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Spinal Epidural Arteriovenous Fistulas: Arterial and Venous Approaches to Embolization

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Summary: The authors describe two patients with multiple spinal arteriovenous fistulas and exclusive epidural venous drainage. Both presented with a myelopathy caused by compression by large epidural veins. Both had arterial embolizations, but one required a venous approach to achieve a stable clinical result.

Index terms: Arteriovenous malformations, spinal; Fistula, arteriovenous; Fistula, therapeutic blockade; Interventional neuroradiology

Spinal arteriovenous malformations (AVMs) can be divided into intramedullary AVMs and dural arteriovenous fistulas (AVFs) according to their arterial supply (1). Intramedullary AVMs are usually fed by the anterior and posterior spinal axes, whereas dural AVFs are fed by radicular, dural branches. Variations occur, such as the perimedullary AVF fed by either the anterior or posterior spinal axis (2, 3). All of the above-mentioned malformations drain into the perimedullary coronal venous plexus. There are a few reports of epidural AVFs draining exclusively into the epidural veins (4–8). These AVFs were spontaneous (4, 5, 7), traumatic (6), or related to neurofibromatosis (8). In addition, there are a few reports of spinal AVFs with both epidural and intradural venous drainage (5, 9, 10). In this report, we describe two patients with AVFs and exclusive epidural venous drainage. Embolotherapy in one patient required both the arterial and venous approaches, whereas the other patient has only required the arterial approach thus far. A successful transvenous embolization of a perimedullary fistula was reported by Halbach et al (presented at the 29th Annual Meeting of the American Society of Neuroradiology, Washington, DC, June 1991).

Case Reports

Patient 1

A 57-year-old left-handed man had a 4-year history of progressive weakness of his right arm and leg, numbness below the right knee, and urinary hesitancy. Examination revealed 1/5 power of right shoulder abduction, 3/5 power of right elbow flexion, 3/5 power of right hip flexion and 4/5 power in the rest of the muscle groups on the right. The right toe was upgoing. Computed tomography (CT) showed an extradural mass at C3–C4 (Fig. 1A); it was initially interpreted as a nerve sheath tumor, prompting a surgical exploration. At surgery, a vascular malformation was discovered. Angiography revealed multiple AVFs at C4–C5 on the right draining into the epidural venous system. The arterial feeders included: the right vertebral artery, the right ascending cervical artery, the right inferior thyroid artery, multiple branches of the right costocervical artery, and small branches from the left vertebral artery (Fig. 1B and C).

Over the next 4 years, the patient received a series of seven arterial embolizations. The initial embolization, with two #16 contrast-filled Goldvalve balloons (Nycomed Ingénor, Paris, France), closed the two largest fistulas from the right vertebral artery as well as the right vertebra itself. The initial clinical improvement was dramatic, but within 6 months, the patient began to deteriorate with recurrent pain, numbness, and weakness. Over the next 2 years, the AVFs were embolized from the costocervical, inferior thyroid, and thyrocervical branches with a 50:50 mixture of *N*-butylcyanoacrylate (NBCA):lipiodol through a Tracker-18 catheter (Target Therapeutics, San Jose, CA). These embolizations closed the feeders, and penetration into the epidural venous pouch was recognized on a number of occasions.

After each session, clinical improvement was evident but the patient would deteriorate after 4 to 6 months. Retrograde shunting down the distal right vertebral artery was embolized with the same NBCA mixture. Again, the patient improved but 2 weeks later he suddenly lost the

