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Endovascular Techniques for Vascular Malformations of the Spinal Axis

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The purpose of this article is to discuss the use of endovascular techniques in the management of vascular lesions of the spinal axis. As with any other therapeutic modality, in deciding whether endovascular treatment is indicated, it is essential that the treating physician have a clear understanding of the pathologic entity in question, a clear understanding of the overall treatment plan, and a realistic understanding of the capabilities and limits of endovascular therapy. Without such an understanding, appropriate patients may be denied valuable treatment options and inappropriate patients may undergo procedures that are not in their best interest.

The use of percutaneous catheter-based embolization for treating conditions of the craniospinal axis began to be reported in the early 1960s, but applications were limited until improved devices began to become available in the 1970s and 1980s. Most important in driving this development was the introduction of variable stiffness "microcatheters" and, subsequently, suitable embolic materials. A variable stiffness catheter is a catheter that is stiffer at the proximal end than it is at the distal end. This design resulted in a catheter with a small soft tip that could be safely navigated within the vascular system much more distally than was previously possible. With these microcatheters, it became possible to access vascular territories percutaneously that otherwise could be reached only through direct surgical exploration. Indeed, it became possible to access vascular territories that may not normally be safely approached surgically. In turn, this improved access created the opportunity to deliver embolic agents in a "superselective" manner. It has been less than 20 years since high-quality variable stiffness microcatheters became readily available, but in this brief time, there has been rapid evolution of technology and techniques. These advances have allowed embolization to play a more prominent role in the treatment of many neurovascular conditions.

Endovascular anatomy

Spinal cord

The spinal cord receives its nourishment from one anterior spinal artery (ASA) and two posterior spinal arteries. The ASA lies in the anterior median sulcus, and the paired posterior spinal arteries lie on either side of the dorsolateral surface of the spinal cord. The numerous branches of the ASA travel within the anterior median sulcus and are responsible for providing supply to greater than two thirds of the spinal cord, including the anterior and lateral corticospinal tracts. The posterior spinal arteries primarily supply the posterior columns.

Anterior spinal artery

Paired ASAs originate at the craniovertebral junction as branches of the fourth segment of the

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vertebral artery. The takeoff of the ASAs is distal to the origin of the posterior inferior cerebellar arteries. The two ASAs then converge to form a single ASA ventral to the caudal brain stem or rostral spinal cord. The ASA then continues within the anterior median sulcus for the entire extent of the cord and conus medullaris. In the cervical region, the ASA receives further contribution via branches of the vertebral artery as well as the ascending cervical branch of the thyrocervical trunk. One anatomically constant radicular artery at the C5 or C6 level is known as the artery of cervical enlargement. Radiculomedullary branches of the supreme intercostal artery and the thoracic/lumbar radicular arteries supply the ASA in the thoracic and lumbar region. The most prominent of these radicular vessels is known as the arteria radicularis magna, more commonly referred to eponymously as the artery of Adamkiewicz. This artery commonly arises from the lower intercostal or lumbar arteries, most frequently on the left side between the levels of T10 to L2. It is a slender midline vessel measuring 0.5 to 1 mm in diameter. On an anterior-posterior projection, the artery has an ascending segment that makes a characteristic hairpin turn into a descending ASA located in the midline. Whenever possible, the artery of Adamkiewicz should be identified angiographically before embolization of middle/lower thoracic and rostral lumbar lesions.

Posterior spinal artery

A pair of posterior spinal arteries supplies the posterolateral aspect of the spinal cord. They originate in the upper cervical region, typically branching from the vertebral arteries. They receive supply from the segmental radiculomedullary arteries, which form a hairpin configuration similar to the supply of the ASA. The ASA anastomoses with the posterior spinal arteries at the conus medullaris to form a luxuriant arterial network.

Spinal column

Supply to the anterior and lateral aspect of the vertebral body is primarily via branches of the vertebral artery and the ascending cervical branch of the thyrocervical trunk in the cervical spine. In the thoracic and lumbar spine, branches of the intercostal arteries and lumbar radicular arteries, respectively, are the predominant arteries. The epidural spaces ventral and dorsal to the spinal cord and thecal sac contain a rich plexus of vessels. In the ventral epidural space, these vessels lie beneath the posterior longitudinal ligament and contribute to the vascularity of the vertebral body. In the dorsal epidural space, these vessels richly supply the lamina and a portion of the posterior spinous process. A plexus from the main trunk of the dorsospinal artery lines the outer surface of the lamina and the posterior spinous process.

Endovascular techniques

The ability to carry out high-quality spinal angiography is an essential prerequisite to the embolization of spinal lesions. High-resolution, preferably biplane, angiography is required. As with cerebral angiography, "subtraction" techniques are used to provide detailed images of vascular structures. Subtraction techniques require that a "mask" image is first acquired; then, after contrast injection, the original mask image is "subtracted" from the postcontrast images, resulting in greatly enhanced visibility of the contrast agent. When there is patient movement between the time of the initial or mask image and the time when the contrast-injected images are acquired, the mask no longer matches the new position and image quality is markedly degraded. Similarly, motion artifact can be problematic when catheters and devices are being maneuvered. When manipulating catheters and endovascular devices, progress is visually monitored using real-time subtracted fluoroscopic images known as "roadmaps." As long as there is no motion, these images remain clear, but even subtle movements render the roadmaps useless. Motion from breathing or intestinal peristaltic movements can also make the images uninterpretable. The importance of minimizing this motion artifact is further exaggerated when interventions are undertaken. It is crucial, for example, that there be no patient motion during the injection of an embolic agent, because any significant motion results in poor visualization of the embolic material being delivered. Spinal embolizations can be lengthy procedures, and it is difficult for the awake or sedated patient to remain adequately still. Because of these issues with patient motion, the authors prefer having their patients under general anesthesia for all but the most straightforward of spinal embolizations. Under general anesthesia, patient motion is largely eliminated. Likewise, ventilation can easily and reliably be suspended during key periods of image acquisition or during delivery of embolic agents. Glucagon can be administered to reduce intestinal motility, again

helping to minimize motion artifact and enhance image quality.

In the process of optimizing image quality, it remains equally important that patients and staff are protected from excessive exposure to radiation. Appropriate attention must be paid to radiation shielding, fluoroscopy time, and other issues relating to radiation exposure.

When endovascular neurosurgical procedures are performed with the patient under general anesthesia, many advocate using electrophysiologic monitoring to compensate for the inability to carry out clinical monitoring. Motor evoked potentials have been anecdotally reported to be particularly useful [1].

