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Spinal Arteriovenous Fistulas and Arteriovenous Malformations – Complicated Vasculature and Surgical Imaging

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1. Introduction

Spinal arteriovenous shunts (AV shunts; including spinal AV fistulas and AV malformations) are heterogeneous entities that can render devastating neurological sequelae by hemorrhage (hematomyelia, subarachnoid hemorrhage, and subdural/epidural hematoma), venous congestion, mass effect, and vascular steal [1-5]. These lesions have been challenging entities to treat because of their complicated vasculature and the high-vulnerability of the spinal cord.

In the absence of accumulated knowledge of the pathophysiology of each entity, early classifications were based on the anatomical characteristics, which could be confusing [6-10]. With evolution of imaging technology such as various MR imaging, CT angiography and selective spinal angiography, ability to examine the angioarchitecture of these lesions has improved significantly. In addition, intraoperative diagnostic modalities have been developed that aid in open microsurgery before, during, and after the resection of these lesions. Increased knowledge of the angioarchitecture and pathophysiology of spinal AV shunts has led to the development of a multidisciplinary approach to these lesions.

This article, after the description of spinal vessel anatomy and some practical classification schemes, follows the recent advancement of spinal vascular imagings and intraoperative diagnostic tools for open microsurgery, which are indispensable for the effective treatment of spinal complex vascular lesions.

2. Spinal vasculature

To understand the pathophysiology of spinal AV shunts, a profound knowledge of spinal vessel anatomy is indispensable.

2.1. Segmental spinal arteries

Blood supply to a metamere that consists of vertebral body, paraspinal muscles, dura, nerve root and spinal cord is derived from segmental arteries, that are present in the fetus for each metameres (the 31 spinal segments). The segmental arteries potentially supply all the tissues on one side of a given metamere. Each metamere is centered at the level of the intervertebral disc, and each vertebra is therefore supplied by two consecutive segmental arteries on each side which build sufficient transverse and longitudinal anastomoses. These extra- and intraspinal anastomoses provide excellent collateral circulation, and protect the spinal cord against ischemia. The segmental arteries course along the vertebral body posteriorly, supplying blood to the vertebral body through perforating arteries. While the muscular branches run further posteriorly to the segmental muscles, the spinal branches enter the spinal canal through the intervertebral foramen and divide into the following three branches: the anterior and posterior branches which supply blood to the bony structure, and the radicular artery which supply blood to the dura and the nerve tissue at every segmental level [11].

Lasjanias *et al.* proposed a useful classification of the radicular arteries based on their region of supply, i.e., radicular, radiculopial and radiculomedullary [12]. The first type of radicular artery is a small branch, present at every segmental level, whose supply is restricted to the dura and the nerve root. The second type of radiculopial artery supplies blood to the nerve root and superficial pial system, including the posterior spinal arteries. The third type of radiculomedullary artery supplies the nerve root, superficial pial system, and the anterior spinal artery. Although the anterior and posterior spinal arteries are connected with the superficial pial collateral system, this classification provides a reasonable basis for planning treatment. In fact, a given segmental artery may connect to both the anterior and posterior arteries. These three types of the radicular artery are classified in terms of their contribution to each spinal artery.

Whereas each radicular artery supplies blood to the spinal cord in the embryo, the number of radicular arteries supplying the spinal cord decreases following a transformation and fusion in the postnatal life [13]. At certain segmental levels, the radicular artery has a persistent supply to the spinal cord that reaches either the anterior surface via the ventral nerve root or the posterolateral surface via the dorsal nerve root. Between two to 14, (on average, 6), radiculomedullary arteries persist as the result of this ontogenic reduction in feeding arteries. The radiculopial arteries are also reduced to fewer than 11 and 16 arteries.

2.2. Arteries of the spinal cord

The anterior spinal artery, with a diameter of 0.2–1.0 mm, traverses along the anterior median sulcus and typically originates from the intracranial portion of the vertebral arteries. The

posterior spinal arteries, with a diameter of 0.1–0.4 mm, originate from the preatlantal part of the vertebral artery or from the posterior inferior cerebellar artery (PICA). These arteries course from the cervical spine to the conus medullaris but are not capable of feeding the entire spinal cord. They are reinforced at various segmental levels by the radiculomedullary/radiculopial arteries [12]. The blood flow derived from these segmental arteries may be both caudocranial and craniocaudal. The most well-known radiculomedullary artery is the artery of Adamkiewicz (arteria radicularis magna), which arises close to the thoracolumbar enlargement. On the entire surface of the cord, both anterior/posterior spinal arteries anastomose via an extensive pial network (vasacorona). At the level of the conus medullaris, the posterior spinal arteries are connected to the anterior spinal artery through the anastomotic semicircles [11].

The intrinsic arteries of the spinal cord can be divided into two perforating systems—the sulcal (central, sulco-commissural) arteries originate from the anterior spinal artery, and the perforating rami arise from the vasacorona. Sulcal arteries, with a diameter of 0.1–0.25mm, are centrifugal and supply the largest part of the gray matter. They penetrate into the parenchyma through the anterior median fissure, course to one side of the cord and branch mainly within the gray matter. These arteries can anastomose via the transmedullary arteries with the deep perforating arteries from the vasacorona. Numerous perforating rami, with a diameter of up to 0.05 mm, penetrate the white matter, forming a centripetal system [11].

2.3. Spinal venous system

The venous drainage from the spinal cord comprises the small superficial pial veins that open into the superficial longitudinal veins. On the surface of the cord, blood accumulates in essentially two longitudinal veins—the anterior and posterior spinal veins. The anterior spinal vein is located under the anterior spinal artery in the subpial space. The posterior spinal vein, located in the subarachnoid space perimedullarily, takes a course independent of the posterior spinal arteries. The perimedullary venous system is more variable in course, size, and localization than the arterial system. Intraparenchymal transmedullary venous anastomoses may be present; these midline anastomoses, 0.3–0.7mm in diameter, connect the anterior and posterior spinal veins while receiving no tributaries from the intrinsic spinal cord veins [14].