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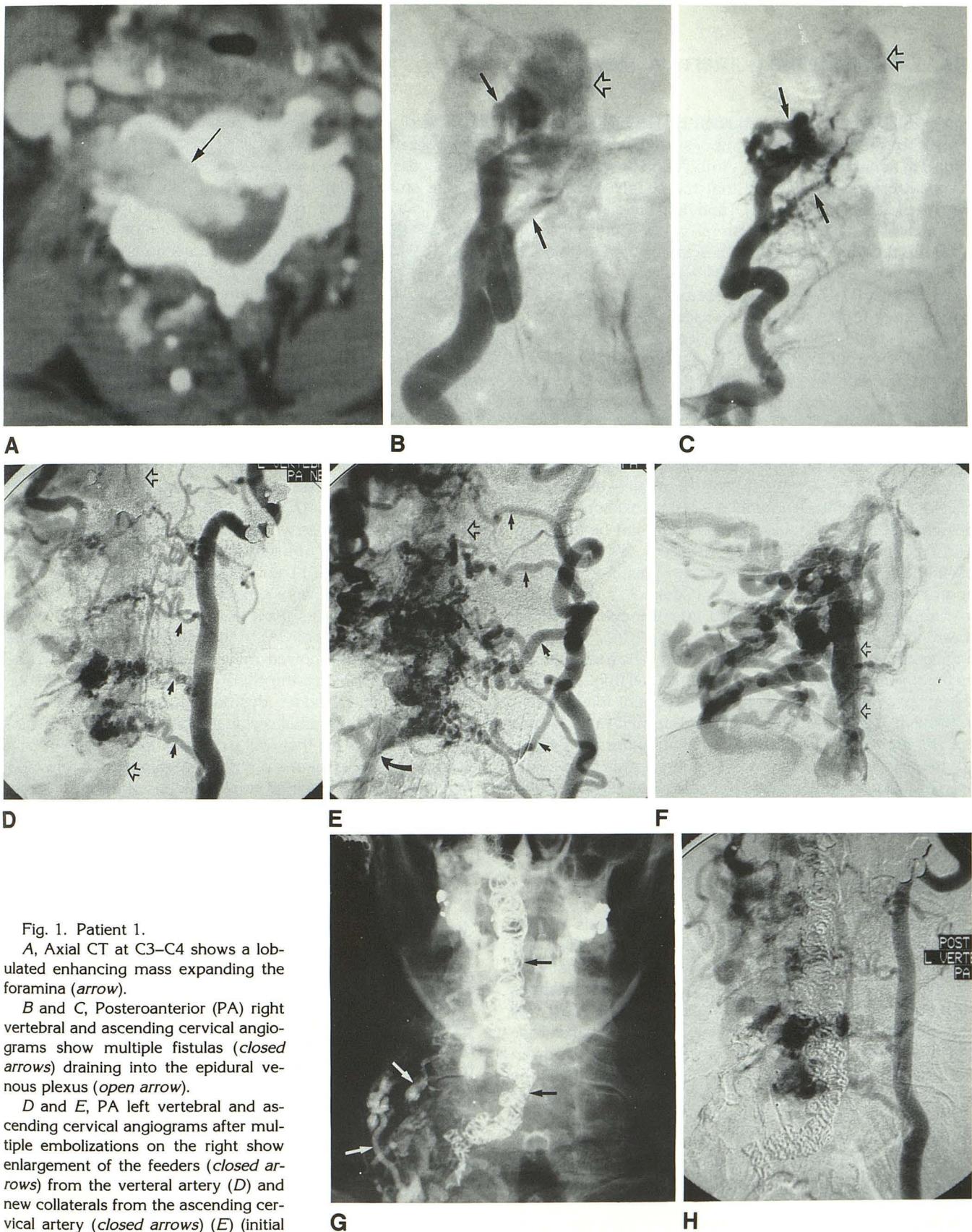


Fig. 1. Patient 1.

A, Axial CT at C3–C4 shows a lobulated enhancing mass expanding the foramina (arrow).

B and C, Posteroanterior (PA) right vertebral and ascending cervical angiograms show multiple fistulas (closed arrows) draining into the epidural venous plexus (open arrow).

D and E, PA left vertebral and ascending cervical angiograms after multiple embolizations on the right show enlargement of the feeders (closed arrows) from the vertebral artery (D) and new collaterals from the ascending cervical artery (closed arrows) (E) (initial left vertebral and left ascending arteriograms not shown). Note filling of the epidural venous plexus (open arrows) draining into the vertebral vein (curved arrow).

F, Lateral cervical epidural venogram shows the epidural venous plexus (open arrows) and extensive paraspinal and suboccipital venous drainage.

G, PA plain film shows the multiple coils in the epidural venous plexus (black arrows) and the liquid adhesive agent (white arrows) in multiple vessels.

H, PA left vertebral angiogram after the coil embolization shows that the epidural venous plexus no longer drains the fistulas.

Figs. 1A through C used with permission from Willinsky R, Terbrugge K, Lasjaunias P, Montanera W. The variable presentations of craniocervical and cervical dural arteriovenous malformations. *Surg Neurol* 1990;34:118–123.

ability to abduct his right shoulder. He remained stable for 1 year but then developed numbness and weakness in the left leg in addition to the right. Ambulation became difficult. A repeat angiogram showed that the supply to the AVFs from the left vertebral (Fig. 1D) and right subclavian arteries had increased, and new collateral vessels had developed from the right occipital and left ascending cervical arteries (Fig. 1E).