Microcatheters are categorized as one of two broad types. Catheters may be "over the wire" or "flow directed." Over-the-wire systems are advanced toward their target using a fine curved guidewire that is steered toward the target area using torque to direct the wire and then advancing the catheter over the wire. Flow-directed catheters are generally softer and more flexible. As the operator advances the proximal end of the catheter, the distal tip is carried forward passively with the arterial blood flow. Because of their dependence on flow, these catheters are best suited for use in the treatment of high-flow lesions, such as arteriovenous malformations (AVMs). These catheters are generally of smaller diameter and thus more restricted in their capacity to deliver embolic agents. The development of extremely fine guidewires (ie, 0.008-in diameter) has meant that to some extent, a flow-directed catheter can be delivered by flow but with some guidewire assistance. Within each class of catheter, there are a multitude of commercially available variations.

Embolic agents may be classified as liquid or particulate. Liquid agents may be "glue" type agents, such as N-butyl-cyanoacrylate (NBCA), or sclerosing agents, such as ethanol. Recently, ethylene vinyl alcohol copolymer dissolved in dimethyl sulfoxide (Onyx liquid embolic system; Micro Therapeutics, Irvine, California) has been reported in the treatment of spinal AVMs with good results [2]. This material is not yet approved for use in the United States. Particulate agents may be larger particulates, such as metallic coils (generally platinum), or smaller injected particles, such as polyvinyl alcohol. In general, the larger the particle is, the larger is the size of vessel that will be occluded. This may be desirable, or it may result in an occlusion more proximal to the target lesion than is desired.

Classification of spinal vascular malformations

Spinal vascular malformations are relatively uncommon, and the role of embolization varies depending on the type of malformation and the practice patterns of a given institution. Many different classification systems exist for spinal vascular malformations, but there remains widespread disagreement and confusion regarding nomenclature for these entities [3–11]. Recently, Spetzler et al [12] proposed an updated classification system clarifying the nomenclature and discussing a recently described and clinically distinct entity, the conus AVM. The conus malformation has also been discussed as a separate entity by Hurst et al [13]. The classification system proposed by Spetzler et al [12] is particularly helpful in that the nomenclature is clearly descriptive and replaces a bewildering array of overlapping and confusing names. Additionally, the classification system incorporates recent advances in imaging and new clinical insights enhancing understanding of these lesions, thereby permitting a more logical and easily understood framework. Box 1 from their publication lists the proposed replacement classification, whereas Tables 1 and 2 give the clinical summaries, respectively, for arteriovenous fistulas (AVFs) and AVMs as they occur in the spinal axis.

To summarize their classification, most vascular lesions relevant to this article are divided into AVFs or AVMs. Spinal aneurysms are separate

Box 1. Proposed classification of spinal cord vascular malformations by Spetzler and colleagues

- Neoplastic vascular lesions
  - Hemangioblastomas
  - Cavernous malformation
- Spinal aneurysms
- Arteriovenous fistulas
  - Extradural
  - Intradural
    - Ventral
    - Dorsal
- Arteriovenous malformations
  - Extradural-intradural
  - Intradural
    - Intramedullary
      - Compact
      - Diffuse
  - Conus

Table 1
Summary of clinical characteristics in arteriovenous fistulas

Characteristic	Extradural	Dorsal intradural	Ventral intradural
Pathophysiology	Spinal cord compression, venous congestion, vascular steal	Venous congestion, rare hemorrhage	Compression (venous aneurysm), hemorrhage, vascular steal
Presentation	Progressive myelopathy	Progressive myelopathy	Progressive myelopathy
Diagnostic modality	MRI, angiography	MRI, angiography	MRI, angiography
Previous nomenclature	Epidural	Dural AVF, long dorsal, type 1A, others	Types IVA (small), B (medium), and C (large), perimedullary

Abbreviation: AVF, arteriovenous fistula.

From Spetzler RF, Detwiler PW, Riina HA, Porter RW. Modified classification of spinal cord lesions. J Neurosurg (Spine 2) 2002;96:145–56; with permission.

but, of course, may exist in conjunction with high-flow spinal vascular lesions [14]. Fistulas may be extradural or intradural, and the intradural fistulas may be dorsal or ventral. AVMs may be intramedullary or combined extradural-intradural. The intramedullary lesions may be compact or diffuse. The conus AVM, as noted previously, is seen as a distinct entity. Again from this publication are included artist's illustrations of the AVFs and AVMs that afflict the spinal axis (Figs. 1–7) [12]. Information regarding the role of endovascular techniques in treating these lesions is considered below, grouped by lesion type. Discussion focuses on the AVFs and AVMs, because endovascular techniques are likely to play a key role in the treatment of these lesions. The rare spinal artery aneurysm that is not associated with a fistula or malformation may require surgical wrapping to ensure preservation of the artery [15].

Extradural spinal arteriovenous fistulas

Extradural spinal arteriovenous fistulas (ESAVFs) are rare. They are also known as epidural AVFs. Patients with these lesions most typically present with progressive neurologic deficits or pain. Symptoms may be secondary to direct compression of neural structures by the enlarged vascular channels or may occur as a result of venous hypertension or arterial steal [16]. Diagnosis is usually made by MRI or magnetic resonance angiography, but digital subtraction angiography is necessary for accurate characterization of the lesion.

ESAVFs may often be treated and cured by endovascular techniques alone. These lesions may have high flow rates. Endovascular closure of the fistula usually requires elimination of a small length of the distal feeding artery proximal to the fistula as well as the fistula itself and the proximal portion of the draining vein. To reduce the risk of the embolic material traveling beyond the target zone, it is preferable to use fibered detachable coils rather than liquid embolics. The disadvantage of using fibered coils is that larger catheters are required, and it may be more difficult to reach the target lesion with these larger catheters. Occasionally, a transvenous approach rather than a transarterial approach may facilitate endovascular access to the fistula [17].

Table 2
Summary of clinical characteristics in arteriovenous malformations

Characteristic	Extradural-intradural	Intramedullary	Conus medullaris
Pathophysiology	Compression, vascular steal, hemorrhage	Hemorrhage, compression, vascular steal	Venous hypertension, compression, hemorrhage
Presentation	Pain, progressive myelopathy	Acute myelopathy, pain, progressive myelopathy	Progressive myelopathy radiculopathy
Diagnostic modality	MRI, angiography, high-flow, multiple feeders	MRI, angiography	MRI, angiography
Previous nomenclature	Juvenile AVM, metameric AVM	Classic AVM, glomus type	None

From Spetzler RF, Detwiler PW, Riina HA, Porter RW. Modified classification of spinal cord lesions. J Neurosurg (Spine 2) 2002;96:145–56; with permission.

