

Spinal dural arteriovenous fistula in a patient with gait disturbances

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CLINICAL HISTORY

We present a case of a man who came to our hospital with complaint of progressive gait disturbances, paresthesias, erectile dysfunction, bowel and bladder retention.

IMAGING FINDINGS

We present a case of a 67-year-old patient with no remarkable previous clinical history. He came to our hospital complaining of 40 day progressive gait disturbances especially for the initial steps, paresthesias in his left lower limb, and specifically asked, he also referred progressive erectile dysfunction and 20 to 25 days evolution of bowel and bladder retention.

The patient was sent to the radiology department for a lumbar MRI with suspicion of medullar compression. In the sagittal T2-weighted sequences cord oedema was perceived and perimedullary flow voids were identified (Fig. 1). On heavily T2-weighted sequences (constructive interference in steady state, 3D-CISS) a coiled or serpentine vascular structures was appreciated (Fig. 2) and the diagnosis of DAVF type I was done. A week later a selective angiography confirmed the diagnosis. Injection of contrast agent into the second left lumbar artery caused the filling of a dural malformation which drained into a dilated varicose vein located in the left and posterior of the spinal cord (Fig. 3). After superselective catheterisation of the feeding artery endovascular embolisation with Onyx was done. Complete embolisation of the DAVF was proved with a post procedure arteriography.

Within the next weeks the patient referred an improvement in the lower limbs sensitivity and the gait disturbances, being able to walk about 5-6 Km a day, but he did not notice an improvement in the bowel retention. Follow-up MRI showed resolution of the spinal cord oedema (Fig. 4).

DISCUSSION

Spinal DAVFs are the most commonly encountered vascular malformation of the spinal cord and account of approximately 70% of all vascular spinal malformations [1]. They are usually underdiagnosed because the presenting clinical symptoms are not specific [2].

There are 4 groups of DAVFs. DAVF type I is the most common (up to 80%), it is due to a spinal dural arteriovenous fistula, present within the dura mater with intradural draining veins. DAVF type II (15-29%) or intramedullar glomus type consists of direct arteriovenous communications forming a compact nidus within the spinal cord. DAVF type III or juvenile type is the less frequent (about 7%) and it consists of direct arteriovenous communications, without capillary bed, involving the spinal cord. Finally the DAVF type IV is a direct intradural, extramedullar arteriovenous communication from the anterior or posterior spinal arteries to a draining vein without capillary bed.

Most fistulas are solitary lesions typically found in the thoracolumbar region usually between T6 and L2. Spinal DAVFs are acquired diseases, but their aetiology is unknown. The AV shunt is located inside the dura mater near the root of the spinal nerve. The DAVF causes an increase in the spinal venous pressure due to arterialisation which decreases the AV pressure gradient and leads to a decreased drainage of normal spinal veins and venous congestion [3]. This vascular congestion leads to intramedullary oedema, chronic hypoxia and progressive myelopathy [4].

Initial symptoms are nonspecific and typically include gait disturbances, difficulty in climbing stairs, and sensory symptoms such as radicular pain, paresthesias and sensory loss which initially can affect only one limb. Other symptoms usually seen in the late course of the disease are bowel and/or bladder incontinence, urinary retention and erectile dysfunction. These symptoms are progressive with time as the myelopathy progresses [5].

Usually the diagnosis is suspected with an MRI study and it is confirmed with a digital subtraction arteriography. Spinal cord oedema is generally present on T2-weighted images, and it appears as centromedullary badly-delineated hyperintensity along multiple segments. The coiled or tortuous vascular structures are better appreciated on heavily T2-weighted sequences such as 3D-CISS (constructive interference in steady state) and FIESTA (fast imaging employing steady-state acquisition) or 3D turbo spin echo. Localising the shunt previous to the angiography is of great value. On selective angiography, stasis of contrast agent in the radiculomedullary arteries, especially the anterior spinal artery, is usually seen.

The treatment is to occlude the shunting. There are two options to occlude DAVF, surgical treatment occluding the intradural vein or the endovascular therapy using liquid embolic agents after superselective catheterisation of the feeding artery [6].

In conclusion spinal DAVF are a rare but treatable cause of progressive paraplegia. The MRI usually raises the possibility of this diagnosis, so the neuroradiologists play an important role in the diagnosis and treatment. Even though the symptoms are not specific, the MRI findings of cord oedema and perimedullary dilated vessels without any intramedullary nidus of vessels are typical findings of a DAVF.