At this time, the venous approach was chosen. The right vertebral vein was catheterized with an 8-5 coaxial guiding catheter from a femoral venous access. The epidural vein was catheterized with a Tracker-25; venography showed drainage into a myriad of suboccipital and paraspinal veins (Fig. 1F). The pressure in the epidural vein at C4 was 50 mm Hg. The Tracker-25 was used to deliver 10 0.25-inch coils (Cook, Bloomington, IN; 8 mm in diameter) into the distal epidural pouch. Because there was no change in the flow, a #16 contrast-filled Goldvalve balloon was placed into the distal pouch. A second balloon was not detached because a stable position could not be achieved. The Tracker-25 was then replaced with the 5-F component of the 8-5 coaxial system. Through this 5-F catheter, approximately 70 0.38-inch coils (Cook; 3, 5, and 8 mm in diameter) were tightly packed into the epidural pouch down to the vertebral vein at C8 (Fig. 1G). A control angiogram in the left vertebral artery showed that the epidural vein was excluded from the fistulas (Fig. 1H). Over the next few days to weeks, the patient dramatically improved. The power in his right leg returned to 4+/5, and the left leg returned to normal. The numbness in his legs completely resolved. His inability to abduct his right shoulder persists. He has remained stable with respect to his symptoms and neurologic examination for more than 1 year.

Patient 2

A 41-year-old man presented with paresthesia in both legs, bilateral leg weakness, and bladder dysfunction. This was associated with severe right lower back pain that kept him up at night. He had multiple vascular malformations removed from the lower posterior aspect of the chest wall on the right. An examination revealed slight increased tone and hyperreflexia in the lower limbs with normal power; his toes were downgoing. There was tenderness over the lower right posterior rib cage and an accompanying bruit.

CT showed a large extradural mass at T9 extending into the paraspinal region (Fig. 2A). Magnetic resonance (MR) showed the displacement of the cord to the left, but no abnormal signal within the cord on T2-weighted sequences was seen. Angiography revealed multiple large shunts into a dilated epidural venous plexus from the right T8 and T9 intercostal arteries (Fig. 2B and C). The right T7, T10, and T11 and the left T7, T8, T9, T10, and T11 intercostal arteries participated by collateral flow (Fig. 2D and E). The artery of Adamkiewicz arose from the left T10 intercostal (Fig. 2E). The spinal cord circulation time was normal, with the venous phase of the selective left T10 intercostal angiogram evident at 18 seconds.

Over a 3-month period the patient was treated by embolizations via the arterial route. There were three sessions; the Tracker-18 was used for all embolizations. The initial embolizations into the right T8 and T9 intercostal arteries were with mixtures of NBCA:lipiodol of 80:20 and 90:10, respectively. The high concentration of NBCA was used to shorten the polymerization time and avoid extensive uncontrolled venous embolization in these high-flow fistulas. There was no evident venous penetration of the embolic mixture. After the first embolization, the patient had substantial clinical improvement, but his symptoms returned after 2 weeks. The venous approach was then used, but catheterizing the epidural venous plexus from either the left or right femoral veins could not be accomplished. These attempts were made with the Tracker-18 through azygos, lateral sacral, and ascending lumbar veins. The arterial approach was again used, and in two sessions, the right T8 intercostal artery was embolized once, the T9 twice, and the T10 twice with a 67:33 mixture of NBCA:lipiodol. These vessels were embolized more than once because of incomplete closure by the liquid adhesive. The right T7 and T11 and the left T7, T8, T9, and T11 intercostal arteries were embolized with 150- to 250- μ m micron PVA particles (Interventional Therapeutics Corporation, San Francisco, CA). Angiography at the time of the last embolization showed minimal shunting at T9 on the right (Fig. 2F). After the third embolization, the patient's back pain and bladder dysfunction had resolved completely. He had minimal residual intermittent burning sensation in his left leg that was a deafferentation type of pain syndrome. The patient's physical examination is now normal, and he has remained stable over his 10-month follow-up period.

Discussion

Unlike the congestive myelopathy of spinal dural AVF (11), epidural fistula may produce a myelopathy because of the mass effect from large epidural veins. The severe radiculopathy in patient 1 may have been caused by the mechanical compression by the epidural plexus or could have been produced by inadequate venous drainage for the multiple nerve roots. In our patients, the intradural coronal veins did not participate in the shunts; thus, the spinal cord circulation time was normal (12). This result supports the work of Tadie et al, suggesting an "antireflux" mechanism in the radicular veins as they pierce the dura (13). This antireflux arrangement would prevent reflux into the intradural coronal venous system in the face of high pressure in the epidural veins. However, there are reports of AVF with both intradural and epidural venous drainage (5, 9, 10). Either these shunts crossed the dural boundaries, or the antireflux mechanism works to a variable degree.

