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ABSTRACT

Spinal dural arteriovenous fistulas (SDAVFs) are vascular lesions caused by the interactions between the arterial and venous vascular systems of the spinal cord, leading to venous congestion of the spinal cord. They are the most frequent vascular malformation of the spine. These acquired lesions occur most commonly in the thoracic and lumbar spine of middle-aged men. Although the natural history of SDAVFs is not clearly defined, their clinical course is characterised by progressive neurologic decline leading to functional disability.

Here we report a case of a 60-year-old male patient admitted with complaints of back pain that irradiated to the lower legs, intermittent claudication, who then developed progressive paraparesis, gait disorder, and sphincter disturbances. The symptoms were typically progressive for four months with increasing severity. On MRI, on sagittal T2-weighted images, the cord oedema is depicted as a centromedullary hyperintensity, extended in dorso-lumbar (Th4-L1), and numerous serpiginous vessels dorsal to the spinal cord. Based on physical examination and magnetic resonance imaging findings, a preliminary diagnosis of SDAVF was made. The diagnosis was confirmed by spinal angiography, with a selective injection of the left Th9 segmental vessels, which demonstrates a dorsal intradural AVF. The aim of treatment in SDAVF is to occlude the shunting zone, the most distal part of the artery, together with the most proximal part of the draining vein, either by superselective embolisation with a liquid embolic agent or by neurosurgical approach. Our patient was treated by Microsurgery.

Spinal dural AVFs diagnosis should not be overlooked, and it should be kept in mind that early diagnosis and treatment prevent severe morbidities. The gold standard for the diagnosis is selective spinal angiography. Angiography is used for localization of the vascular lesion and treatment. Microsurgery and endovascular embolisation are effective methods for the treatment of SDAVF. Following occlusion of the fistula, the progression of the disease can be stopped, and improvement of symptoms is typically observed.

In this article, we report a case of SDAVF Intradural dorsal AVFs presenting with progressive paraparesis, gait and sphincter disturbances, bladder in the light of literature data.

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Introduction

Dural arteriovenous fistula (DAVFs) are complex vascular lesions composed of dural arteries and intradural veins that are connected by abnormal, low-resistance, arteriovenous connections within the dura mater. Although some cases are congenital, the more common adult form is an acquired lesion. These lesions arise on naturally existing arteriovenous connections in the normal anatomy of the dura mater, but these connections have augmented flow secondary to a pathological process, such as venous hypertension. They may occur in any dural location, such as the cerebral convexity, posterior fossa, or spinal cord,

A spinal dural arteriovenous fistula (SDAVF) is an abnormal connection between arteries and veins in the dura, the outer lining of the spinal cord.

Normally, arteries and veins are connected by tiny vessels called capillaries. Capillaries handle the transition from higher to lower pressure. But in an SDAVF, the capillaries are missing. Arteries and veins are directly connected to each other. The direct connection between artery and vein is called a fistula. This abnormal connection leads to a number of problems. Any of these problems with blood flow, pressure, and nutrient delivery can cause injury to the tissues around the SDAVF. Tissue death in these areas can cause neurological dysfunction.

Dural AVFs were classified type I according to the classification of Anson and Spetzler In 1992 who classified spinal cord vascular malformations into IV types. [1]

Type I: Dural AVFs are the most common type of vascular malformation, accounting for 70% of all spinal vascular malformations [2]. These fistulas are created when a radiculomeningeal artery feeds directly into a radicular vein, usually near the spinal nerve root in the thoracolumbar region. [3]

It may induce abnormal flow of the blood from the arterial system to the venous system, venous hypertension, venous occlusion, intramedullary edema, and progressive myelopathy. [4-6]

The etiology of dural AVFs is unknown, Since SDAVF is a rare entity with an unknown etiology, its diagnosis can be easily overlooked. [7-8]. these lesions are found primarily in men between the fifth and eighth decades of life. [9-11].

In 2002 and then again in 2006, Spetzler and colleagues [12] modified the previous classification of spinal AVMs, the authors propose a modified classification system for spinal cord vascular lesions into three primary categories: neoplasms, aneurysms, and arteriovenous lesions**.**

The third category, spinal cord arteriovenous lesions, is divided into arteriovenous fistulas and arteriovenous malformations (AVMs). Arteriovenous fistulas are subdivided into extradural and intradural lesions, with the latter being either dorsal or ventral.

Intradural AVFs are the most controversial lesions in terms of origin, pathophysiology, and treatment. Intradural dorsal and intradural ventral lesions are distinct entities.