The perimedullary venous collectors drain into the epidural venous plexus through the radicular veins. The transition from the perimedullary vein to the radicular vein forms a hairpin course, similar to the arterial configuration. Drainage of blood from the spinal cord is directed to the epidural plexus without retrograde flow. Though there is no valve structure in the spinal venous plexus, the nature of this reflux-impending mechanism is a matter of dispute. Some histological studies showed that the anatomical features act as an antireflux device—a substantial narrowing and bending of the vessels at its transdural course [14, 15]. These characteristics result either from the close proximity of a nerve root or from the presence of a bulge of dural collagenous fibers with a glomus-like appearance, which were supposed to act an antireflux mechanism physiologically.

The epidural venous plexus extends as a continuous system from the sacrum to the skull base and is located within the fatty and fibrous tissues of the epidural space. This valveless system

is connected with the azygos/hemiazygos venous systems in the thoracolumbar region, and with the vertebral and deep cervical veins in the cervical region.

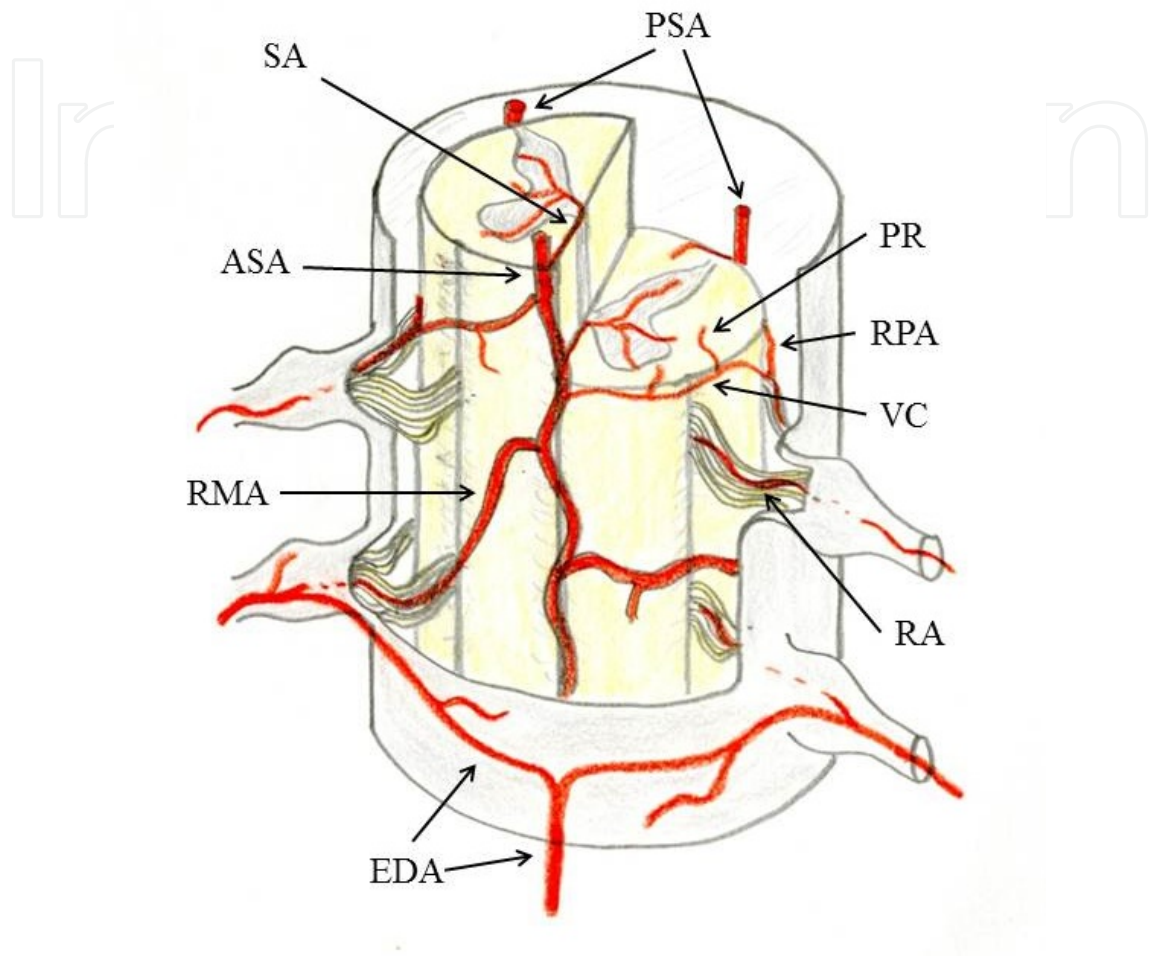


Figure 1. Arteries of the spinal cord. ASA: anterior spinal artery, PSA: posterior spinal artery, RMA: radiculomedullary artery, RPA: radiculopial artery, RA: radicular artery, SA: sulcal artery, VC: vasacorna, PR: perforating ramus, EDA: epidural anastomosis.

3. Classification of spinal AV shunts

3.1. Previous classifications

Many different classifications have been proposed for spinal AV shunts in terms of their location, vascular structure, and pathophysiology [16-23].

Rosenblum *et al.* classified spinal AV shunts into four types, on the basis of their angioarchitecture and clinical manifestations (Table 1) [16]. This simple classification is reasonable and has been widely used. However, it is hard to distinguish between type II (glomus-type

malformations) and type III (juvenile-type malformations) in various preoperative examinations. The difficulty of treatment, prognosis, and onset pattern were not reflected in this classification.

