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# Paraspinal Arteriovenous Fistula with Perimedullary Venous Drainage

C. Cognard, H. Semaan, S. Bakchine, Y. Miaux, S. Thibault, M. T. Sola Martinez, and J. Chiras

**Summary:** A paravertebral presacral posttraumatic arteriovenous fistula drained through the ascending lumbar vein to the epidural plexuses and perimedullary veins. The patient did not have symptoms of myelopathy, only low-back pain and radicular hypoesthesia. The lesion was embolized with a large balloon and clinical symptoms disappeared.

**Index terms:** Fistula, arteriovenous; Arteriovenous malformations, embolization; Arteriovenous malformations, spinal

The major forms of spinal vascular malformations can be classified on the basis of their location (1): (a) intraspinal (ie, inside the spinal canal; intramedullary, perimedullary, or purely epidural or radicular); (b) vertebral (hemangiomas); or (c) paravertebral. Intraspinal malformations always drain into perimedullary veins. Epidural or paraspinal shunts may drain into paravertebral, epidural, or intradural venous systems. Paravertebral shunts are rare and usually located at the cervical level. They may be congenital or posttraumatic. We report a paravertebral presacral fistula, possibly posttraumatic, with perimedullary venous drainage.

## Case Report

A 17-year-old girl presented with a 6-month history of progressively increasing low-back pain, that began after she fell from a horse and landed on her buttocks. The examination revealed only a radicular hypoesthesia in the area of L-4 and L-5 on the left. Plain films showed a widening of the intervertebral L4-5 and L5-S1 foramina, with scalloping of the posterior wall of the vertebral body and pedicles of L-4 and L-5. Magnetic resonance (MR) showed very large vessels inside the L4-5 and L5-S1 foramina (Fig 1, A-C). Widened intradural veins were seen from the level of L-5 to T-5. Numerous large venous ectasias were present in front of the conus medullaris, which was shifted backward. There was no abnormal signal intensity or enlargement of the conus or spinal cord. Selective spinal angiography (Fig 1, D and E) disclosed a left paraspinal arteriovenous fistula (AVF) fed by a greatly

enlarged iliolumbar artery and a small left branch of the medial sacral artery. The single direct high-flow fistula drained via the ascending lumbar vein to the epidural veins through the L4-5 and L5-S1 radicular veins. The intradural drainage then ascended to the conus with large premedullary venous ectasia. These ectatic veins drained through lumbar veins to the epidural venous plexuses and into the anterior medullary vein, which could be followed to T-5. The Adamkiewicz artery was fed by the left T-2 intercostal artery. The spinal venous return was never opacified.

Embolization was deemed the treatment of choice. The primitive iliac artery was catheterized with a 9F catheter. One balloon (Ingenor 12) filled with 1.2 mL of contrast was detached at the level of the shunt (Fig 1F). Another one was detached downstream. The control angiogram showed the exclusion of the fistula (Fig 1G). Follow-up MR obtained 1 month later (Fig 1, H-I) showed the disappearance of enlarged epidural and intradural veins. The large venous ectasias at the level of the conus were thrombosed with hypersignal on T1- and T2-weighted images. The patient remained asymptomatic 7 months after the embolization.

## Discussion

The difference between spinal cord and spinal dural arteriovenous malformations was described by Kendall and Logue (2) and Merland et al (3). These malformations with a nidus or shunt inside the spinal canal and their clinical consequences have been the subject of numerous reports. Paraspinal arteriovenous malformations or AVFs, much rarer, are not as well known (1, 2). At the cervical level, paraspinal arteriovenous shunts fed by the vertebral arteries are more common. They can be direct arteriovenous shunts or real malformations with a nidus. They are frequently traumatic or congenital in origin.