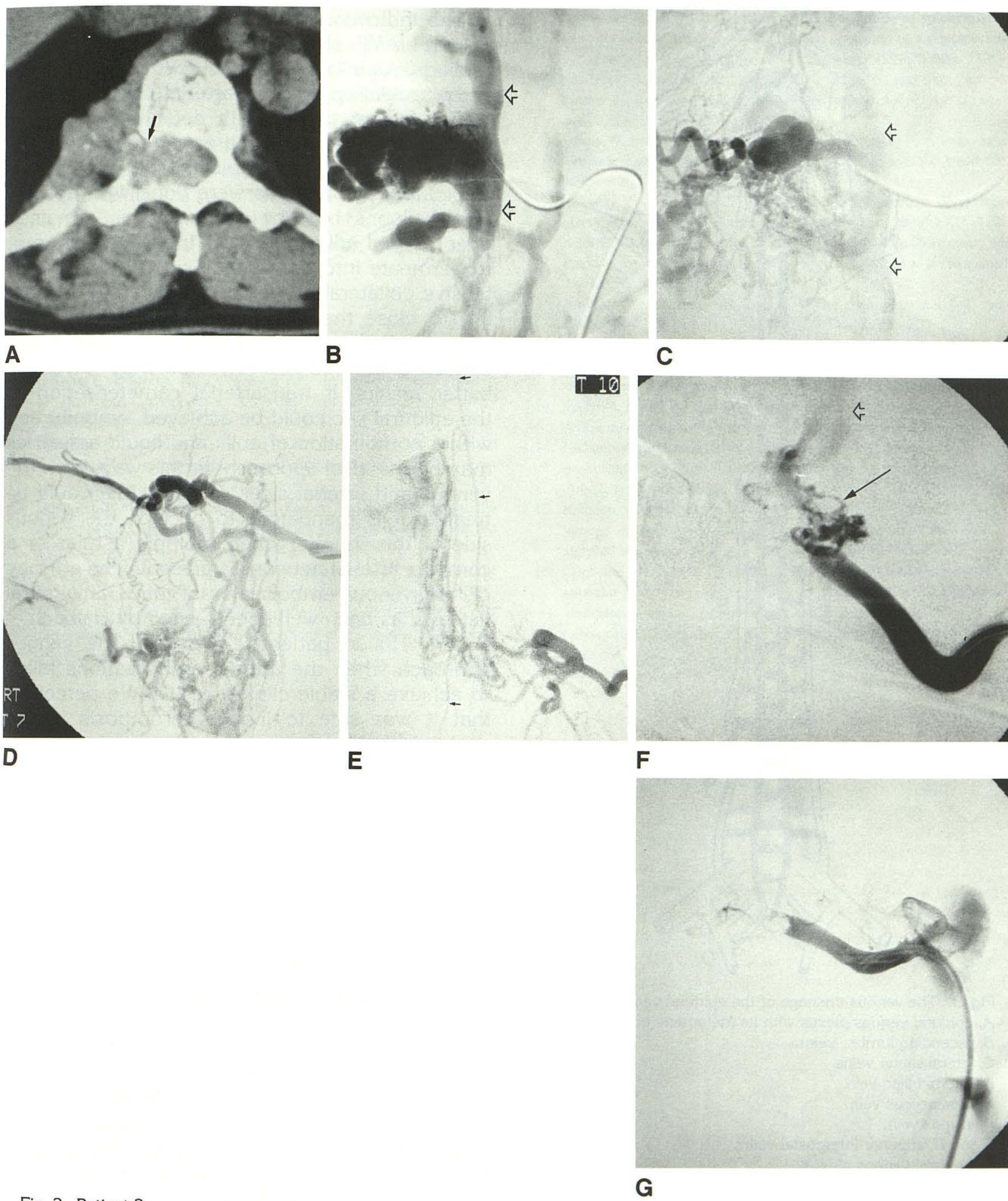


Fig. 2. Patient 2.

A, Axial CT at T9 shows an extradural-paraspinal mass (arrow) eroding the pedicle.

B through E, PA intercostal angiograms show multiple fistulas into the epidural venous plexus (*open arrows*) at T9 on the right (B), T8 on the right (C), and extensive collaterals from T7 on the right (D), as well as T10 on the left (E). Note the normal anterior spinal axis (*small arrows*) (E).

F and G, PA right T9 and T8 intercostal angiograms after arterial embolizations with liquid adhesives show a residual small fistula at T9 (*closed arrow*) (F) draining into the epidural vein (*open arrow*).