Intradural dorsal AVFs, previously referred to as type I dural AVFs under the older naming convention. The new classification divides these lesions into two subtypes based on their number of feeding vessels. Type A, with a single feeding artery, and Type B, with multiple feeding arteries. [13]

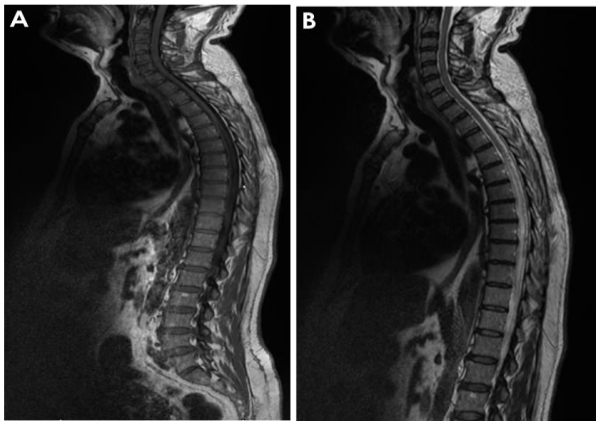
It is presumed that SDAVFs are acquired diseases, though their exact etiology is not known. The AV shunt is located inside the dura mater close to the spinal nerve root where the arterial blood from the radiculomeningeal artery (the artery that supplies the nerve root and meninges but not necessarily the spinal cord) enters a radicular vein, where the latter passes the dura at the dorsal surface of the dural root sleeve in the intervertebral foramen and forms a fistula, arterializing the coronal venous plexus [14]. This transition is classically located directly underneath the pedicle of the vertebral body, which is supplied by the injected segmental artery. The increase in spinal venous pressure due to arterialization diminishes the AV pressure gradient, thickening of the intramedullary veins and leads to a decreased drainage of normal spinal veins and a venous congestion with intramedullary edema because the intramedullary veins and the radicular vein share a common venous outflow [15-16]. This congestion, in turn, leads to chronic hypoxia and progressive myelopathy. [17]

SDAVFs are low-flow shunts their primary pathophysiology is venous hypertension. that produce hypertension of the spinal cord veins. Patients generally present with a gradual onset of back pain and radiculopathy, followed by paraparesis and bowel/bladder dysfunction. In rare cases, patients may suffer a more rapid decline from venous thrombosis [18]. Hemorrhage is extremely rare. [19]

CASE REPORT

We report a case of a 60 year-old male with no particular history, He was conscious, and cooperative with stable vital signs, who initially presented a painful intermittent claudication, followed by the installation of lower extremity weakness, gait disorder, and sphincter disturbances, for four months with an increasing severity. Symptoms progressed gradually.

Physical examination showed paraparesis slowly progressive, more marked on the right side, the muscle strength was 5/5 in the upper extremities, 3/5 in the right lower extremity and 4/5 in the left lower extremity. There was no spasticity. The deep tendon reflexes of the bilateral patella and Achilles tendons were hyperactive. The Babinski reflex was not evoked in either feet. Sensitive disorder, hypoesthesia was observed starting from Th9 level "sensitive level".



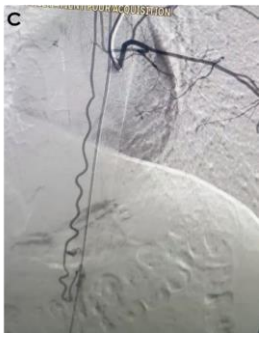


Figure 1: (B), on sagittal T2-weighted magnetic resonance image, the cord edema is depicted as a centromedullary hyperintensity, extended in dorso-lumbar (Th4-L1), and numerous serpiginous vessels dorsal to the spinal cord, (C) A Supraselective spinal microcatheter angiography reveals a dorsal intradural arteriovenous fistula supplied by a branch of the left T5 segmental artery.

Based on these findings, a whole spinal column MRI was performed, showed on T2-weighted sequences (Fig.1.B), the spinal cord displays a centromedullary hyperintense signals over several segments, extended in dorso-lumbar (Th4-L1), and numerous dorsal serpiginous structures peri-medullary, rising after injection, evoking a FAVDR. Electromyography and cranial MRI findings were also normal.