Type I: Dural AV fistula
Type II: Glomus-type intradural AV malformation
Type III: Juvenile-type intradural AV malformation
Type IV: Intradural AV fistula
a : simple extramedullary fistulas fed by a single arterial branch
b : intermediate-sized fistulas with multiple, dilated arterial feeders
c : giant multipediculated fistulas

Table 1. Classification by Rosenblum *et al.*

Spetzler *et al.* advocated a classification system dependent on radiological and intraoperative findings, which closely reflects the difficulty of treatment (Table 2) [17, 18]. This classification, mainly intended as an aid to the open surgery, was noted for its anatomical detail of the shunts and a new categorization of “conus medullaris AV malformations.” Unfortunately, different pathologies are ascribed to the same group in this classification, which can create confusion. For example, spinal dural AV fistulas and fistulous spinal cord AV malformations on the dorsal surface of the spinal cord belong to the same category (intradural dorsal AV fistulas).

AV fistulas
Extradural
Intradural
Ventral (small shunt, medium shunt, large shunt)
Dorsal (single feeder, multiple feeders)
AV malformations
Extradural-intradural
Intradural
Intramedullary
Compact
Diffuse
Conus medullaris
Neoplastic vascular lesions
Hemangioblastoma
Cavernous malformation
Spinal aneurysms

Table 2. Classification by Spetzler *et al.*

Rodesch *et al.* classified spinal AV shunts into the following three groups from genetic and hereditary points of view (Table 3) [19, 20]: (1) Genetic hereditary lesions that are caused by a

genetic disorder affecting the vascular germinal cells. Spinal cord malformations associated with hereditary hemorrhagic telangiectasia (HHT) fall into this category. (2) Genetic nonhereditary lesions (such as somatic mutations) that share metameric links, including the Cobb syndrome, affecting the whole myelomere. Patients typically present with multiple shunts of the spinal cord, nerve root, bone, paraspinal muscle, subcutaneous tissue, and the skin. Klippel-Trenaunay and Parkes-Weber syndromes also belong to this group. (3) If there is no evidence of genetic disorder, a lesion is assumed to be a single lesion. It is likely that some of these isolated, apparently sporadic lesions may represent incomplete phenotypic expressions of an underlying, undiagnosed genetic or segmental syndrome. This classification reflects the important embryologic aspects of AV shunts; however, most cases (>80%) fall into the last group, which is not advantageous for treatment.

Genetic hereditary lesions : macrofistulae and hereditary hemorrhagic telangiectasia
Genetic nonhereditary lesions : all of which were multiple lesions with metameric or myelomeric associations
Single lesions : which could represent incomplete presentations of one of the above groups

Table 3. Classification of Rodesch *et al.*

Similar to pathology of the brain, spinal AV shunts can be differentiated into dural AV fistulas and spinal cord pial (intraparenchymal) AV malformations, depending on the shunt location and its related angioarchitecture [16, 24].

Spinal dural AV fistulas are fed by the radicular arteries and/or the surrounding vertebral branches, whereas spinal cord pial AV malformations are, similar to their cerebral counterparts, fed by the perimedullary and/or intrinsic arteries of the spinal cord. Spinal cord pial AV malformations may be either of the fistulous type, consisting of a direct connection between artery and vein, or of the nidus type, with an intervening network of vessels. Fistulas are further subdivided into micro- and macrofistulas. Nidi are further subdivided into glomus and juvenile types. Generally, dural AV fistulas are considered an acquired pathology, whereas pial AV malformations are presumed to be inborn vascular lesions.

The term “angioarchitecture” not only describes the morphology of the lesion at a given time, but also places the lesion in a temporal sequence. Most spinal AV shunts induce changes over time; marked venous ectasias may develop, and additional arterial feeders may be recruited. Venous thrombosis may induce spontaneous regression of the lesions. The type of shunt may remain fixed, but some fistulas may approach the nidus-type of malformation with time as a result of extensive pial reflux or intense intrinsic network congestion. The type of shunt implies the clinical presentation, treatment, and prognosis [20].

3.2. Spinal dural AV fistulas

Spinal dural AV fistulas account for 70% of all spinal AV shunts. Men are affected approximately five times more often than women. The disease usually becomes symptomatic in the

elderly population. Most fistulas are located in the thoracolumbar region. Multiple fistulae in the same patient are exceedingly rare [25, 26].

Recently, both cranial and spinal dural AV fistulas have been categorized into ventral, dorsal, and lateral groups on the basis of the embryologic development of the venous drainage [27].

The ventral group consists of dural AV fistulas into those veins that normally drain the structures developed from the notochord (i.e., the vertebral body at the spinal level). These veins are known as the basivertebral venous plexus, which subsequently drains into the anterior internal vertebral venous plexus, located in the ventral epidural space of the spinal canal, which joins the basilar venous plexus and cavernous sinus. They may become symptomatic as a result of compression of the spinal cord or nerve roots by the enlarged epidural venous pouches. Because of the antireflux mechanism, these shunts do not induce the venous congestion of the spinal cord. There have been only a few case reports describing associated perimedullary reflux as a cause of congestive myelopathy. A hypothesis about a possible defect in the valve-like mechanism that normally impedes retrograde flow from the epidural plexus to perimedullary veins has been suggested to explain this finding.

The dorsal group of dural AV fistulas is related to veins that normally drain the spinous process and lamina at the spinal level. Although they are related to the major dural venous sinuses (superior sagittal sinus, torcular herophili, and transverse sinuses) at the cranial level, the corresponding veins at the spinal level are poorly developed and consist of a pair of longitudinal channels (i.e., the posterior internal venous plexus). Patients with dural AV fistulas within this space typically present with spontaneous epidural hematomas. These symptomatic lesions are extremely rare.

The most common “classic” type of spinal dural AV fistulas is categorized as the lateral group. This type represents >90% of all spinal dural AV fistulas that develop in the lateral epidural space at the junction of the veins that connect the spinal cord drainage to the epidural venous system. The fistula is located at the dura mater close to the spinal nerve root, where a radicular artery enters a radicular vein. Obstruction of its adjacent venous outlet, as a result of thrombosis or fibrosis related to aging, will lead to reflux into the perimedullary veins. Increase in spinal venous pressure diminishes the arteriovenous pressure gradient that leads to decreased drainage of normal spinal veins and venous congestion with intramedullary edema. This in turn leads to chronic hypoxia and progressive myelopathy. As a result, patients within this group present with aggressive clinical symptoms and at an older age. A strong male predominance is also observed, which is similar to that seen in the cranially located lateral AV fistulas, such as in the foramen magnum (medulla bridging vein) and tentorial (petrosal bridging vein) locations.

