

### Spinal Dural Arteriovenous Fistula: The Role of PVA Embolization

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The development of selective spinal angiography in the early 1960s permitted assessment of the vascular anatomy of spinal cord arteriovenous malformations (SCAVMs). Subsequently, these lesions were classified as type I—single-coiled vessel type; type II—Glomus type; and type III—juvenile type (1).

The type I single-coiled vessel malformations were recognized as slow-flow shunts, in extramedullary location on the dorsal surface of the spinal cord and extending over many vertebral levels without any distinct transition from arterial to venous elements. Men in the sixth and seventh decades were predominantly affected and presented with progressive radiculomyelopathy. Although Elsberg in 1914 successfully treated a presumed type I malformation by ligating and resecting a large vein at the dural opening of the T8 nerve root without recognizing the true pathology of this lesion (2), the recommended treatment has been long laminectomy with stripping of the single-coiled vessel from the underlying pia and spinal cord as suggested by Shepard (3).

Kendall and Logue in 1977 (4) and Merland et al (5) reported that most of these extramedullary “single-coiled vessel” malformations are arterialized intradural veins emerging from an extradural or dural arteriovenous (AV) fistula near a nerve root exit. With recognition of the true pathologic nature of these lesions, much of the confusion related to SCAVMs has been resolved.

Unlike SCAVMs, spinal dural arteriovenous malformations (SDAVFs) usually present in middle age (fifth to seventh decades) and over 80% are in males. The initial symptoms commonly are leg weakness, sensory disturbance and pain in the back or root distributions (6, 7). Sphincter dysfunction or impotence may be presenting symptoms. None of the documented SDAVFs presented with hemorrhage. The symptoms are, in general, slowly progressive with or without

remission. Step ladder-like deterioration or rapid progressive paraparesis is only occasionally observed. At the time of diagnosis, over 70% of patients have motor weakness, paresthesia, and bladder dysfunction.

The basic pathology of SDAVFs is an abnormal AV shunt on the surface of the dura at the level of an intervertebral foramen of the thoracolumbar spine (4–7). The shunts are usually single AV communications. However, a network of small arterial feeders may be present. A single tortuous draining vein emerges from the dura accompanying the dorsal nerve root to reach the perimedullary venous system of the cord. Manelfe et al (8) observed a network of tortuous capillary channels with an afferent artery and an efferent vein on the dorsal extradural surface of the spinal dura, which he called a “Glomerulus.” Although it shows characteristics of an AV communication, its physiologic role and relation to pathologic dural fistulae have not been proven.

It is generally accepted that the pathophysiology of SDAVFs is based on increased venous pressure in the coronal venous plexus and medullary veins (4–7). Anatomically, the venous system of the spinal cord is divided into intrinsic and extrinsic veins. Suh and Alexander in 1939 (9) reported the presence of monocuspid valves in the intrinsic venous system, similar to the valve-like structures of the radiculomedullary veins at the dural exit. These valves prevent transmission of increased intraspinal or systemic venous pressure to the spinal cord under physiologic conditions. It is still unclear how the reversal of flow develops in the radiculomedullary veins and the increased pressure in the coronal venous plexus affects the intrinsic circulation directly. Venous hypertension in the coronal plexus causes venous congestion and stagnation in the radial veins of the intrinsic system. This in turn reduces the AV pressure gradient resulting in diminished intra-

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medullary blood flow and ischemic myelopathy (10, 11). An interesting observation in SDAVFs is that there are fewer medullary veins throughout the spinal cord that are normally as numerous as the medullary arteries. Thus, venous hypertension affects multiple segments of the thoracolumbar spinal cord.

The therapeutic strategy is to reduce venous pressure by eliminating AV shunts. The surgical approach has evolved from stripping the "coiled vessel" to clipping a draining vein with coagulation or resection of an AV fistula (5-7, 12). To remove a nidus of an AV shunt, it is often necessary to perform duroplasty at the level of the lesion as demonstrated on spinal angiography.

Endovascular treatment of SDAVFs is as effective as surgery if the nidus and the draining vein are permanently occluded. Two types of embolic materials have been widely used to occlude AV shunts. One is particulate embolic agents, such as polyvinyl alcohol (PVA) foam or dura mater and the other is a liquid tissue adhesive, such as isobutyl-2-cyanoacrylate (IBCA) or *N*-butyl-cyanoacrylate (NBCA). As Nichols et al (13) and others (14, 15) have reported, a high incidence of recurrence was observed following particulate embolization of SDAVFs. The ineffectiveness of PVA embolization is due to several reasons. Proximal occlusion of the arterial feeders is a common occurrence. Although a draining vein may measure 0.5-1.5 mm (13), the actual AV shunt is smaller in caliber and it may be even microscopic, ranging from 50-60  $\mu$ m in certain cases. Therefore, the smallest particles of PVA cannot lodge in the fistula.

In addition, as Quisling demonstrated (16), the irregular surface of PVA creates high friction against the vessel wall so that particles stop without completely occluding it. This partial occlusion induces blood stagnation and thrombosis. This thrombus is eventually reabsorbed and recanalization occurs.

The presence of extensive microscopic collateral circulation along the dura is another reason for the recurrence (8). Even if particles are lodged at the arterial side of the AV shunt, the draining veins cannot be occluded since they are larger in caliber. The collateral vessels then enlarge and again feed the fistula.

The same phenomenon can occur after liquid embolization when the proximal segment of the draining vein is not occluded. The advantages of NBCA embolization over PVA are that it is possible to occlude the draining vein and the chance of recanalization is minimal.

The risks of particle embolization are not less as Nichols et al indicated (13). In the era of superselective catheterization, NBCA may be safer than PVA particles in experienced hands.

I strongly agree with the recommendations of Nichols et al (13) in treating SDAVFs. Endovascular treatment is less invasive than surgery, its morbidity is less, and it ensures earlier recovery for the patients. If embolization has failed, surgery can still be done. I believe PVA embolization does not have any role in treating SDAVFs.

Finally, I strongly recommend that patients be followed closely and aggressively. Periodic clinical and radiologic assessments, including MR and spinal angiography, are essential to achieve complete cure.

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