Spinal Vascular Malformations: A Review

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Abstract

This article summarizes the senior author's experience (RFS), which has been condensed to a schematic system based on broad categories reflecting the anatomy and angioarchitecture of spinal vascular formations. Anatomically, spinal vascular malformations can occur within or outside the dura (i.e., intra- or extradural) and are located anteriorly or posteriorly with respect to the spinal cord. Based on their angiographic appearance, the presence of a nidus defines an arteriovenous malformation (AVM). A lesion is considered an arteriovenous fistula (AVF) if no nidus is present.

Vascular malformations involving the spinal cord represent a heterogeneous and challenging group of entities with a broad clinical spectrum that ranges from neurological deficits that develop progressively over years to an insidious presentation caused by an acute hemorrhage. Multiple classification systems are frequently used to categorize these lesions based on multiple clinical and angiographic criteria. This confusing welter of nomenclature only adds to the complexity of treating these challenging lesions.

This article summarizes the senior author's experience (RFS) [36] (Table 1), which has been condensed to a schematic system based on broad categories reflecting the anatomy and angioarchitecture of spinal vascular formations. Anatomically, spinal vascular malformations can occur within or outside the dura (i.e., intra- or extradural) and are located anteriorly or posteriorly with respect to the spinal cord. Based on their angiographic appearance, the presence of a nidus defines an arteriovenous malformation (AVM). A lesion is considered an arteriovenous fistula (AVF) if no nidus is present.

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Cavernous Malformations

Cavernous malformations can occur anywhere within the spinal canal and are characteristically "occult" during catheter-based angiography. Although it is controversial to consider cavernous malformations as neoplastic lesions, they share some features common to neoplasms. They have the potential to grow and are associated with a chromosomal abnormality. They also can be familial or acquired.[10] The familial form is associated with a dominant trait [34] located on chromosome 7q.[30] They also can be induced by radiation.[24] Histologically, they are identical to the cavernous malformations that occur in the brain [4] and the same as spinal hemangiomas that occur within the vertebral bodies.

Cavernous malformations can become symptomatic abruptly from a new hemorrhagic event, or they can manifest with progressive neurological decline related to compression of the spinal cord. Symptoms are based on the location of the lesion within the spinal cord. T2-weighted magnetic resonance (MR) images show blood in different stages within a well-demarcated lesion with mixed signal intensity at the core and low signal intensity on the periphery (hemosiderin). These characteristic features are commonly known as the "popcorn" appearance.

The clinical history of patients with spinal cord cavernous malformations is one of acute neurological decline that improves to a certain degree but without regaining the patient's baseline functional status. After a couple of hemorrhages, the patient typically has sustained significant impairment.[32]

Symptomatic patients usually require surgical resection to stop the progression. It is important to treat patients soon after their symptoms

develop because early treatment is associated with a better chance of improvement than delayed treatment. In one study, 76% of patients undergoing early treatment improved compared to only 52% ($p<0.02$) treated late (a mean of 3 years after they first became symptomatic).[40] The main prognostic factor is a patient's preoperative status.[10]

Patients with acute hemorrhage or progressive neurological deficits should be considered candidates for surgical resection. In patients who are clinically improving or in patients with minor symptoms, the risk of surgery must be weighed against the possibility of irreversible damage from an acute hemorrhage. Typically, lesions that are small, exophytic, or located dorsally are the most accessible.

During surgery the cavernous malformation is identified as an area of blue discoloration within the spinal cord (Fig. 1). Surgical resection can be accomplished by preserving the glial rim as a dissection plane around the lesion. During surgery retraction should be minimized. Bipolar electrocoagulation should be set on low power or reduced to a minimum to avoid injury to the spinal cord. The associated venous anomaly should always be preserved to avoid catastrophic consequences.