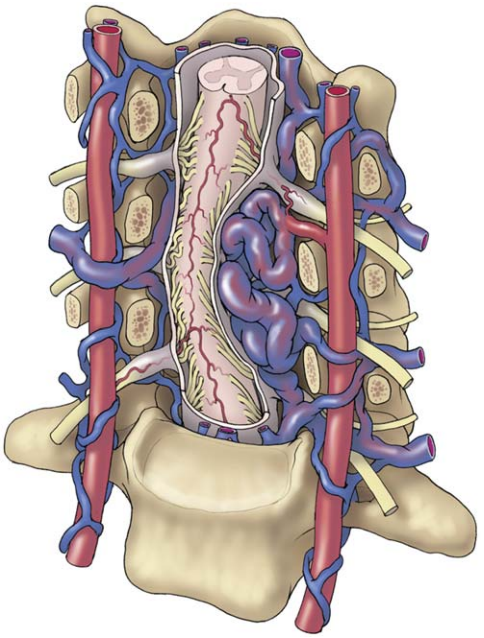

Fig. 1. Illustration of extradural arteriovenous fistula with thecal sac compression from venous engorgement. (*From* Spetzler RF, Zabramski JM, Flom RA. Management of juvenile spinal AVMs by embolization and operative excision: case report. J Neurosurg 1989;70(4): 628–32; with permission.)

Fig. 1 shows an artist's rendition of an ESAVF at the level of the cervical spine. Fig. 8 shows a clinical example of a large ESAVF identified in an 18-month-old girl after a vascular bruit was heard. This lesion was nicely demonstrated by magnetic resonance angiography. Coil embolization of the fistula resulted in angiographic cure of the lesion, elimination of the bruit, and continued normal neurologic function at the most recent follow-up 4 years after treatment.

Intradural dorsal spinal arteriovenous fistulas

These lesions have also been referred to as "spinal dural arteriovenous fistulas" or "type 1 spinal arteriovenous malformations." The treatment of intradural dorsal spinal arteriovenous fistulas (DSAVFs) is controversial. The urgency of this controversy is heightened because DSAVFs are well known as a preventable cause of paralysis, but it is also well known that the diagnosis is frequently delayed and that many patients present late to the neurosurgeon. Increased awareness and better imaging techniques have resulted in more

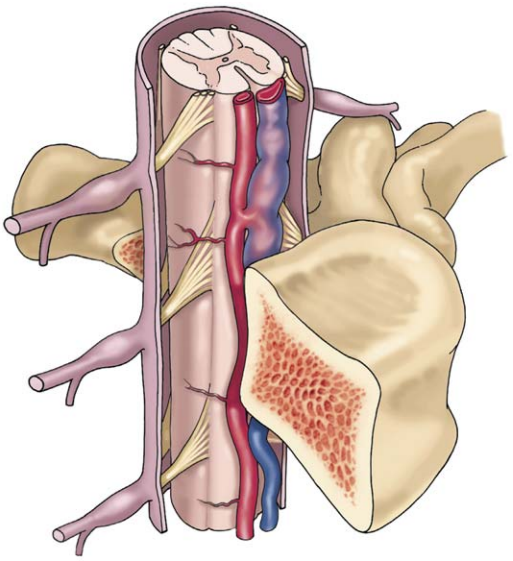

Fig. 2. Illustration of ventral arteriovenous fistula with significant venous engorgement. (Courtesy of Barrow Neurological Institute; with permission.)

frequent diagnosis of this problem. MRI and magnetic resonance angiography are proving highly useful in identifying the fistulas and documenting improvement of abnormal findings after successful treatment. For example, Lee et al [18] have shown that T2 imaging reliably shows signal hyperintensity in the spinal cord and that this hyperintensity diminishes after successful DSAVF treatment. They have also shown magnetic resonance angiography to be reliably diagnostic for DSAVFs.

As with intracranial dural AVFs, the key to the effective treatment of DSAVFs is the accurate identification of the AVF, followed by its complete and durable elimination. Whether this should be achieved by direct surgical obliteration or by endovascular embolization is at the core of the treatment controversy. If treatment is incomplete, DSAVFs are likely to cause a stepwise neurologic deterioration resulting in paralysis. The elimination of the fistula is likely to halt this progression; for this reason, cure rather than palliation must be the treatment goal. Indeed, with cure of DSAVFs, substantial neurologic recovery can occur, particularly if the duration of symptoms before cure is short [19]. At issue are the success rate, effectiveness, and durability of embolization as compared with open surgical treatment.

Careful analysis is required to understand the precise site of the arteriovenous connection,

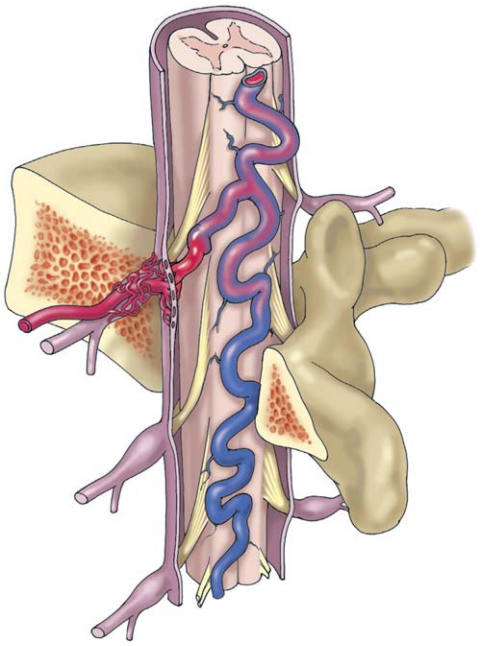

Fig. 3. Illustration of dorsal spinal arteriovenous fistula. The site of the fistula is intradural. (Courtesy of Barrow Neurological Institute; with permission.)

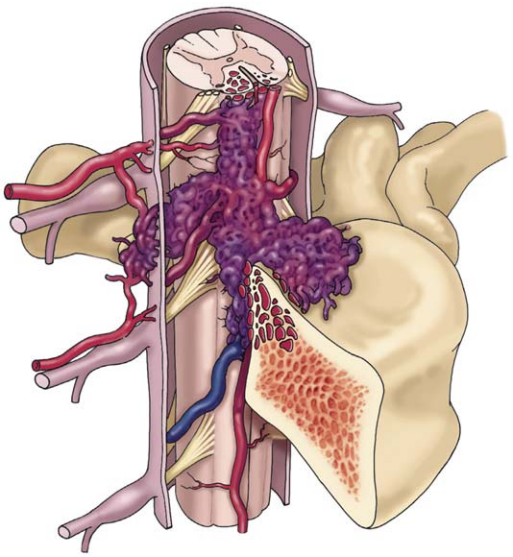

Fig. 4. Illustration of extradural-intradural arteriovenous malformation involving neural and bony elements. (*From* Spetzler RF, Detwiler PW, Riina HA, Porter RW. Modified classification of spinal cord vascular lesions. J Neurosurg (Spine 2) 2002;96:145–56; with permission.)

because recruitment of multiple feeding arteries and draining veins can present a confusing picture. Our understanding of the typical location of these fistulas was greatly enhanced by McCutcheon et al [20], who used microangiographic techniques on surgical specimens to demonstrate the characteristic anatomy of the lesions, specifically by demonstrating the intradural site of the AVF connection. Armed with this understanding and combining it with high-quality superselective angiographic techniques, these fistulas can be confidently identified, and percutaneous access to the fistula itself with a microcatheter is often straightforward. In this situation, embolization can be performed rapidly and safely. Similarly, superselective angiography through the microcatheter can also be valuable in identifying the spinal artery if it is associated with the arterial pedicle feeding the fistula. In this setting, embolization should not be used because of the risk of unintended embolization of the radiculomedullary arterial supply to the spinal cord. At other times, localization of DSAVFs can be challenging, particularly in elderly patients or patients with advanced atherosclerotic changes. In some instances, microcatheter access to the fistula is not possible. Large fistulas with multiple feeding arteries may also be difficult to treat endovascularly.

