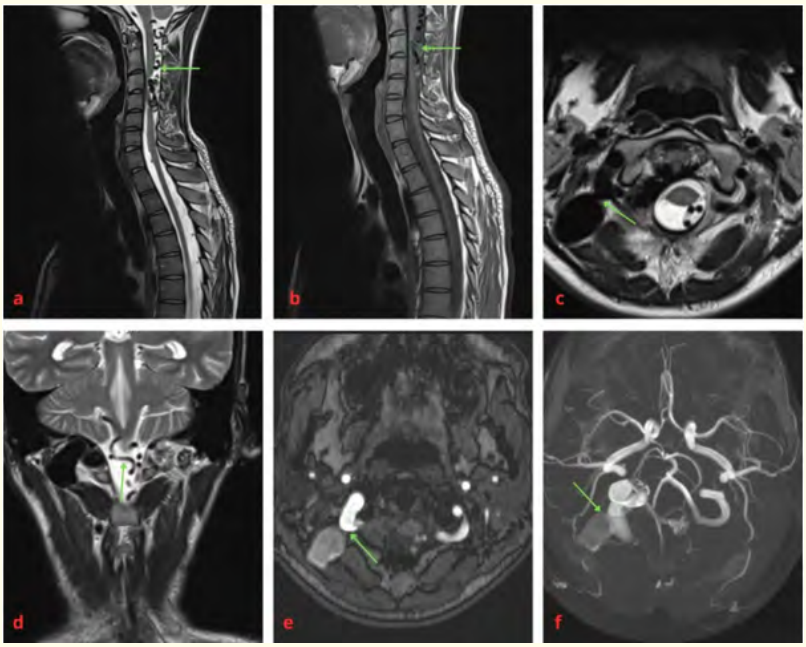

Figure 1: *Pre-embolization MRI images in sagittal T2 (a), sagittal T1 (b), axial T2 (c), coronal T2 (d), contrast injection (e), and arterial TOF (f) showing an extracranial arteriovenous fistula at the level of C1 and C2 between the right vertebral artery and a suboccipital venous plexus containing dilated intracanal veins with spinal cord compression.*

Pre-embolization angiography revealed a significant dilation of the right vertebral artery measuring 7 mm in diameter, with a direct arteriovenous fistula in full canal between the right vertebral artery (segments V3-V4) and the right extraspinal venous plexuses via a venous ectasia, with a 4 mm diameter shunt. Venous drainage occurs through highly dilated and tortuous intracanal veins located on the right side, both anteriorly and lateromedially to the spinal cord. These veins drain partly into the right brachiocephalic vein and partly into a dilated, tortuous, ascending retro-medullary vein with an aneurysmal ectasia at the level of C3, draining into the right basal vein to join the Galenic system. There is a retrograde feed of the fistula through the opacified right V4 segment, supplied by the left vertebral artery.