Symptoms of congestive myelopathy are unspecific. Usually, the deficits progress gradually; however, acute disease onset and progressive development interrupted by intermediate remissions is also possible. Mass effects and hemorrhagic changes are rare as presenting symptoms. Without therapy, the prognosis of this disease is grim, because it results in irreversible motor weakness, sensory disturbance, and bladder-bowel dysfunction [25, 26].

There are two options in the treatment of spinal dural AV fistulas; surgical occlusion of the intradural reflux vein, and endovascular therapy employing embolic material into the fistula. Surgery is a relatively simple and safe intervention, resulting in long-term shunt occlusion in

98% of cases [28]. In endovascular therapy, the embolic glue material must pass the fistula and occlude the proximal segment of the draining vein to prevent subsequent intradural collateral filling of the fistula. The success rates of endovascular therapy have been reported to vary between 25% and 75% [29-31]. Following complete occlusion of the fistula, the progression of the disease may cease; however, only two-third of all patients show regression of their motor weakness and only one-third show an improvement in sensory disturbances. Impotence and sphincter disturbances are seldom reversible [26].

3.3. Spinal cord pial AV malformations

Approximately 20%–30% of all spinal AV shunts are spinal cord pial AV malformations [16, 24, 32]. Similar to brain AV malformations, they are fed by arteries of the spinal cord and drained by spinal cord veins. These high-flow shunts might be intra- and/or perimedullary in location and can be differentiated into fistulous, glomus, and juvenile types, according to their shunt type and hemodynamic flow pattern. Fistulous and glomus types are often present within different compartments of the same AVM.

Fistulous AV malformations (“perimedullary AV fistulas”) are direct AV shunts located superficially on the spinal cord and can possess intramedullary compartments. The shunt may be supplied by the anterior or posterior spinal artery, while the draining veins are the superficial perimedullary veins. This type can be subdivided into three types depending on the size of the feeding vessel, the volume of the shunt, and the drainage pattern [21]. Type I fistulas are small AV malformations; neither the feeding artery nor the draining vein is dilated, and the shunt volume is low. Type II fistulas are medium-sized AV malformations fed by one or two dilated arteries, whereas type III fistulas harbor multiple massively dilated arterial feeders and have a large shunt volume.

Glomus AV malformations have a nidus closely resembling that of a brain AV malformation and usually having an intramedullary location. The superficial nidus compartments may also reach the subarachnoid space. Because of the many anastomoses between the anterior and posterior arterial feeding systems of the spine, this type is typically fed by multiple arteries derived from both the anterior and the posterior systems. Drainage is into dilated vessels of the spinal cord.

Juvenile AV malformations are typically large with both fistulous and glomus compartments not only involving the spinal cord but also neighboring tissues such as the dura, vertebral body, and paravertebral muscle.

Venous congestion, hemorrhage, space-occupying effects and vascular steal have been attributed to the pathogenesis of spinal cord AV malformations. If the AV malformation does not present initially with an acute hemorrhage, symptoms are unspecific. The glomus type tends to become symptomatic in younger children and adolescents, whereas the fistulous type becomes symptomatic in young adults [16, 31].

The first attempt at treatment of spinal cord pial AV malformations tends to be an endovascular procedure, with the exception of most of type I fistulous AV malformations. Direct microsurgery should be conducted in type I fistulous malformations because the small caliber of the

feeding artery prohibits placement of a catheter close to the fistula. Type II and III fistulous malformations have dilated feeding vessels enabling superselective catheterization close to the fistula and subsequent obliteration. In the glomus type, glue or particles can be employed to obliterate the nidus. In the juvenile type, only those parts of the malformation should be treated that are most likely to account for the symptoms [22-24].

4. Diagnostic imaging

When spinal lesions are suspected, MRI should constitute the first diagnostic modality.

In spinal dural AV fistulas, MRI demonstrates the combination of intramedullary signal alterations and perimedullary dilated vessels [25]. On T2-weighted images, the cord shows centromedullary hyperintensity over multiple segments. On T1-weighted images, the swollen cord is slightly hypointense and enlarged. Following contrast administration, diffuse intramedullary enhancement may be seen as a sign of chronic venous congestion with a breakdown of the blood–spinal cord barrier. In the further course of the disease, the spinal cord will be atrophic. The dilated and tortuous perimedullary vessels are mainly on the dorsal part of the cord and can be seen on the T2-weighted images as flow void signals. If they are small, however, they might be seen only after contrast enhancement. The coiled or serpentine vascular structures may be well delineated on heavily T2-weighted sequences. Shunts may occur anywhere from the level of the foramen magnum to the sacrum, and localization of these lesions may be difficult and challenging, especially in cases in which the edematous change of the cord occurs at a considerable distant from the shunt.

The typical appearance of spinal cord pial AV malformations is a conglomerate of dilated vessels with peri- and intramedullary locations that appear on T2-weighted images as flow void signals [32, 33]. Depending on their flow velocity and direction, these abnormal vessels are delineated as mixed hyper/hypointense tubular structures on T1-weighted images. Contrast enhancement may vary. Venous congestive edema may be present as an intramedullary hyperintensity on T2-weighted images with concomitant swelling of the cord. The image might become even more complicated if hematomyelia and subarachnoid hemorrhage are present; that might demonstrate varying signal intensities depending on the time course. MRI can identify the location of the nidus in relation to the spinal cord and dura. Especially in the small perimedullary fistulous type (type I), contrast media must be given to detect subtle venous dilatations.