Lumbar paravertebral malformations draining into the spinal canal are very rare (4, 5). This case raises three interesting points: the

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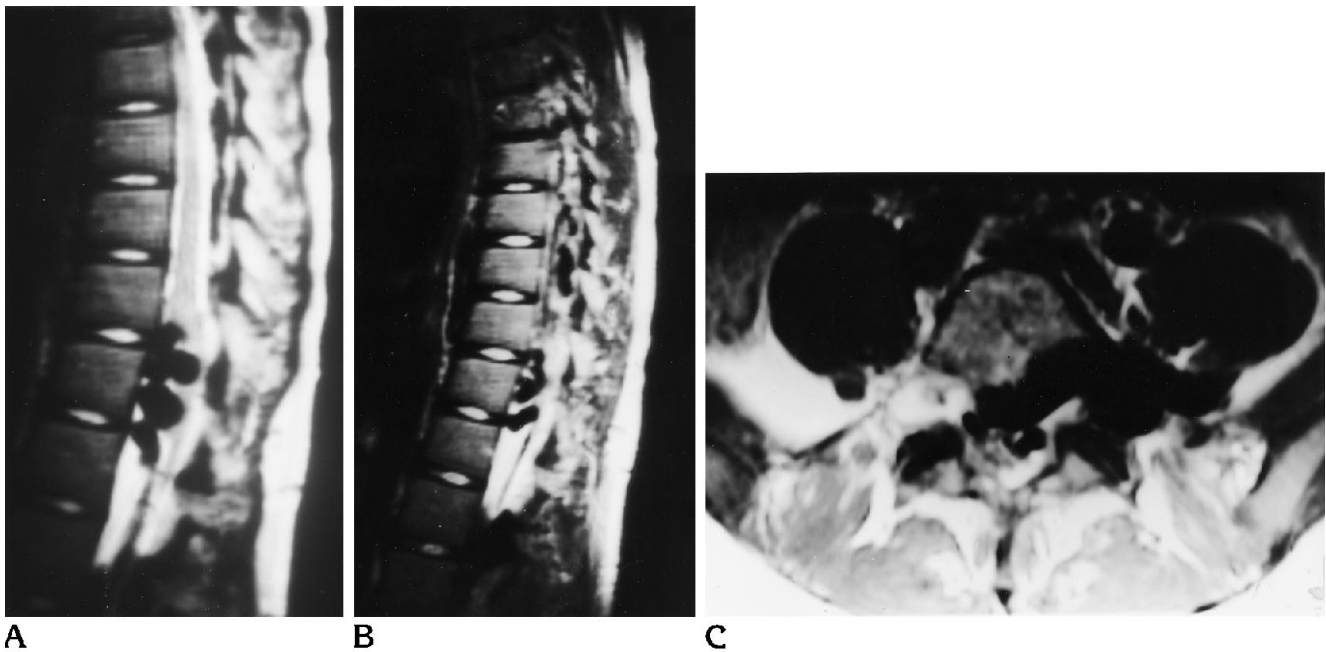


Fig 1. A and B, T2-weighted fast spin-echo (4500/91 [repetition time/echo time]) sagittal MR images show dilated flow void structures with ectasia compressing the conus medullaris (A) and the dilated vein located to the *left* and *posterior* of the cord (B).

C, Fast spin-echo proton density-weighted (3500/15) axial MR image. Vascular dilatations occupy the spinal canal and the *left* L5-S1 foramen. *Figure continues.*

origin of the AVF, the clinical presentation regarding the venous drainage, and the simple endovascular treatment.

The first symptoms of the fistula appeared 6 months after a fall from a horse. The absence of an arterial network or nidus and the appearance of single-hole fistula are consistent with an acquired posttraumatic shunt. Nevertheless, if the lesion had been present for only 1 year, it is difficult to explain the bone changes, scalloping of the posterior wall of the vertebral body, and enlargement of neural foramina. Such bone remodeling would occur from a longstanding abnormality. In this very high-flow direct shunt, the pulsations of the arterialized vein could have induced more-rapid bone changes. Further evidence against a traumatic cause is the extent of trauma. Posttraumatic AVFs are usually caused by direct penetrating wounds, from stabbing or gunshot, or by major trauma with bone fractures. It is difficult to imagine that a fall from a horse, without any combined injury, caused an isolated AVF of the iliolumbar artery, which is located anterior to the sacrum. It is difficult to determine whether this AVF was really posttraumatic or whether it was a congenital or longstanding AVF that became symptomatic after the fall.