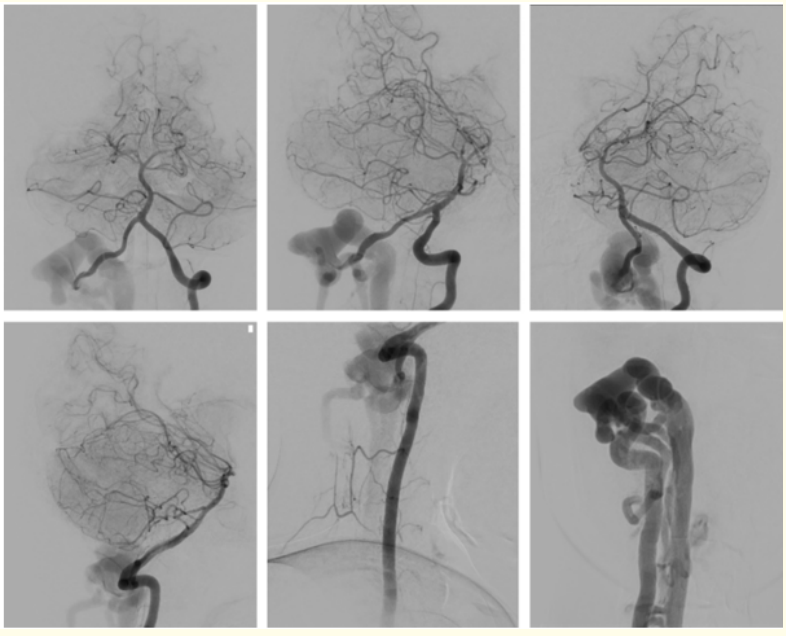

Figure 2: *Angiographic images before embolization showing significant dilation of the right vertebral artery with a direct arteriovenous fistula in full canal between the right vertebral artery (segments V3-V4) and the right extraspinal venous plexuses with a venous pouch.*

The patient underwent a posterior laminectomy at the C3-C4 and C5 levels followed by endovascular closure of the arteriovenous shunt. An MRI performed 2 days later showed the absence of enhancement of the right vertebral artery after Gadolinium injection, with no visible circulating venous structure, decompression of the cervical spinal cord, and the presence of a venous pouch at the level of C4 without mass effect or spinal cord signal abnormality.

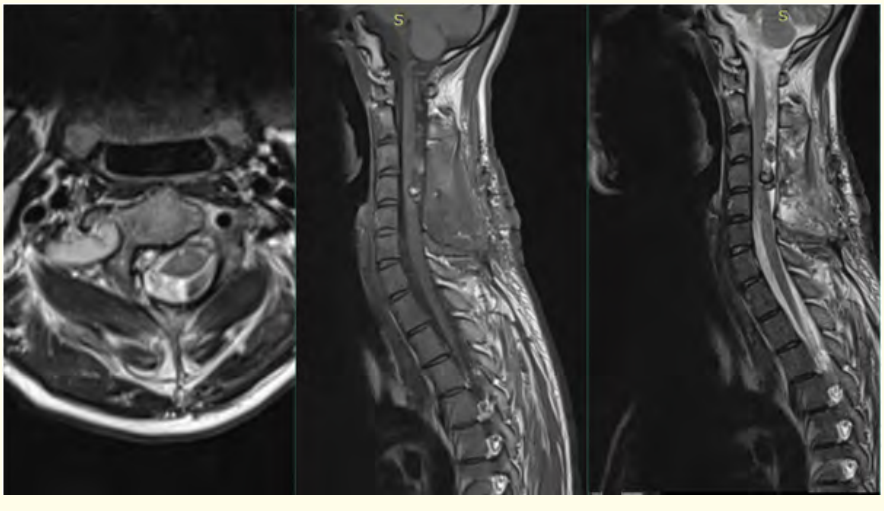

Figure 3: *Post-embolization MRI images showing the absence of visible circulating venous structure, decompression of the cervical spinal cord, with the presence of a venous pouch at the level of C4 without mass effect or spinal cord signal abnormality.*

Clinical follow-up reported a significant improvement in neurological symptoms.

Discussion

Arteriovenous fistulas (AVFs) acquired in the spinal column are rare vascular anomalies specific to this anatomical region. They are typically induced by iatrogenic damage associated with various procedures such as cervical surgery, central venous catheterization, chiropractic manipulations, diagnostic cerebral angiography, percutaneous nerve blocks, radiotherapy, or penetrating traumatic injuries [3,4].

AVF symptoms may present as myelopathy or cervical neuralgia, attributed to arterial reflux into the pial veins of the spinal column or radicular compression due to dilated epidural veins [4]. The onset of symptoms is influenced by factors such as blood flow velocity, venous drainage pattern, and the duration of the lesion [5]. Acute neurological deterioration is mainly associated with arterial steal following rapid blood flow or disruption of venous drainage, leading to venous hypertension. Blunt traumas are an extremely rare cause of spinal arteriovenous fistulas [6,7].