Spinal contrast-enhanced dynamic MRA has contributed to shunt localization in some cases [34-37]. The technique of first-pass gadolinium-enhanced MRA can demonstrate early venous filling, which indicates the level of the shunt. Multidetector-row helical CT angiography with intravenous contrast injection (IV-CTA) can provide spinal vessel images showing the surrounding bony structure [38, 39]. These images allow a wide survey of possible shunts, but the spatial and temporal resolution is inadequate for planning the treatment strategy. Furthermore, cine review of the entire spine is not possible because of the limitations of acquisition time and the limited field of view (FOV).



Figure 2. Sagittal sections of T2-weight MRI. Left: spinal dural AV fistula. Right: spinal cord AV malformation.

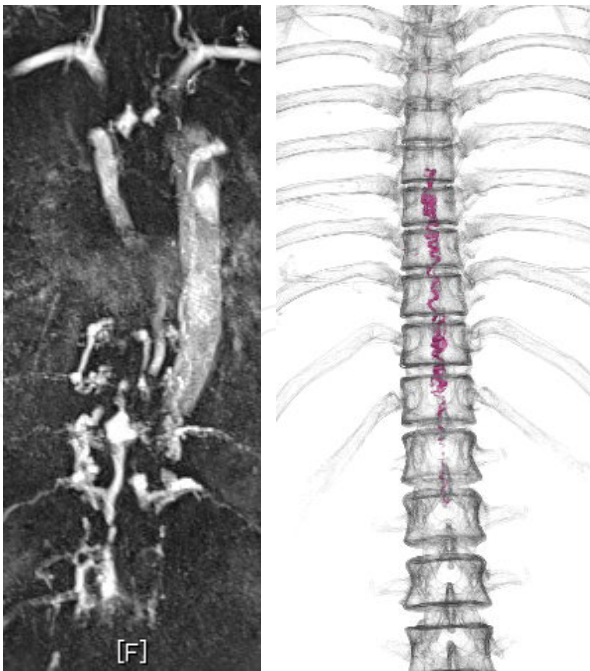


Figure 3. Less-invasive spinal vascular imaging. Left: Spinal contrast-enhanced dynamic MRA of fistulous-type spinal cord AV malformation (type III). Right: Multidetector-row helical CT angiography with intravenous contrast injection (IV-CTA) of fistulous-type spinal cord AV malformation (type II).

Although MRI, MRA and IV-CTA have the distinct advantage of less-invasive examination of the spinal cord and surrounding structures, their findings do not provide any hemodynamic information. Until date, selective spinal digital subtraction angiography (DSA) is the standard (and indispensable) for diagnosis, treatment, and follow-up examination of spinal vascular lesions [24, 37]. However, spinal DSA is an invasive procedure. Complete spinal DSA includes all vessels that may feed the spinal cord, i.e., all intercostal and lumbar arteries, the cervical feeders including both vertebral arteries, the thyrocervical trunk, the deep cervical artery, and the internal iliac arteries. Selective angiography of all segmental arteries can often be time-consuming, require multiple catheterizations, involve long radiation exposure times, and use large volumes of contrast agents. Furthermore, multiple catheterizations of small segmental arteries can lead to vessel injury and thromboembolism [40, 41].

5. Intra-arterial (intra-aortic) contrast injection CT angiography

Anatomical localization of the shunts is not only imperative for definitive therapy but can also complement spinal DSA, possibly leading to reduction in the number of selective catheterizations and fewer procedural complications. The radicular and spinal arteries are small vessels with a diameter of 0.5–1.5 mm and are surrounded by osseous structures. This anatomic feature decrease the contrast-to-noise ratio (CNR) in IV-CTA [42]. Although robust contrast enhancement is necessary for detection of small vessels, IV-CTA has limitations with respect to elevating vessel enhancement, because the contrast material is diluted in pulmonary circulation [43]. To delineate such minute vessels, a method that increases the bolus characteristic of contrast medium is preferable. To enhance the spatial resolution of spinal CTA, we tried multiphase intra-arterial (intra-aortic) contrast injection CT angiography (IA-CTA) in advance of spinal DSA. We found that IA-CTA could track the normal/abnormal spinal arteries to the aorta in detail. Multiphase dynamic imaging can discriminate the arterial component from the draining veins, which improves the precision of diagnosis.

A 4 (or 5) Fr Pigtail Catheter was advanced to the proximal portion of the descending aorta for thoracolumbar lesions or the proximal portion of the ascending aorta for cervicothoracic lesions. Patients were then transferred to the CT room, and 80 mL of iodinated contrast material (Iopamiron 300 mg I/mL; Nihon Schering K.K., Osaka, Japan) was injected via the catheter at a rate of 4 mL/s. The CT scan started 5 seconds after starting the injection and was consecutively repeated to obtain early- and late-phase images. Two datasets were reconstructed from the two consecutive scans. Image datasets were transferred to a workstation. An oblique coronal multiplanar reconstruction fitting the curvature of the spine was obtained. Exactly the same MPR sections were obtained from the second phase to distinguish the feeding arteries from the draining veins, which were in close proximity. Curved planar reformation (CPR) and three-dimensional volume rendering were applied to display an overview of the lesions. Detection of the arterial feeders confirmed the presence of a connecting vessel ascending from the intervertebral foramen to the lesions, and the absence of further enhancement in the second phase compared with the first phase. Continuity was confirmed by paging oblique coronal MPR or axial images. Spinal DSA with selective catheterization was subsequently performed with reference to the findings of IA-CTA.

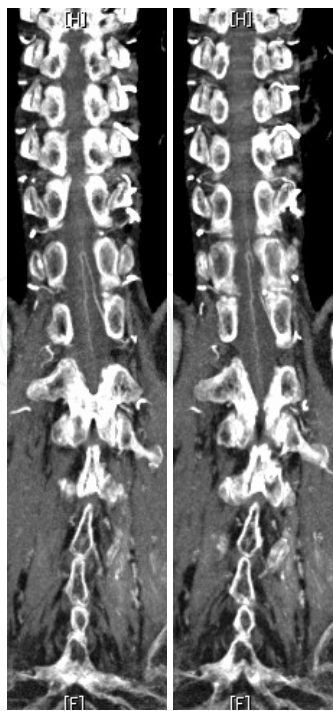


Figure 4. IA-CTA (arterial phase) demonstrating the radicular arteries and the artery of Adamkiewicz clearly.