An interesting finding is the symptoms presented by the patient. The sensory deficit with hypoesthesia in the left L-4 and L-5 radicular distribution is probably caused by mechanical compression by the greatly enlarged radicular veins or perhaps by inadequate venous drainage of the nerve roots. The absence of symptoms of myelopathy is surprising. This very high-flow fistula drained first into epidural and then into intradural medullary veins that could be followed on angiography to the T-5 level. In addition, the venous return of the anterior spinal artery was never opacified. The idea, proposed by Aminoff, that increased medullary venous pressure is responsible for progressive myelopathy is today commonly accepted (6, 7). A sign of venous hypertension is prolonged spinal circulation with a delayed or absent opacification of the venous return of the Adamkiewicz artery (3, 8). The other radiologic sign of venous hypertension is the enlargement and T2-weighted hypersignal of the cord or conus (9–11). Our patient had medullary veins arterialized by the high-flow shunt and no visible anterior spinal artery venous return. Nevertheless, she did not have myelopathy or abnormal-signal cord on MR.

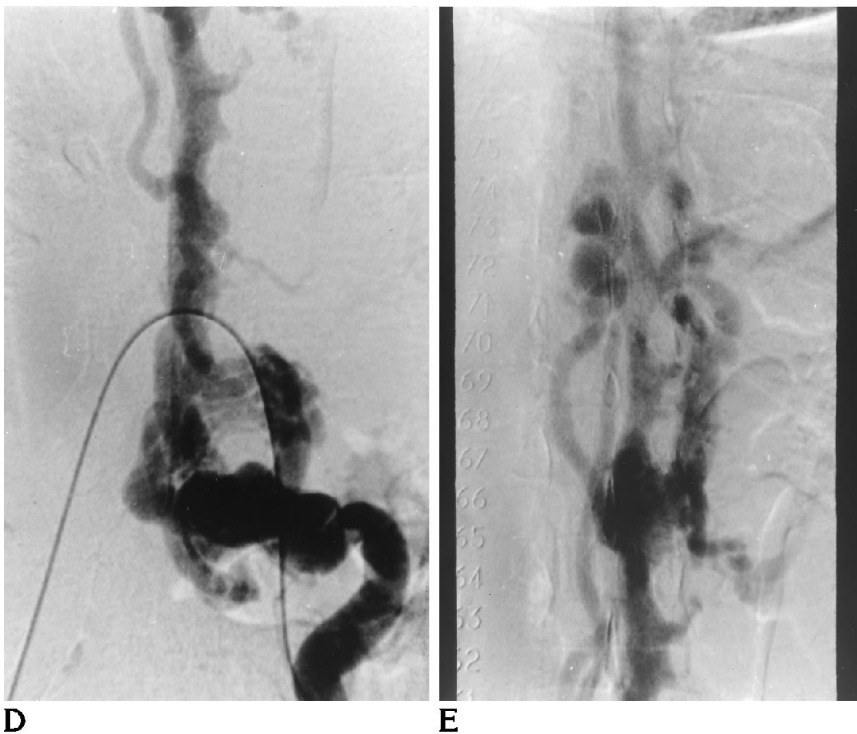
In dural spinal AVFs with perimedullary venous drainage, patients often present with pro-

Fig 1, *continued*.

*D and E*, Selective iliolumbar angiography.

*D*, Lumbar level: greatly dilated iliolumbar artery drains directly through a unique direct shunt into the ascending lumbar vein, then into the epidural vein through the L4-5 and L5-S1 foramina.

*E*, Venous drainage at the dorsolumbar level: intradural anterior perimedullary vein. Note the numerous epidural draining veins. *Figure continues*.



gressive myelopathy. The microscopic and low-flow dural shunts drain into perimedullary veins. The drainage is slow, often ascending to the cervical level. In 1980, Merland et al suggested that the ascending venous distension is caused by the absence or lack of draining veins from the spinal cord to the epidural plexus (3). They

considered dural fistulas a global venous disease with either an abnormal number of veins or more probably a blockage of the venous return (thrombosis).