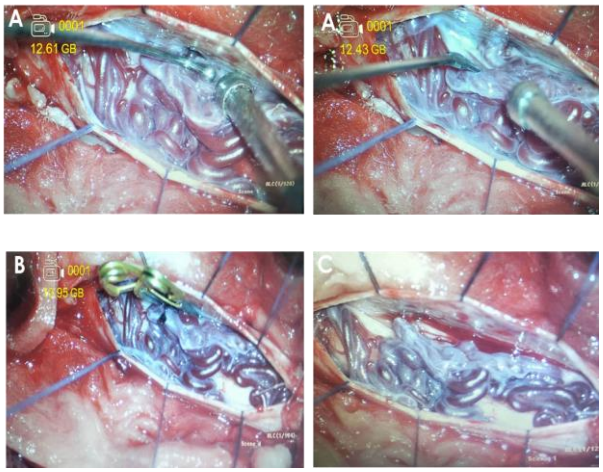


Figure 2. Intraoperative photograph reveals several dilated vessels consistent with a fistula. (A) Identification of the dural arteriovenous fistula. A single feeding arterial pedicle at the left Th9 nerve root sleeve was identified as the fistulous point and after confirming with clampage test (b), the fistula was disconnected (C).

Based on physical examination and magnetic resonance imaging findings, a preliminary diagnosis of SDAVF was made. The diagnosis was confirmed by spinal angiography (Fig.1.C), with a selective injection of the left Th9 segmental vessels demonstrates a dorsal intradural AVF.

This situation was evaluated as a SDAVF and, based on the angiography performed for accurate diagnosis and treatment purposes, SDAVF between the radicular artery and the perimedullary veins was found on the Th9 level on the left.

Therefore, the patient was referred for microsurgery to allow for direct access to the fistula.

During the surgical treatment, the patient was in the prone position, and the appropriate spinal level is verified by radiography. A midline approach with sub-periosteal dissection exposes the posterior elements overlying the level of pathology. A laminotomy Th8–T10 is performed in one piece and is preserved to repair the defect at the completion of the procedure. A durotomy was made, and a large arterialized vein was visible on the dorsal surface of the spinal cord. The fistula was isolated, (Fig.2.A) coagulated, and ligated (Fig.2.C) after a clamping test done using a clip to identify the arterialized vein

(Fig.2.B), instead of other such as indocyanine green (ICG) videoangiography by lack of these modern techniques. Interruption of the medullary vein usually produces a visible change in the vein within a few minutes. The obliteration of the lesion was confirmed by formal postoperative digital subtraction angiography (DSA), and the patient had an uncomplicated recovery with improvement in his pain and no new motor deficits. An outpatient rehabilitation program including passive and active assisted range of movement exercises, muscle strengthening exercises, neuromuscular electrical stimulation, and balance and coordination exercises was initiated from the first week after the operation. Additionally, the patient was consulted with the relevant clinics for bladder and bowel dysfunction.

Discussion

Dorsal Intradural AVFs consist of a direct connection between a radicular feeding artery and the coronal venous plexus; this direct connection leads to arterialization of the venous plexus, venous congestion, and edema. In addition to venous outflow obstruction, arterialization of these veins produces venous hypertension. Spinal dural AVFs are associated with no etiologic factors or comorbid conditions. There is a robust male predominance, and these AVFs are considered to be acquired lesions because of their onset in adulthood.

Clinical symptomatology of this congestive myelopathy is rather unspecific and might consist of hypo and paresthesias, paraparesis, back pain that might irradiate to the lower legs, impotence, and sphincter disturbances [20]. Usually, the deficits are slowly progressive; however, an acute onset of the disease and a progressive development interrupted by intermediate remissions are also possible. Without therapy, this results in irreversible para- or even tetraplegia [21].

On MRI, the combination of cord edema and perimedullary dilated vessels are the characteristic findings and should lead to the diagnosis. On T2-weighted sequences, the cord edema is depicted as a centromedullary, not well-delineated hyperintensity over multiple segments that is often accompanied by a hypointense rim.The cord is swollen and might demonstrate contrast enhancement as a sign of chronic venous congestion and ischemia. The perimedullary vessels are dilated and coiled and can be observed on the T2-weighted images as flow voids [17].

Neither the location of pathologic vessels nor the intramedullary imaging findings seem to be related to the height of the fistula. Localization of the fistula can sometimes be very difficult leading to lengthy and even multiple catheterization procedures during spinal digital subtraction angiography (DSA). Therefore, noninvasive diagnostic techniques such as contrast-enhanced MR angiography with relative fast acquisition protocols have been developed [22–24]. On selective angiography, stasis of contrast material in the radiculomedullary arteries, can be seen [25]. After injection into the segmental artery harboring the AVF, the early venous filling and the retrograde contrast uptake of the radiculomedullary veins are visualized. Often, an extensive network of dilated perimedullary veins is visible.