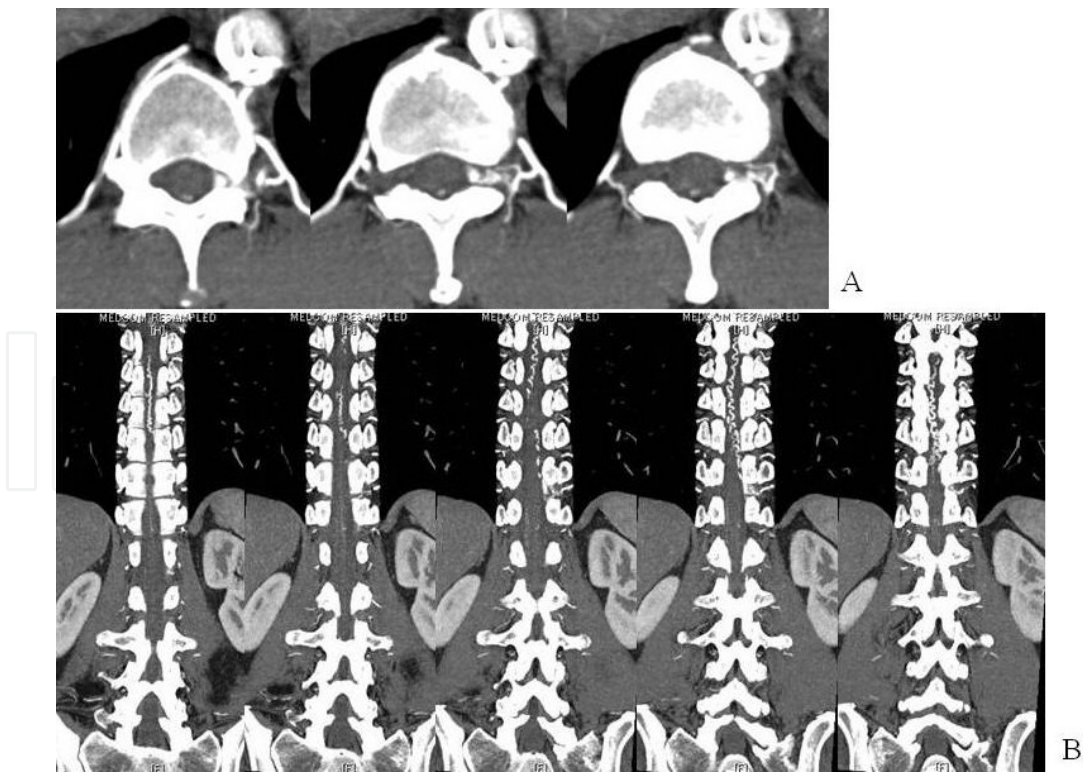


Figure 5. Spinal dural AV fistula with a feeder originated from the left T11 intercostal artery. A: serial axial sections of IA-CTA. B: serial coronal sections of IA-CTA.

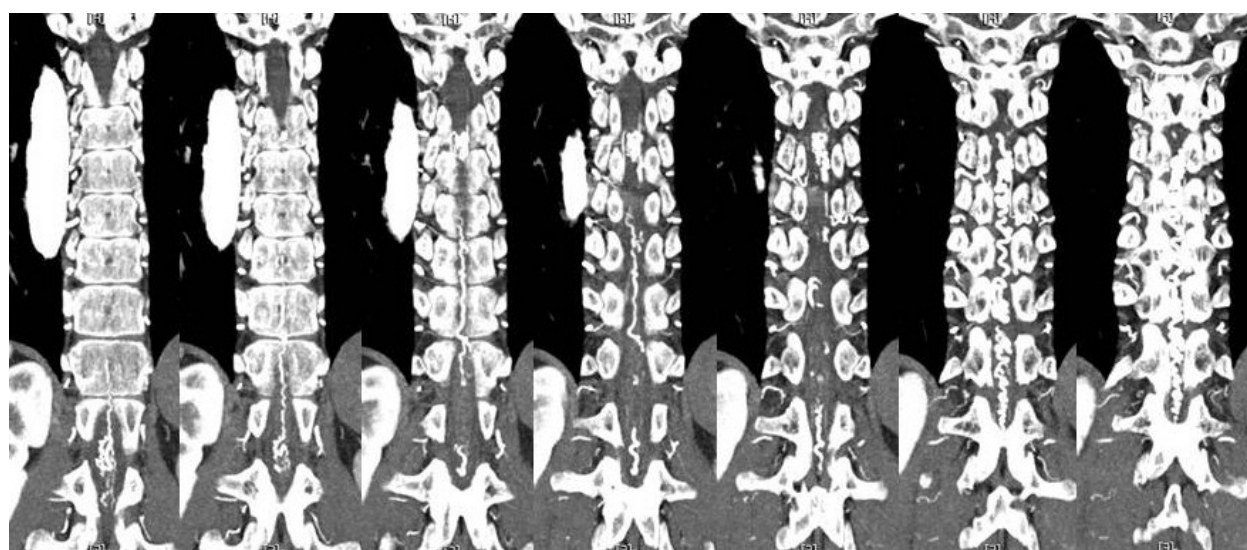


Figure 6. Serial coronal sections of IA-CTA demonstrating fistulous-type spinal cord AV malformation (type II), fed by rt. T9 and lt. T8 intercostal arteries.