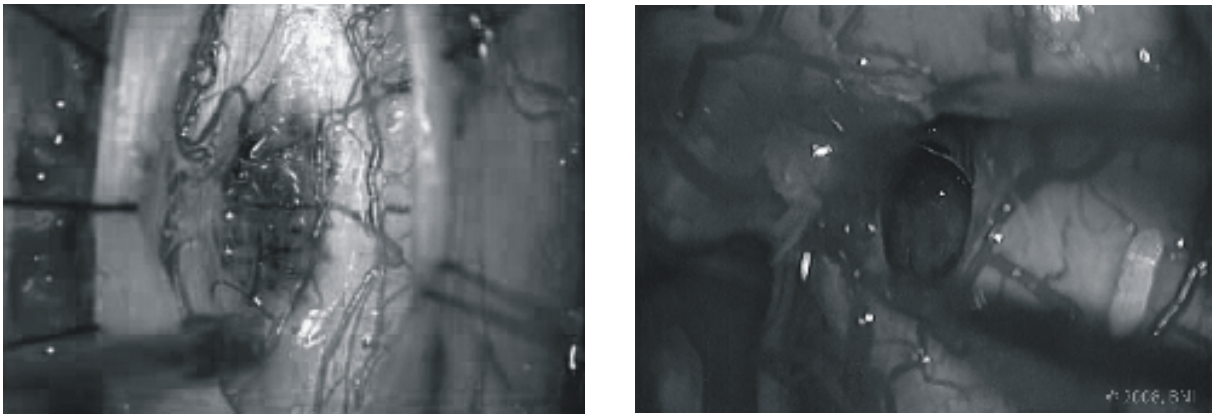


Figure 1. Intraoperative photographs showing a (A) cavernous malformation and (B) the resection cavity. Used with permission from Barrow Neurological Institute.

Complete surgical resection is necessary because residual lesions can regrow and are also associated with the possibility of catastrophic consequences.[39] In the senior author's experience with more than 45 cavernous malformations of the spinal cord, the Frankel functional outcome score of 91% of the patients remained the same or improved. Five (11%) patients presented with new hemorrhagic episodes related to incomplete resection and improved after a second operation (Kim L and Spetzler RF, unpublished data). If symptoms recur after surgery in the absence of residual cavernous malformation, the possibility of a tethered cord should be considered.

Because cavernous malformations are angiographically occult, endovascular therapy has no role in their treatment. Radiation therapy also has no role in their treatment.

AVMs and AVFs

AVMs are characterized by the presence of a nidus within the spinal cord while AVFs have a direct connection between an artery and a vein with no intervening capillary network.

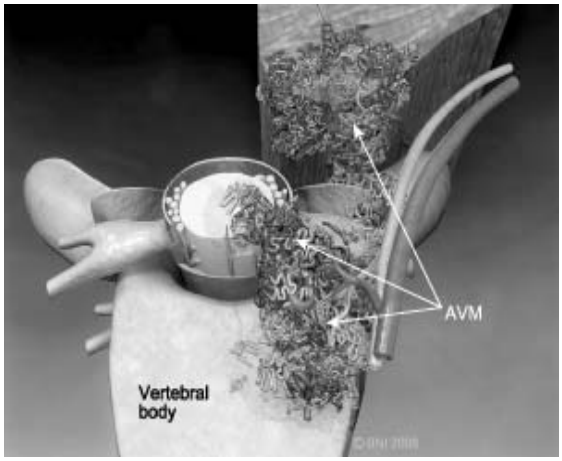
AVFs can be either intra- or extradural. Intradural lesions are located either ventrally or dorsally with respect to the spinal cord. AVMs can be extradural and intradural simultaneously, or they can be purely intradural and then subdivided into intramedullary and conus medullaris AVMS.

AVMs

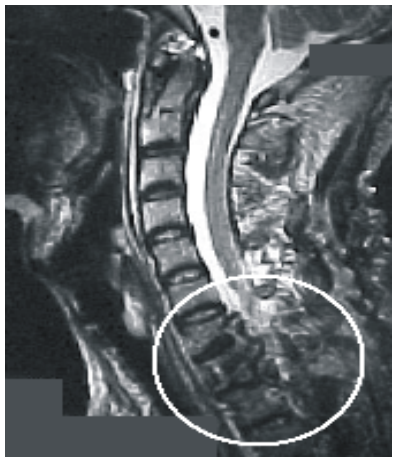
Intradural-Extradural AVMs

Intradural-extradural AVMs are also known as type III, metameric, or juvenile AVMS or as Cobb's syndrome. These complex lesions usually involve the entire spinal segment including the skin, muscle, bone and spinal cord, with no respect for anatomical boundaries. They most often involve the thoracic spine but can extend a few levels causing diffuse and broad