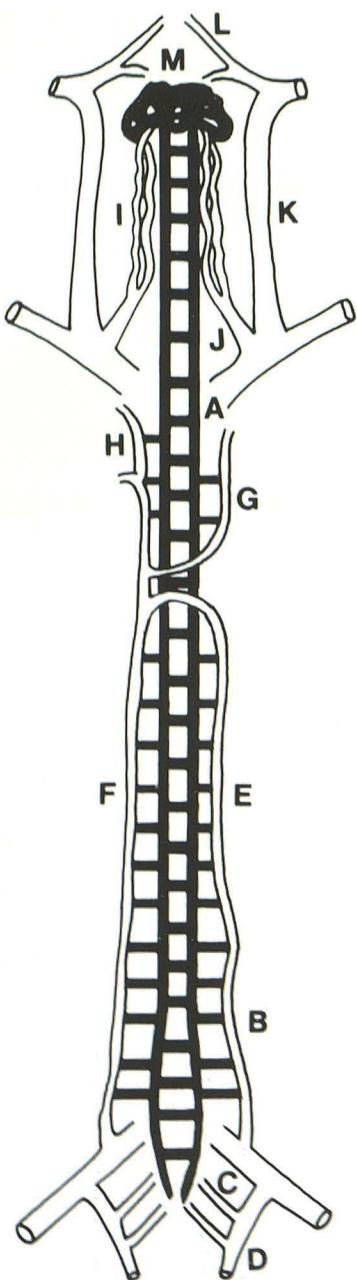


Fig. 3. The venous drainage of the epidural venous plexus.
 A, epidural venous plexus with its metameric connections.
 B, ascending lumbar veins.
 C, lateral sacral veins.
 D, internal iliac vein.
 E, hemiazygous vein.
 F, azygous vein.
 G and H, superior intercostal veins.
 I, vertebral venous plexus.
 J, vertebral vein.
 K, internal jugular vein.
 L, inferior petrosal sinus (connects with the suboccipital venous plexus through the anterior condylar vein).
 M, suboccipital venous plexus.
 Modified from Theron J. Exploration radiologique du système veineux intra-rachidien. *Encyclo Med Chir Paris Neurologie* 17032, G-10, 1979.

The indication for the treatment of a spinal epidural AVF should be based on the clinical findings. As with intracranial AVF, the preferred endovascular approach is arterial. Single-hole fistulas are best treated with detachable balloons with preservation of the parent artery. In our two patients, there were multiple fistulas. In patient 1, the initial embolizations were done with detachable balloons but the other fistulas remained open. Liquid adhesives were tried in an attempt to permeate into the venous side. However, extensive collaterals developed, indicating our failure to close the fistulas. Perhaps embolization with liquid adhesives by a "flow-arrest" technique instead of a "free-flow" method may have had better results. If transarterial catheterization of the epidural sac could be achieved, embolization with a combination of coils and liquid adhesives may be the ideal approach. In this way, both the venous and arterial sides of the fistula could be treated. It is mandatory to occlude the venous side of the shunt(s) when multiple fistulas or a complex arterial network is present. The efficacy of transvenous embolization of intracranial dural fistulas has been well documented by Halbach et al (14, 15). In patient 1, we used the venous approach when the arterial embolizations failed to achieve a stable clinical result. We perceived that it was safe to induce thrombosis in the epidural pouch because the pressure in the sac was 50 mm Hg and the patient's symptoms were progressive. Transvenous embolization of the intraspinal epidural veins produced a dramatic clinical improvement, indicating that a vascular mass produces symptoms by both high pressure and pulsation. This observation supports the rational and observed clinical improvement after intracranial coil embolizations of symptomatic giant arterial aneurysms (16).

The epidural venous system is a plexiform venous lake without valves that has metameric connections with the paraspinal longitudinal efferents (lateral sacral, ascending lumbar, azygous) and vertebral veins (Fig. 3) (17). In patient 1, the large vertebral vein allowed catheterization of the epidural plexus through a metameric connection. In patient 2, we were unable to catheterize the epidural plexus from the azygous, ascending lumbar, or lateral sacral veins. Transvenous catheterization is difficult because of valves and the small metameric connections. If the shunts are large, the venous side of the fistula may be accessible from a transarterial route.

In summary, epidural AVFs, having an exclusive epidural venous drainage, present as masses and are distinct from dural and intradural shunts. Endovascular therapy via an arterial approach is the initial treatment of choice. Occlusion of both the venous and arterial components of the fistulas is recommended. When necessary and feasible, the venous access may result in a dramatic clinical improvement and thus can be part of the treatment strategy of patients with this unusual condition.

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