6. Direct surgery under the guidance of intra-arterial injection indocyanine green (ICG) videoangiography

The goal of treatment for spinal AV shunts is the extirpation of shunt vessels without compromising the spinal cord circulation. In spinal cord pial AV malformations in particular, however, this is not feasible because of the complexity of the vasculature. For direct surgery of spinal cord AV shunts, various intraoperative diagnostic modalities have been used to assess the hemodynamics and to identify the shunting vessels, such as intraoperative spinal DSA, Doppler ultrasonography and videoangiography using indigo carmine and indocyanine green [44-50]. Intraoperative spinal DSA is a standard tool for demonstrating shunting flow and confirming extirpation [49]. However, its resolution is not adequate to assess the precise anatomy of the vasculature. Intra-arterial injection of indigo carmine via a catheter introduced for the spinal DSA has the advantage of visibility with conventional light optics; however, when compared with ICG fluorescence, the development and washout of the blue dye is less clear, and the time resolution and utility in discerning arterial or venous components is significantly limited [45]. Recent advances in microscope-integrated videoangiography using ICG have made it possible to visualize blood flow and hemodynamic changes in real time. A technique using intravenous injection of ICG has been used for cerebrovascular surgery, however, it is limited to detect the flow direction and velocity and to repeat the examination frequently [46-48]. To enhance the temporal and spatial resolution of this technique in spinal vascular surgery, we introduced intraarterial injection ICG videoangiography with selective catheterization [50].

After induction of general anesthesia, a 5-Fr long metallic introducer vessel sheath was inserted into the femoral artery in the operating room. After surgical exposure of the lesions, an

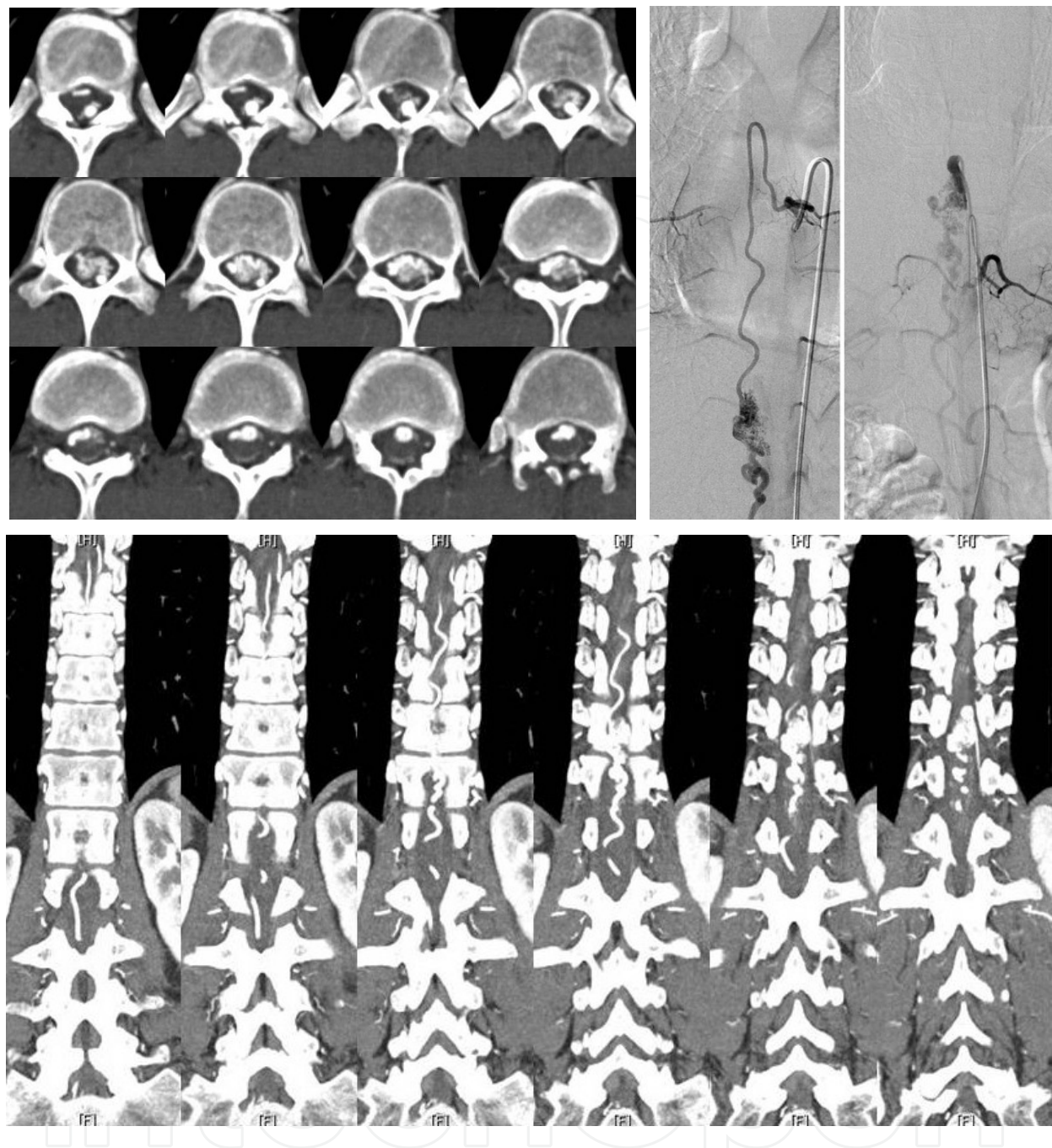


Figure 7. Glomus-type spinal cord AV pial malformation. Serial axial sections of IA-CTA showing the location of the intramedullary nidus and perimedullary varix. Coronal sections indicating 2 arterial feeders originated from the left T8 and T12 intercostal arteries, which was confirmed by spinal DSA.

angiographic catheter was placed into the proximal portion of the feeding artery for intraoperative DSA and ICG injection. When ICG solution (0.06 mg in 5 ml saline) was injected through the catheter, the feeders and then the drainers were illuminated with fluorescence and became clearly distinguishable using a fluorescence surgical microscope. Temporary occlusion applied close to the shunts led to immediate disappearance of the shunt flow. In addition to