Higher rates of recurrence have been reported after treatment of DSAVFs by embolization compared with after treatment by open surgical obliteration. For this reason, some advocate embolization if it is technically feasible and reserve surgery for failure of or recurrence after embolization, whereas others advocate surgical obliteration as the primary modality of therapy. When a strategy of using embolization as the first line of treatment is used, published case series report DSAVF embolization cure rates of between 30% and 75%, depending on the series [15,21–25]. Song et al [26] reported that of 27 DSAVFs for which NBCA embolization was planned, treatment was technically feasible initially in 20 (75%) of 27 lesions. Of the 20 lesions actually embolized with NBCA, cure was achieved in 18, but recurrence of the fistula occurred in 3.

Despite the reported low morbidity of endovascular treatment for DSAVFs, some concerned about the risk of endovascular failure continue to advocate open surgical treatment as the primary mode of therapy [27–30].

Some of the recurrences of DSAVFs after embolization have been reported to occur because

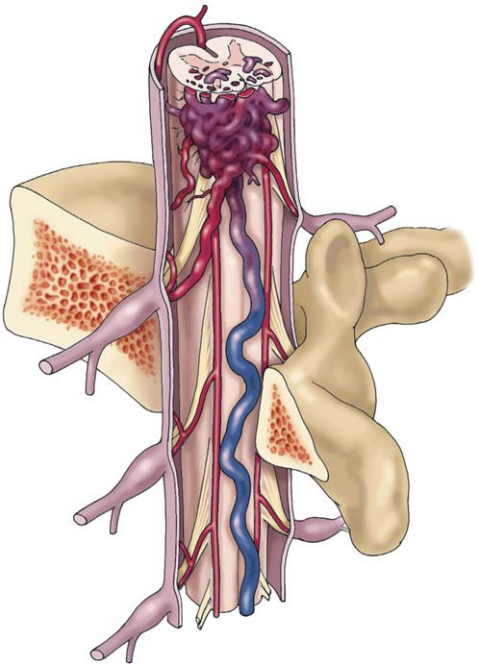

Fig. 5. Illustration of intramedullary compact arteriovenous malformation. Angiography is crucial in defining arterial supply. (Courtesy of Barrow Neurological Institute; with permission.)

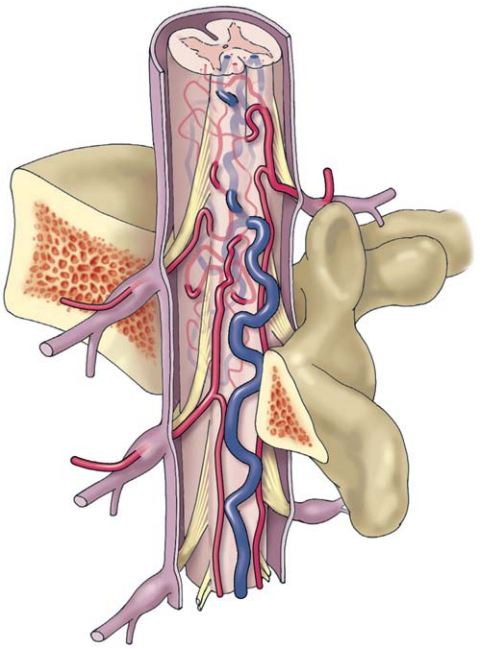

Fig. 6. Illustration of intramedullary diffuse arteriovenous malformation. (Courtesy of Barrow Neurological Institute; with permission.)

of partial embolization or embolization with particulate embolic materials. Partial embolization for treatment of DSAVFs is not helpful; in this regard, proximal embolization with particulate agents (eg, polyvinyl alcohol) is to be especially avoided [29,31]. Particulate embolizations carry the risk of proximal occlusion of the feeding arteries, obscuring the angiographic visualization of the fistula without improving the clinical condition. The fistula remains patent and the venous pressures pathologically elevated because the proximal feeding artery occlusion does not eliminate continued supply of the fistula through the rich collateral supply that typically exists more distally (ie, in the region of the fistula). Similarly, embolic agents that do not result in permanent occlusion of the fistula may permit recurrence of the condition. If endovascular treatment is to be considered, it is imperative that the fistula be thoroughly and permanently occluded. If any question in this regard persists, surgical exploration should be considered.

With liquid embolization materials, such as NBCA, DSAVFs can be treated effectively at the same session as the diagnostic angiography with little additional morbidity. Series reviewing endovascular treatment have found that NBCA embolization can be effective; however, as noted previously, there are treatment limitations. Adequate embolization must be achieved. "Adequate" embolization has been defined by Niimi et al [22] as (1) use of a liquid embolic material, (2) penetration of the embolic material to the fistulas or the draining vein, (3) angiographic disappearance of the fistulas or their drainage after embolization (determined by bilateral angiogram of two pedicles above and two pedicles below the level of the shunt as well as by angiography of the bilateral pedicles at the level of the shunt), and (4) no compromise of the venous drainage of the spinal cord after embolization. They reported adequate embolization in 87% of their patients after the introduction of variable stiffness microcatheters.

Fig. 9 illustrates a typical DSAVF treated endovascularly with NBCA, resulting in cure of the lesion.