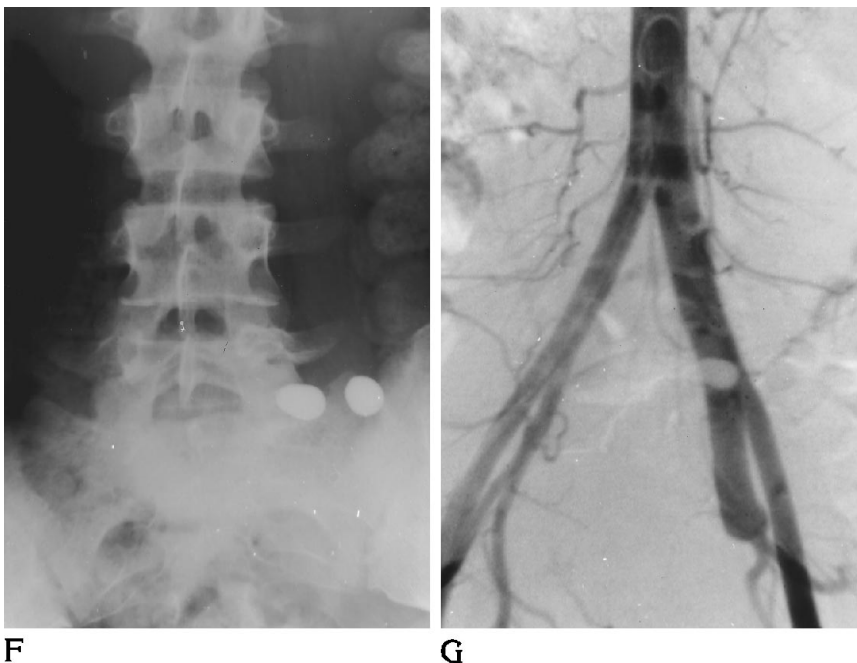
In our case, the medullary veins are greatly dilated and arterialized with a fast circulation, but numerous dilated draining veins are patent.

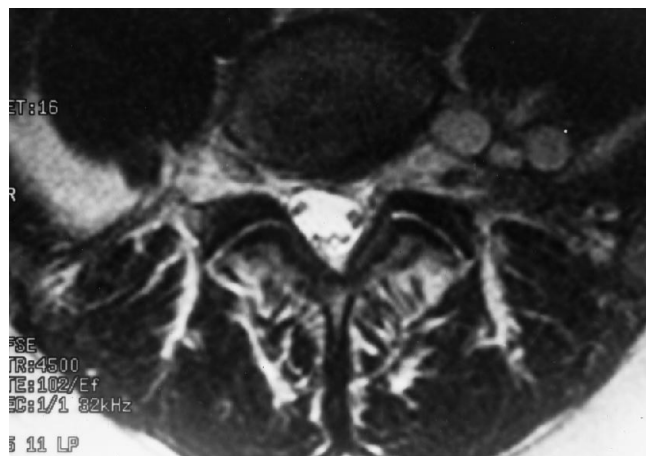
Fig 1, *continued*.

*F and G*, Results of embolization.

*F*, Plain film. Note the two balloons in the region of the shunt.

*G*, Global control angiogram; occlusion of the shunt. *Figure continues*.





H



I



J

Fig 1, continued.

H-J, MR images obtained 3 weeks after embolization.

H, Fast spin-echo T2-weighted (4500/102) axial image at the level of L5-S1. Note the two balloons and disappearance of the dilated veins.

I, Fast spin-echo T2-weighted (4500/102) and J, T1-weighted (540/13) sagittal images; disappearance of the dilated epidural and intradural veins. Hypersignal in the venous dilatations at the thoraco-lumbar junction on both T1- and T2-weighted images is caused by the thrombosis.

This probably accounts for the lack of myelopathy and explains the normal appearance of the cord and conus on MR. The venous return of the Adamkiewicz artery was not visible, perhaps because it was diluted by the venous return of the fistula.

Another question concerns the risk of intradural bleeding in this case. The shunt is paravertebral, but the large venous ectasias visible in front of the cord could possibly be responsible for hemorrhage, as in intracranial dural fistulas (12).

The treatment was achieved by an endovascular approach. A large balloon (Ingenor 12) was necessary to occlude the very wide single shunt. Just after the treatment, the low back pain and radicular hypoesthesia disappeared. The follow-up MR showed the thrombosis of large venous ectasias and disappearance of dilated intradural veins.

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