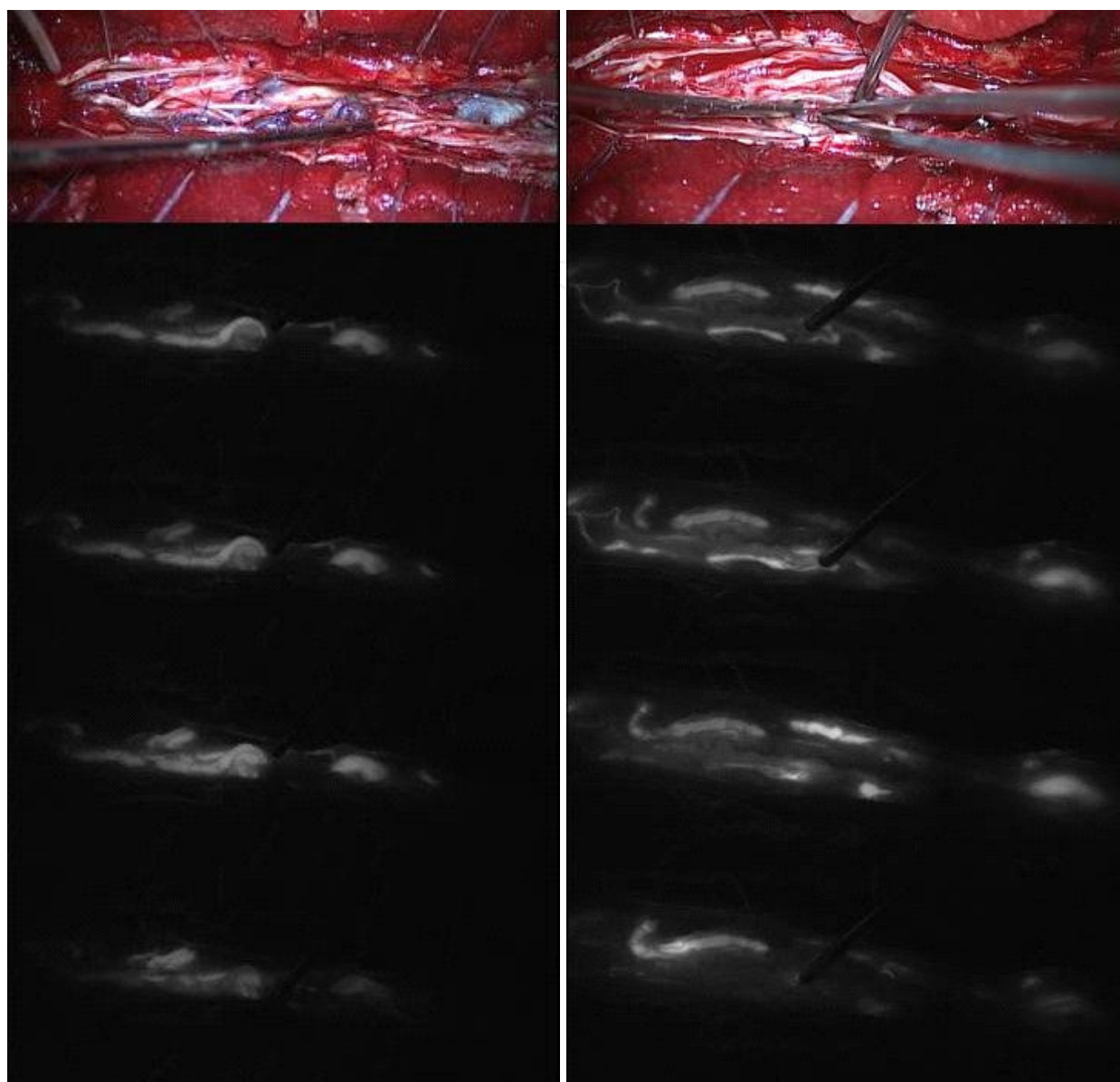


Figure 8. Intraoperative views of spinal cord AV malformation at the conus medullaris. Left: Indigo carmine videoangiography and serial photographs of IA-ICG videoangiography illuminating the feeders, shunts, and drainers in order. Right: IA-ICG videoangiography demonstrating the intraoperative flow alteration clearly.

fluorescence angiography, Doppler ultrasonography and intraoperative DSA were used to confirm extirpation of the AV shunts. Selective IA-ICG videoangiography also showed the patency of the normal anterior/posterior spinal artery, which could not be visualized by intraoperative DSA.

ICG videoangiography by intravenous bolus injection is performed with 10–25 mg ICG dissolved in saline. Once ICG is injected intravenously, the dye is diluted in the systemic circulation and carried to the tributary of observation via arterial flow. The background fluorescence remains for several minutes until the systemic concentration of ICG was de-

creased through hepatic excretion, which disturbed the detection of alterations in vessel flow. By using intra-arterial selective catheterization to the vicinity of the pathology, a much smaller dose of ICG can visualize the vessels without any background. Flow dynamics such as direction, velocity and alteration after temporary occlusion were well visualized. The resulting quick washout allows differentiation of the phase of the filling, thus identifying the feeder, the drainer, and the shunts in between. The small dose allows repetition of the examinations. The temporal resolution is far superior to that obtained by the intravenous injection technique. With test occlusion applied to one surface vessel to the other, and by observing alterations in the filling pattern in a real time, it becomes possible to identify the feeders and the drainers and thereby localize the embedded shunts. These are the great advantages of the intra-arterial injection over intravenous administration. The feasibility of multiple repeated injections helps in obtaining precise flow-dynamic information. When faced with complex spinal cord AV malformations, such repeated real-time information is indispensable for the localization and obliteration of the hidden shunts.

7. Conclusion

Despite the considerable advances that have been made in imaging and endovascular technology, spinal AV shunts continue to pose significant therapeutic challenges. Endovascular therapy and direct surgery with effective intraoperative diagnostic aids have a complementary role in the management, and understanding of the pathophysiology of these shunts and information about the detailed vasculature of the lesions is indispensable for achieving optimal results.

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