Spinal Intraosseous Arteriovenous Fistula in the Fractured Vertebral Body


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**CASE REPORT**

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**SUMMARY:** A 68-year-old woman presenting with progressive paraparesis was diagnosed with an AVF involving a previously fractured L1 vertebral body on which feeders from multiple segmental arteries converged. The most distinctive feature in our patient was that the fistula was located in the vertebral body. Transarterial embolization of the segmental arteries with coils and glue resulted in total obliteration of the fistula, which in turn resulted in symptom improvement.

**ABBREVIATION:** AVF = arteriovenous fistula

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**E**pidural or paraspinal AVF is very rare, and few cases have been reported in the literature.1,4 It is not a well-known entity, and it is not always easy to distinguish an epidural AVF from a dural AVF when reflux into the perimedullary veins is present. Although intradural reflux is rare in paravertebral shunts, it may show angiographic findings similar to those of a dural AVF and venous hypertension of the spinal cord.2 We report a case of trauma-related intrasosseous fistula directly draining through the L1 basivertebral vein into the epidural, paraspinal, and perimedullary venous plexuses.

**Case Report**

A 68-year-old woman presented with a 5-month history of a tingling sensation in both legs and a 2-month history of gradually progressive paraparesis. In addition, she also had a 9-year history of back pain after falling from a ladder.

Neurologic examination revealed that she was unable to walk steadily and had paraparesis (grade IV/V). Sensory examination demonstrated paresthesia and hypesthesia of the perianal area and both lower extremities, symmetrically. Urinary retention and fecal incontinence were noted. Her deep tendon reflexes were slightly increased in the bilateral lower extremities. Pathologic reflexes, such as ankle clonus and Babinski sign, were positive.

Engorged intrasosseous and perimedullary veins accompanying cord signal-intensity changes were detected on CT scans (Fig 1A, -B) and MR images (Fig 2). Spinal angiography showed intrasosseous fistulas and multiple feeders from bilateral L1 and L2 and right T12 segmental arteries (Fig 3).

She was diagnosed with trauma-related epidural or paraspinal AVF. We confirmed that the venous flow drained mainly via the intrasosseous L1 basivertebral vein into the epidural venous plexus on the left in a downward direction; then, the flow drained into the left paravertebral veins on the spinal angiography. We tried embolization through the left femoral vein but failed to find the connection between the epidural or paravertebral vein and the left femoral vein or inferior vena cava. Transarterial embolization was selected as the treatment technique.

The muscular branches in each segmental artery were protected by coils due to the delay of wound healing, which might have been caused by occlusion of superficial arteries during embolization. Multiple feeders were occluded, including the venous sac, with 33% glue—that is, a mixture of n-butyl cyanoacrylate (Histoacryl; B. Braunn, Melsungen, Germany) and iodized oil (Lipiodol; Guerbet, Aulnay-Sous-Bois, France) at a ratio of 1:2. The exact volume of embolic material was not calculated. The final angiography confirmed total obliteration of the fistula.

Neurologic examination performed 5 days after surgery revealed that the patient’s leg weakness had improved (Grade IV/7/V) and that her perianal hypesthesia and urinary retention had resolved. Follow-up MR images obtained 5 days after the intervention showed a reduction in cord signal intensity, near obliteration of perimedullary signal-intensity voids at the thoracolumbar junction, and thrombotic change in the basivertebral and epidural veins. MR images obtained 7 months later showed the disappearance of cord signal-intensity change and enlarged pial veins. The patient was able to walk without assistance, but she continued to experience a mild residual tingling sensation in the perianal area and in both legs. She regained full control of sphincter function.