Intradural ventral spinal arteriovenous fistulas

Intradural ventral spinal arteriovenous fistulas (IVSAVFs) are rare. This lesion was initially

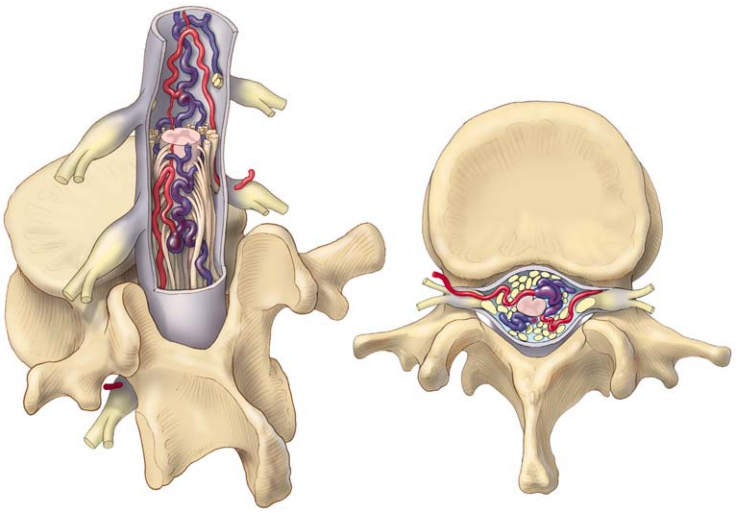

Fig. 7. Illustration of conus arteriovenous malformation. (*From* Spetzler RF, Detwiler PW, Riina HA, Porter RW. Modified classification of spinal cord vascular lesions. J Neurosurg (Spine 2) 2002;96:145–56; with permission.)

described by Djindjian et al [32] as an intradural extramedullary fistula. As the name implies, they are located ventral to the spinal cord, and their primary vascular supply is most often the ASA. They have previously been referred to as type IV lesions or perimedullary fistulas. These fistulas have been subdivided into three categories variously referred to as I, II, and III or a, b, and c in the literature, with the divisions being based on the volume of arteriovenous shunting [9,32–34]. They have been described in association with congenital syndromes, such as Rendu-Osler-Weber and Cobb's syndromes [9,32,35]. Clinical presentation is most often with hemorrhage, but patients may also present with progressive myelopathy, often secondary to compression from enlarged venous varices or venous hypertension [36].

Although IVSAVFs are rare, a clear understanding is important. Because there is no intramedullary component, there is a greater chance for cure and a lower treatment-related morbidity than is the case for intramedullary spinal arteriovenous malformations (ISAVMs). By contrast, the natural history poses significant risk for hemorrhage and neurologic disability. The involvement of the spinal medullary arteries can present major therapeutic challenges. While recognizing the critical vascular supply, these lesions can be accessed for endovascular treatment similar to an ESAVF. Again, because the ASA is frequently involved, the angioarchitecture must be well understood to be certain that closing the fistula will not compromise blood supply to the spinal cord distal to the fistula site. Excellent outcomes have been reported using embolization or surgical resection [36–38]. For larger lesions with relatively simple direct arteriovenous architecture, transvenous rather than transarterial catheterization is sometimes useful [35].

Extradural-intradural spinal arteriovenous malformations

Extradural-intradural spinal arteriovenous malformations (EISAVMs) are formidable lesions. They can rarely be cured without unacceptable morbidity [39]. They have been previously known by various names, such as type III, metameric, or juvenile AVMs. These lesions are characterized by the fact that they may involve multiple tissues (ie, they do not respect tissue boundaries). As a result, the AVM may extensively involve the neural elements, vertebral body, and adjacent cutaneous and soft tissues at the affected level. The moniker "metameric" stems from the fact that they are, in some instances, confined to a single segmental level. Fig. 3 is an illustration of this malformation.

As with other high-flow spinal lesions, symptoms may result from bleeding, direct compression, arterial steal, or venous hypertension [17]. Because these lesions are rarely curable, treatments are palliative and should be performed with

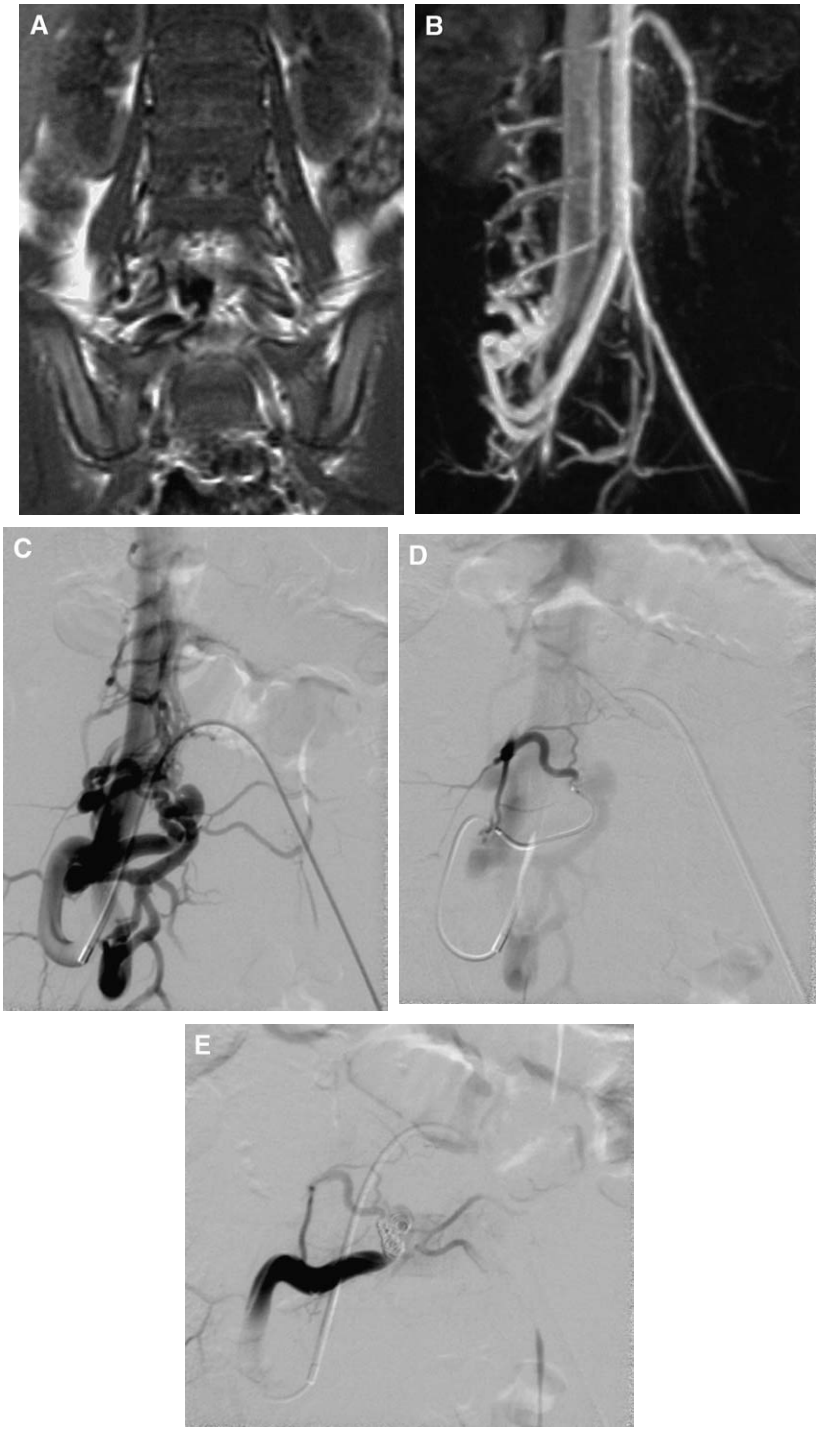

Fig. 8. (*A*) MRI and (*B*) magnetic resonance angiography demonstrate abnormally dilated extradural vessels in an 18-month-old girl. (*C*) Right internal iliac artery injection shows an extradural arteriovenous fistula. (*D*) The microcatheter is positioned on the venous side of the fistula. (*E*) Selective internal iliac artery injection after coil embolization of the fistula confirms its complete obliteration without venous shunting. The patient remains neurologically intact.