We report the first case of a vertebral arteriovenous fistula resulting from blunt trauma caused by a ball hit, which manifested 3 years post-injury without presenting bone fracture, spinal cord contusion, ligament tear, or other associated injuries.

It is crucial to have a thorough understanding of the anatomy of the venous system where the arteriovenous fistula (AVF) forms [4-8]. The vertebral venous plexus consists of internal and external plexuses including anterior and posterior subdivisions, as well as basivertebral veins. Drainage of the vertebral venous plexuses into paravertebral veins can result in retrograde flow into radicular/radiculomedullary veins, leading to radiculopathy. It is also essential to examine the presence of dural or epidural arterial branches connected to the venous system, as radiculopathy is common in cases of epidural AVFs affecting the lumbar and cervical spine. An AVF can induce myelopathy through external compression of the thecal sac or venous hypertension. Both myelopathy and venous hypertension can result from a dural AVF. Cases of acute paraplegia associated with AVFs have been documented, often linked to hemorrhage or spinal cord infarction rather than cord compression. Venous thrombosis following an AVF can lead to catastrophic complications depending on the vascular territory drained by the affected veins. Conversely, epidural venous thrombosis without involvement of radicular veins is less likely to cause spinal cord hemorrhage. Hydrocephalus observed in our patient may be explained by venous hypertension hindering cerebrospinal fluid resorption.

On T2-weighted images, a serpiginous subarachnoid flow void signal, representing dilated perimedullary vessels, is observed in 45% of cases [4,5,9]. Our patient exhibited an enlarged spinal epidural venous plexus at the C1/2 levels. These findings suggest that the lesion likely originated at the V3 segment of the vertebral artery. Some studies have indicated that significant flow voids on T2-weighted images at the dorsal surface of the spinal cord are among the early signs of AVFs. This flow void signal is characterized by a scalloped and irregular appearance of the spinal cord on T1-weighted images [9]. Mass effect is rare, observed in less than 50% of cases. Abnormal enhancement may be detected at the dilated intramedullary and intramedullary venous plexus levels, as well as during post-contrast study [9]. However, although rare, the diagnosis of AVFs should be considered in cases of T2 hyperintense intramedullary signal, flow voids, and venous plexus enhancement with mass effect [9]. In our case, the mass effect was observed concurrently with flow voids, prompting angiographic study.

The use of gadolinium is crucial in spinal MRI for myelopathy. Studies have shown that its use can enhance the sensitivity and specificity of MRI up to 88% positivity in patients with AVFs [9]. Angiography is the gold standard method for diagnosing AVFs [4]. Unfortunately, this procedure is not always straightforward, as low-flow and volume fistulas can be challenging to identify and often go unnoticed. A potential complication is worsening venous congestion, leading to rapid clinical deterioration due to angiography, necessitating emergency surgical intervention [3]. Prior to angiography, MRI evaluation is necessary for accurate identification of the fistula, especially if it is a slow-flow

fistula. However, three-dimensional rotational angiography equipment would be more effective in visualizing complex spinal vascular architecture.

The primary treatment goal is to occlude the fistula, preventing communication between the artery and vein while preserving the vertebral artery [10]. In the case of our patient, the fistulous communication was correctly identified and successfully occluded through a right femoral artery puncture approach followed by catheterization of both vertebral arteries, catheterization of the right vertebral artery with a guiding catheter (6F Chaperon) placed at the distal V2 level, followed by advancement of a microcatheter (Rebar 18) with a microguidewire (Traxcess 14) for catheterization of the venous pouch downstream of the shunt and embolization using numerous coils, leading to the exclusion of the shunt and distal V3 portion. Subsequently, cross-catheterization of the right vertebral artery V4 segment via the left vertebral artery, followed by shunt catheterization and embolization using multiple coils, resulted in the exclusion of the distal V4 segment. The final follow-up showed complete exclusion of the AVF.