**Discussion**

In the present study, we report a unique case of AVF occurring after an L1 compression fracture. There were many interesting findings in this case. The most peculiar finding was that the fistula was located in the vertebral body. It has been hypothesized that trauma can lead to the development of an epidural or paraspinal AVF. There have been some case reports of epidural shunts related to trauma, but definite evidence of vertebral injury, such as a vertebral fracture, was seldom observed on radiologic examination.1,3,4 Several mechanisms of fistula formation have been suggested. Trauma, with or without vertebral fracture, results in microtears of the affected arterial wall and produces an AVF.5 It is also possible that traumatic events can cause thrombosis or thromboophlebitis of the veins. A fistula can develop due to arterial growth during the process of organization and recanalization.5,6 Another mechanism of fistula formation is increased venous pressure due to impaired venous drainage after trauma causing spontaneous occurrence.6

There is no question that trauma was the cause of fistula formation in our patient. AVFs were located in the vertebral body near the surface. The main feeder was the right L1 segmental artery with a high-flow shunt. The shunt was con-
nected to the basivertebral vein and formed a large defect in
the trabecular bone. We hypothesize that the bone defect
might have originated from the fracture line that was made at
the time of trauma. It is possible that the high-pressure blood
flow might have widened the defect as time passed.

Suh et al reported 2 cases of osseous epidural AVFs as a
variant of a paraspinal or epidural AVF. They differentiat-
ed the dilated venous sac that caused the bony defect from dilated
venous ectasia of Rendu-Osler-Weber disease. The latter did
not receive arterial blood supply directly, and the fistula was
not located in the epidural space. They presumed that the os-
seous epidural AVF was located near the bony margin and
recruited multiple feeders from the dura and bone. On the
basis of the ideas of Suh et al, our case is one of osseous epi-
dural AVF, and we believe that it may be a representative case
because the shunt surgery occurred throughout the entire ver-
tebral body. The venous sac only involved a small portion of
the vertebra in the cases reported by Suh et al.

An epidural AVF can cause neurologic symptoms in several
ways. A direct connection between an extradural artery and vein
leads to the development of a high-flow fistula, engorgement
of the epidural venous system, and compression of the spinal cord
by dilated veins or an epidural hematoma. The high pressure in
the epidural venous system may induce intradural venous hyper-
tension, and the shunt surgery of a large volume of arterial blood
into the venous system can take blood from the spinal cord. Di-
rect compression of the spinal cord or nerve root by dilating epi-
dural veins or possible epidural hematoma can cause myelopathy
or radiculopathy. Intradural reflux in an epidural AVF, as seen in
this patient, is rare, and only a few cases have been reported.1,2

The engorged intradural veins and prominent signal-intensity
changes in the spinal cord makes it challenging to differentiate an
epidural AVF with intradural reflux from a dural AVF. The long
duration from the initial trauma until the onset of myelopathy
may be related to the location of the fistula. Because the fistulas
were located in the vertebral body, it may have taken a long time
to form a shunt surgery fistula that was large enough to cause
myelopathy.

Endovascular surgery was the most reasonable treatment

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Fig 1. A and B, Enhanced CT scans show the old L1 compression fracture, the erosion of bony trabeculae caused by dilation of the basivertebral vein (arrow), and longitudinally enlarged epidural veins (arrows).

Fig 2. A, Preoperative sagittal T2-weighted MR image demonstrates high signal intensity and edematous change of the spinal cord from T7 to the conus. Especially, the engorged L1 basivertebral vein gives rise to the bony erosion in the marrow. Engorged perimedullary veins are found anterior and posterior to the spinal cord from C3 to L5 (arrow). No intramedullary nidus could be detected. Dilated paravertebral veins are also seen anterior to the vertebral body from the level of T6 to L1 (arrow). B, Postoperative sagittal image shows thrombosed basivertebral veins at L1 (arrow). There is no evidence of recanalization. Perimedullary veins and cord signal intensity changes are not visible.
technique for this patient. All feeders were accessible. Surgery required extensive exposure of the spinal column. Suh et al. recommended the transvenous approach for effective embolization of multiple feeders, and though we agree with their recommendation, the venous approach was not possible in this case.

Conclusions

We present a rare case of posttraumatic osseous AVF draining directly through the L1 basivertebral vein, with a reflux to the epidural, paraspinal, and perimedullary venous plexuses. Endovascular therapy via a transarterial approach was an effective and minimally invasive treatment technique.

References