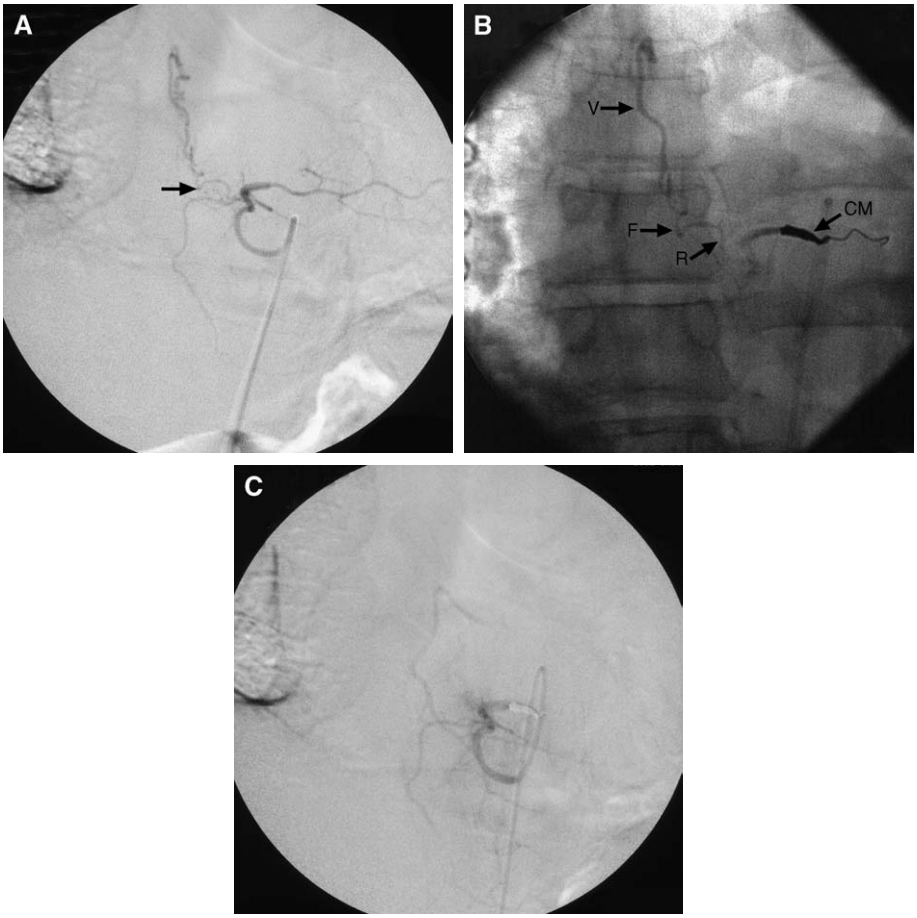

Fig. 9. (*A*) Intercostal artery injection in a 56-year-old man with progressive myelopathy shows an intradural dorsal arteriovenous fistula. (*B*) Unsubtracted image shows the coil mass (CM) within the intercostal branch. Glue is visible within the radiculomedullary feeder (R), fistula (F), and proximal vein (V). (*C*) Postembolization angiogram confirms complete obliteration of the fistula. The patient experienced immediate improvement in lower extremity paresthesias and weakness.

clearly identified goals in mind. The most straightforward situation is seen when symptoms and signs correlate well with a site of direct spinal cord compression by enlarged vascular structures. In this situation, the compressive lesion can be targeted with embolization to reduce or eliminate flow through this component of the malformation. This in itself may be adequate for the treatment goal or may be combined with subsequent surgical decompression. Fig. 10 illustrates a clinical example of an EISAVM. This 29-year-old man presented with progressive myelopathy. Angiography revealed an extensive AVM with large fistulas and high flow, resulting in marked engorgement of the draining veins. The arterial supply to the malformation was extensively embolized. This allowed surgical decompression of the compressing veins without concern for development of iatrogenic venous hypertension. After this staged combined therapy, the patient has enjoyed near-complete recovery from his myelopathy.

Intramedullary spinal arteriovenous malformations

ISAVMs have previously been known (among other names) as type II malformations and "classical" or "glomus" AVMs. The nidus may be discrete and compact, or it may be more diffuse. Angiography is essential to characterize the lesion and to identify potential associated aneurysms. These aneurysms are often identified as the source of subarachnoid hemorrhage in