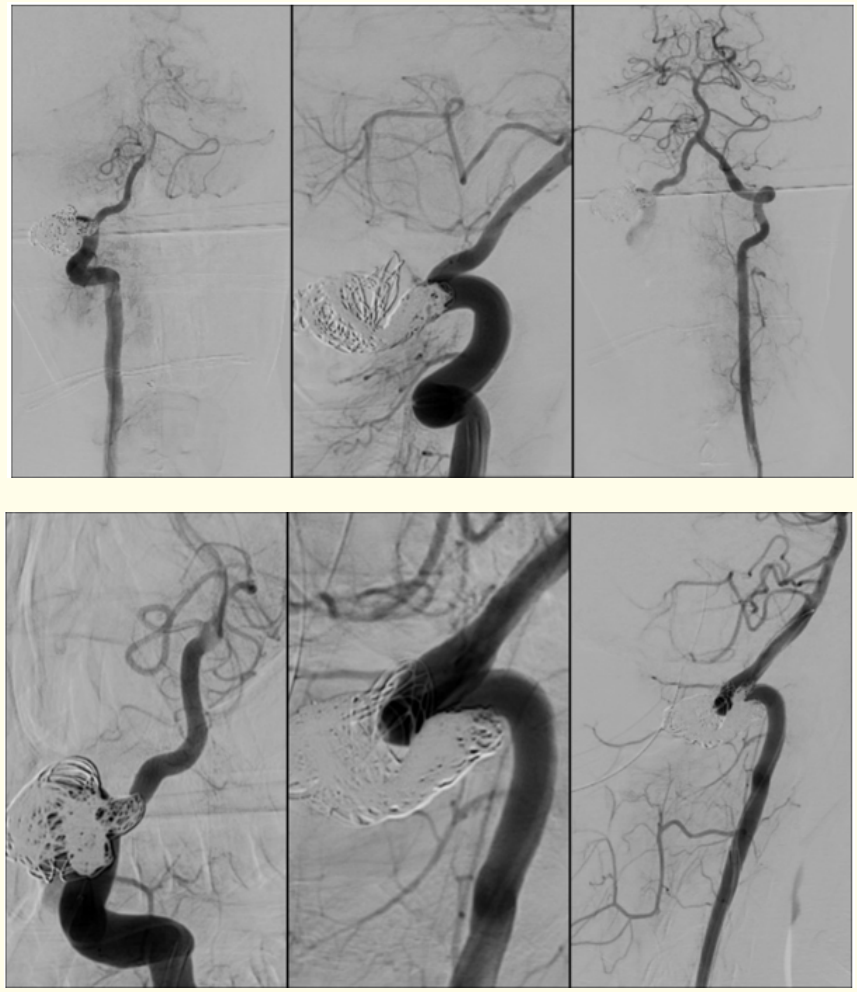

Figure 4: *Post-embolization angiography showing complete exclusion of the AVF.*

In addition to coiling, endovascular occlusion using a balloon is a feasible endovascular treatment option, as described by Briganti., *et al*. However, fistula recurrence may occur due to balloon deflation. The use of a stent in treating the AVF would help maintain vascular patency but could be challenging in very rigid and tortuous vessels [11]. Potential drawbacks of a covered stent include intra-stent thrombosis and incomplete closure of the fistula due to poor positioning [12]. Particulate embolic agents and cyanoacrylate glue are not recommended for high-flow fistulas due to the risk of distal migration and infarction [13]. In addition to the transarterial approach, transvenous embolization could be considered to preserve the vertebral artery [14].

Endovascular treatment of the AVF is preferred over surgery due to postoperative complications such as flow leak symptoms, massive bleeding, and damage to surrounding structures [15].

Conclusion

AVFs due to cervical blunt neck trauma are a rare condition. Imaging is essential as early detection is vital for subsequent management and minimizing complications. The presence of significant flow void images on T2-weighted images at the dorsal surface of the spinal cord are among the early signs of AVFs. This flow void signal is characterized by a scalloped and irregular appearance of the spinal cord on T1-weighted images. Therefore, angiography is the diagnostic and therapeutic method of choice by closing the arteriovenous shunt.

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