The aim of treatment in SDAVF is to occlude the shunting zone, the most distal part of the artery together with the most proximal part of the draining vein [26, 27]. A proximal arterial occlusion will lead to a transient improvement of symptoms, however, owing to the good collateralization of the dura, the fistula is prone to recur within the following months. There are two options in the treatment of SDAVF: surgical occlusion of the intradural vein that receives the blood from the shunt zone – a relatively simple and safe intervention –, or endovascular therapy employing a liquid embolic agent after superselective catheterization of the feeding radiculomeningeal artery [28].

Interruption of the AVF may be accomplished by interrupting the venous drainage from the fistula between the dura and the dilated coronal venous plexus,

Surgical treatment begins with a laminotomy one level above to one level below the dural nidus and the intramedullary vein that drains the fistula. After a midline dural opening, the arterialized medullary vein is identified. Routinely indocyanine green (ICG) videoangiography are use to identify the arterialized vein. [29-31] This vessel almost always penetrates the dura at the site of the dural penetration of the posterior nerve.

After identifying and confirming the arterialized medullary vein, the surgeon coagulates the vein and divides it sharply where it enters the inner layer of the dura. Interruption of the medullary vein usually produces a visible change in the vein within a few

minutes. Routinely ICG videoangiography is perform at the completion of the procedure to ensure that the fistula has been interrupted. Watertight closure of the dura is performed using 6-0 nylon suture reinforced with fibrin glue. Laminectomy plates are used to reattach the lamina to the lateral elements to minimize the incidence of kyphotic deformity. The superficial wound is closed in a multilayered fashion.

Intradural dorsal AVFs may be treated by embolization, surgery, or both. Prior to embolization, micro catheter injections are used to verify the absence of normal spinal cord supply. If a spinal artery is visualized, surgical treatment is recommended.

Surgery may also be indicated if an inability to catheterize a feeder or extensive collateralization prevents complete fistula obliteration, as fistula reconstitution may occur if sufficient penetration of the fistula and draining vein is not achieved. These cases are also best treated with surgery, as proximal access to the fistula has likely been lost.

The goal of embolization is obliteration of the fistulous site as well as of the proximal portion of the efferent intradural arterialized draining vein. However, if the embolic material penetrates too far, occlusion of perimedullary veins may exacerbate venous hypertension.

More recently, reports detailing the use of Onyx as the embolisate have emerged [32]. Following successful embolization, collateral supplyto the fistula must be ruled out by performing microcatheter injections at the same level on the contralateral side, as well as two levels above and below the fistula.

Surgical or endovascular treatement?

Microsurgery has historically been regarded as the treatment of choice for intradural dorsal AVFs,

Although studies directly comparing surgical and endovascular treatment of these fistulas are lacking, a meta-analysis of spinal dAVF treatment was published by Steinmetz et al in 2004. [33] In that analysis, 98% of patients treated with microsurgery exhibited complete fistula obliteration following initial treatment, compared with 46% of patients following embolization, as determined by both radiographic and clinical follow-up. In total, 89% of patients demonstrated improvement or stabilization of neurologic symptoms after surgical treatment.

Although many of the endovascular series utilized both PVA and acrylates, contemporary series using only cyanoacrylates reported recurrence rates of 30 to 75%. Complication rates were low in both series. The authors conclude that surgery may be a more effective treatment strategy for spinal dAVFs. Although most modern series have evaluated acrylic embolization, Onyx embolization is emerging as an effective treatment option for dAVF. The first report in 2003 detailed two patients treated in this manner [34]. An additional report of three patients was published in 2008. [35]

Conclusion

Spinal dural AVFs diagnosis should not be overlooked, and it should be kept in mind that early diagnosis and treatment prevent severe morbidities. Although the diagnosis of Spinal dural AVFs is possible with the help of the suspicion of the clinician and attention of the radiologist during magnetic resonance imaging (MRI), the gold standard for the diagnosis is selective spinal angiography. Angiography is used for localization of the vascular lesion and treatment. Microsurgery and endovascular embolization are effective methods for the treatment of SDAVF.

The goal of treatment for spinal AVFs is permanent elimination of the venous congestion of the spinal cord, to halt the progression of the disease. This goal may be accomplished by interrupting the venous drainage from the fistula between the dura and the dilated coronal venous plexus. Simple interruption of the AVF produces permanent resolution of the venous congestion and improvement of myelopathy in many patients.

In some institutions embolization is used to treat these AVFs, but we maintain that surgery is optimum for two reasons. First, the rates of recurrence and progressive myelopathy associated with embolization are high. Second, in surgical series, the reported morbidity rate is extremely low and the success rate high.

Following occlusion of the fistula, the progression of the disease can be stopped and improvement of symptoms is typically observed. The prognosis depends on both the duration of symptoms as well as the clinical condition prior to therapy.

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