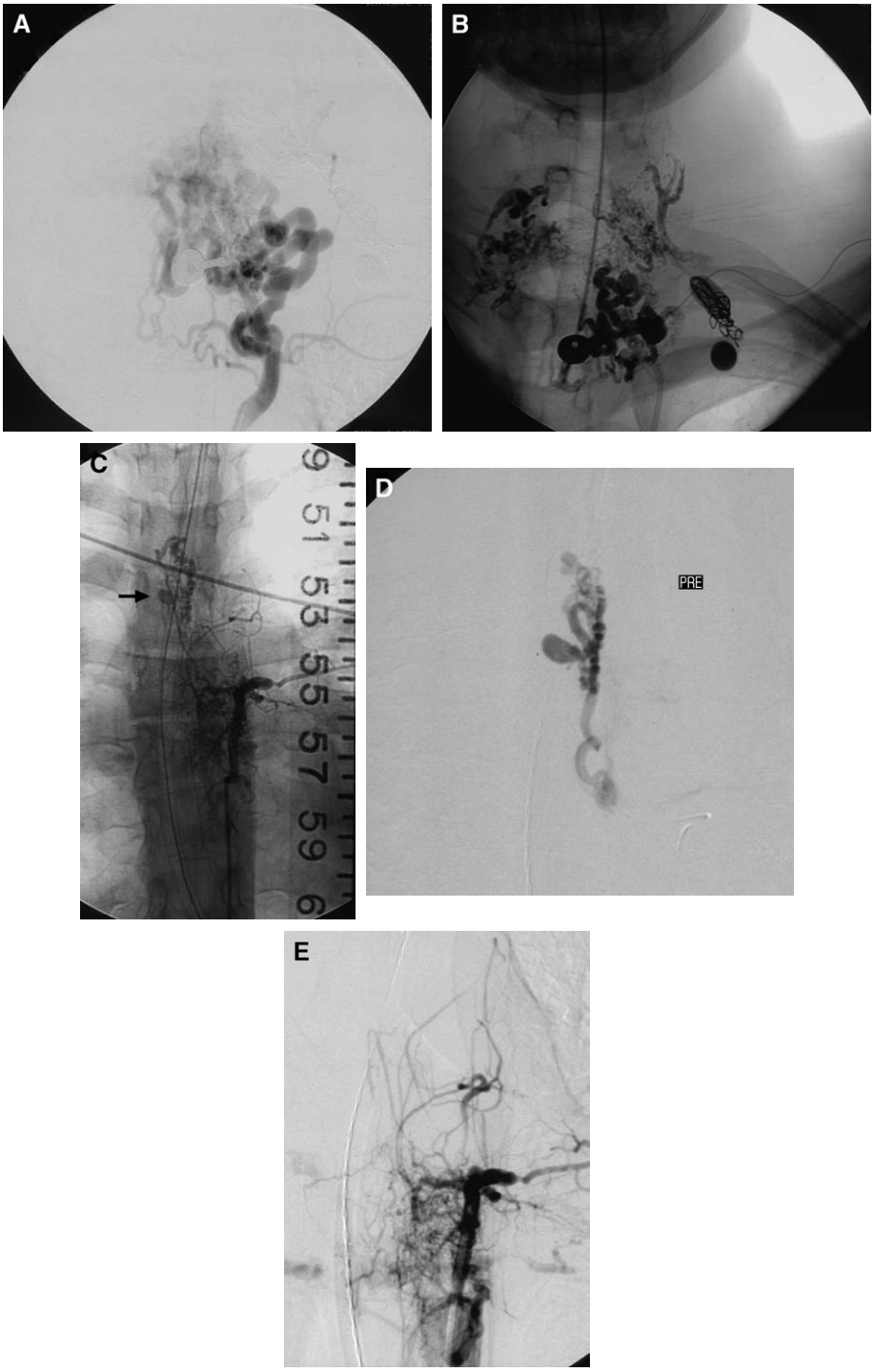

Fig. 10. (*A*) Angiogram shows a large cervicothoracic intradural-extradural (metameric) arteriovenous malformation (AVM) in a 29-year-old man. (*B*) Unsubtracted posterior-anterior view shows extensive coil and glue embolization of the anterior feeders and nidus. (*C*) Thoracic radicular injection shows residual AVM. (*D*) Microcatheter injection further defines the residual AVM and venous varix. (*E*) Posterior-anterior view confirms complete obliteration of the AVM and venous varix. The patient underwent complete resection of the AVM with improvement in myelopathy.

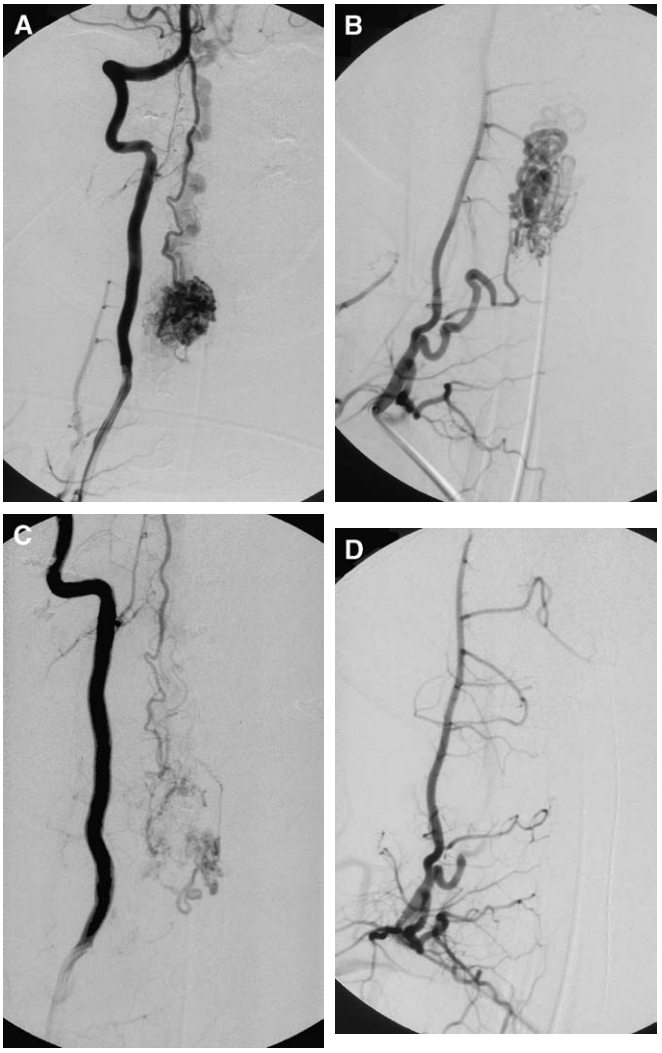

Fig. 11. (*A*) Right vertebral artery injection shows the blood supply to a diffuse intradural arteriovenous malformation (AVM) from the anterior spinal artery in an 18-year-old man with progressive weakness and myelopathy. (*B*) Thyrocervical trunk injection shows further blood supply via the ascending cervical and deep cervical branches. Postembolization vertebral (*C*) and thyrocervical trunk (*D*) injections show significant reduction in the arterial supply and size of the nidus. The patient underwent resection of the AVM, although a small anterior spinal artery feeder to the nidus remains.

patients with ISAVMs. When feeding artery aneurysms are identified, they should be seriously considered for treatment even if they have not produced hemorrhage. It has been reported that aneurysms on feeding arteries of spinal AVMs regress with treatment of the AVM itself in some cases [14,40].

Superselective catheterization is essential if embolization is to be undertaken. The ASA may be used to gain access to the malformation. The superselective angiogram must be carefully analyzed to ensure that no normal branches are put at risk. Embolization may be targeted to symptomatic components, such as ruptured feeding artery aneurysms, or may be used before surgery to facilitate surgical resection. Figs. 11 and 12 show examples of diffuse and compact ISAVMs involving the cervical spinal cord. Superselective