FINAL DIAGNOSIS

Spinal dural arteriovenous fistula type I

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- [4] Krings T, Lasjaunias PL, Hans FJ, et al (2007) Imaging in spinal vascular disease Neuroimaging Clin N Am 17:57-72
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Figure 1. Sagittal T2-Weighted Image

Sagittal T2-weighted image where cord oedema was noticed and perimedullary flow voids were identified.

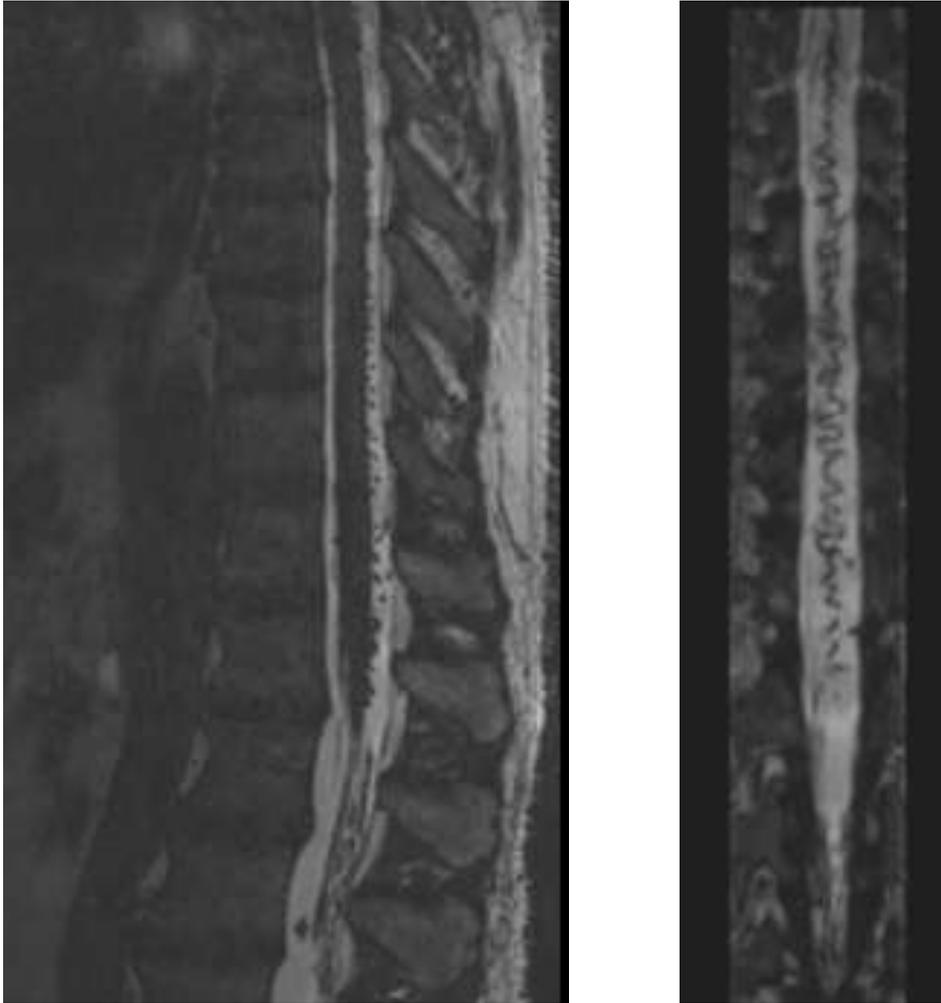


Figure 2. 3D-CISS Images

On heavily T2-weighted sequences (constructive interference in steady state, 3D-CISS) a perimedullar coiled or serpentine vascular structure is appreciated. With this image the diagnosis of DAVF type I was done.

3D-CISS coronal reconstruction where the serpentine vascular structure is appreciated. This is very suggestive of DVAF.



Figure 3. Angiography

Supraselective catheterisation and injection of contrast agent into the second left lumbar artery which caused the filling of the dural malformation that drained into a dilated varicose vein located in the left and posterior of the spinal cord. DAVF type I.



Figure 4. Post-treatment MRI

A sagittal T2-Weighted image taken 5 years after endovascular treatment shows complete resolution of the spinal cord oedema.