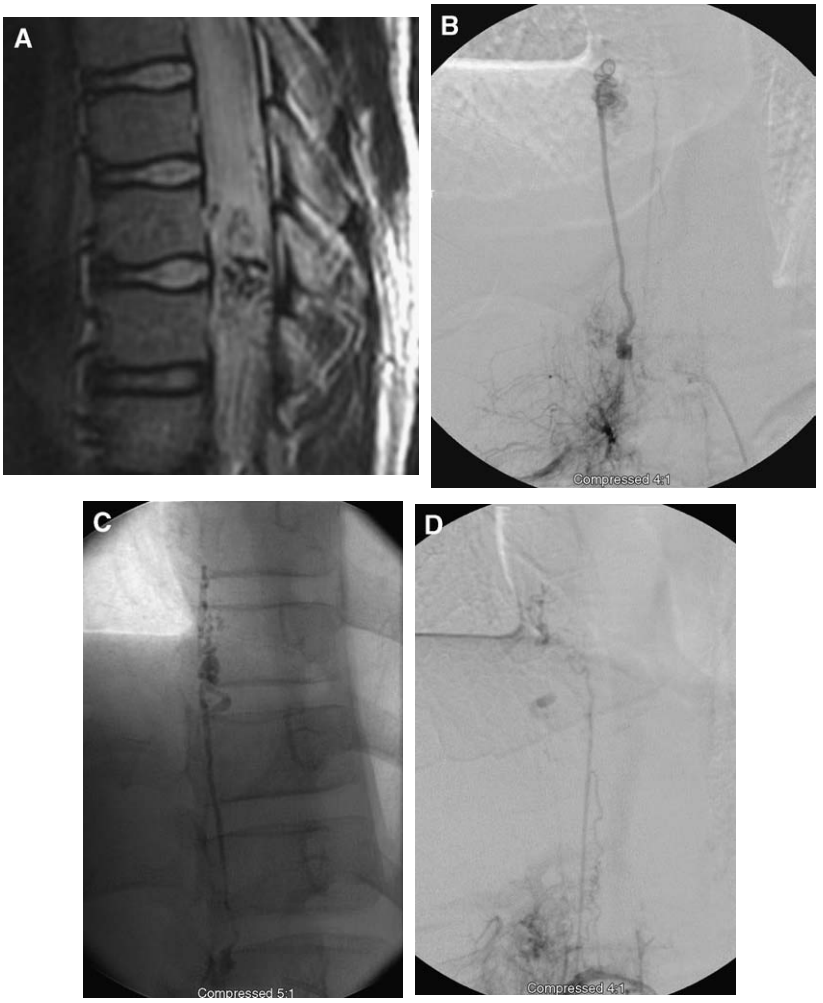

Fig. 12. (*A*) MRI shows a compact intradural arteriovenous malformation (AVM) at T9 to T10 in a 13-year-old boy with a gait disturbance. (*B*) Selective injection of the right T12 radicular branch shows the compact nidus. (*C*) Unsubtracted posterior-anterior view shows the glue cast within the arterial feeder and nidus. (*D*) Postembolization angiogram confirms minimal residual opacification of the AVM. The AVM was completely excised without complication.

angiography demonstrates typical positioning of the catheter before embolization. Recently, Molyneux and Coley [2] have reported excellent angiographic results in two cases treated with the new liquid embolic agent ethylene vinyl alcohol copolymer, which is delivered dissolved in dimethyl sulfoxide (Onyx).

Conus medullaris arteriovenous malformations

Conus medullaris arteriovenous malformations have been described as a distinct lesion by Hurst et al [13] and Spetzler et al [12]. This lesion may present like other conus lesions with combinations of myelopathy and cauda equina symptoms. A combination of a conus-based intramedullary AVM is seen together with multiple large direct AVFs [12]. This lesion is illustrated in Fig. 7. The extensive nature of these malformations makes them difficult to cure by embolization alone, but most flow may be eliminated by embolization, thereby greatly enhancing the chance for cure by surgical resection. Fig. 13 demonstrates a conus malformation embolized to an angiographic cure. This 47-year-old man presented with subarachnoid

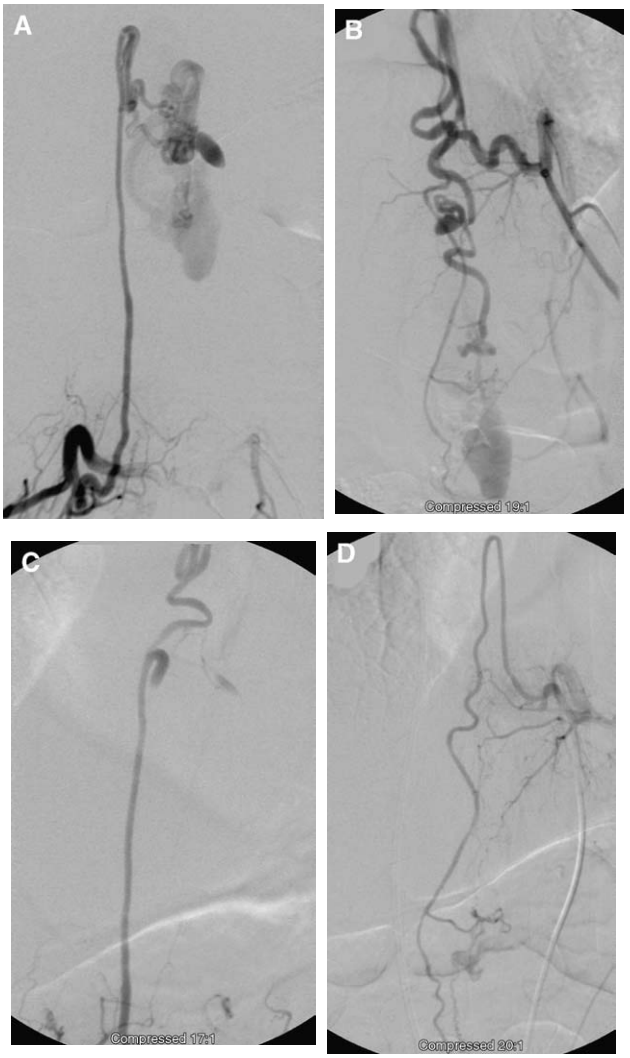

Fig. 13. (*A*) Right L2 injection shows the presence of a complex conus medullaris arteriovenous malformation (AVM) in a 47-year-old man with a history of subarachnoid hemorrhage and myelopathy. (*B*) Left T10 selective injection also shows the blood supply to the conus AVM. Postembolization right L2 (*C*) and left T10 (*D*) injections show almost complete obliteration of AVM supply. The patient underwent complete resection of the AVM. He is ambulatory and has retained bowel and bladder function.

hemorrhage. After embolization, surgical exploration and resection were performed. Interestingly, despite the postembolization impression of angiographic cure, components of patent malformation were identified at the time of surgical resection. For this reason, it may reasonable to consider surgical exploration even after the achievement of excellent embolization. Practically speaking, in most cases, embolization for this lesion is incomplete, and surgical excision should be performed after embolization. In most instances, cure of the lesion is possible and should be considered the treatment goal.

Summary

Endovascular techniques have evolved remarkably in the past 20 years since the advent of the variable stiffness microcatheter. Advances in imaging, embolic materials, and the ability to deliver

embolic agents safely have facilitated dramatic improvements in our ability to care for patients with spinal axis vascular lesions. Even more important in this work has been the accompanying growth in our understanding of these lesions. It is hoped that continued progress will lead to even greater improvements in our ability to care for patients with these conditions.

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