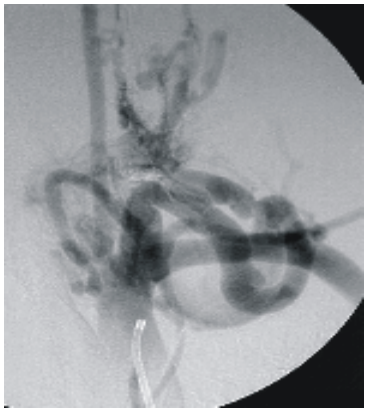
compromise of function. These lesions are highly vascular; therefore, a multidisciplinary multistaged treatment is recommended. Preoperative embolization is key to successful treatment. Nonetheless, treatment is usually only palliative because complete obliteration can rarely be obtained due to the extensive nature of these lesions (Fig. 2).



Gonzalez 2A BnW



Gonzalez 2B



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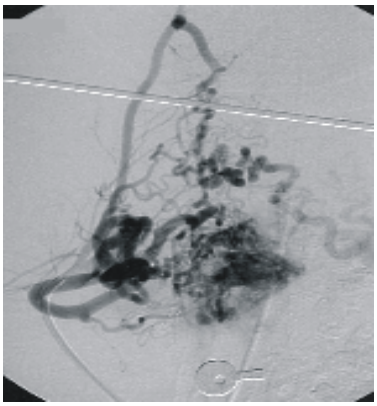
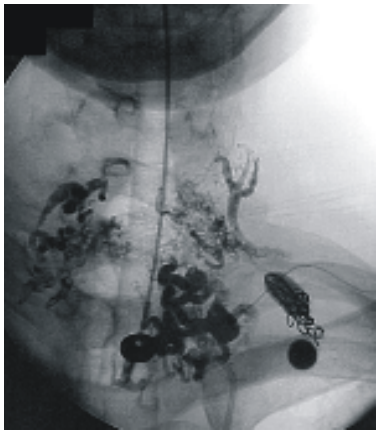


Gonzalez 2D



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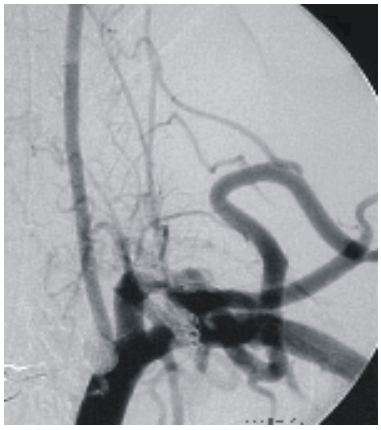


Gonzalez 2F

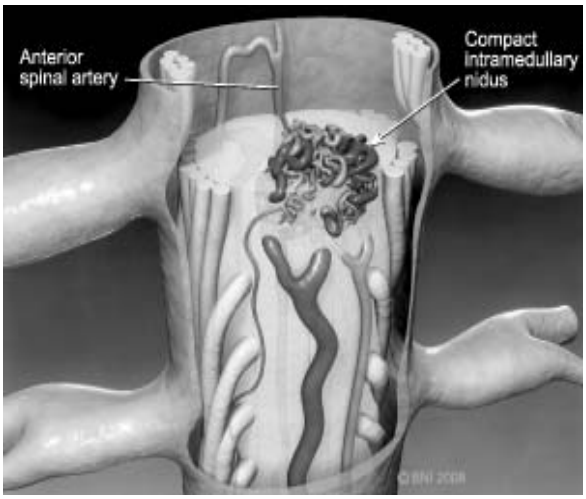
Figure 2. (A) Illustration of a complex metameric AVM. (B) Sagittal T2-weighted MR image showing a cervicothoracic metameric AVM. Note the lack of an anatomic boundary (circle). (C) Left subclavian angiographic injection showing the complexity of this AVM with large venous varices. (D) Right subclavian angiographic injection showing the AVM. (E) Fluoroscopic image showing the glue cast after embolization. (F) Left subclavian angiographic injection showing no evidence of residual AVM. Used with permission from Barrow Neurological Institute.

Intradural AVMs

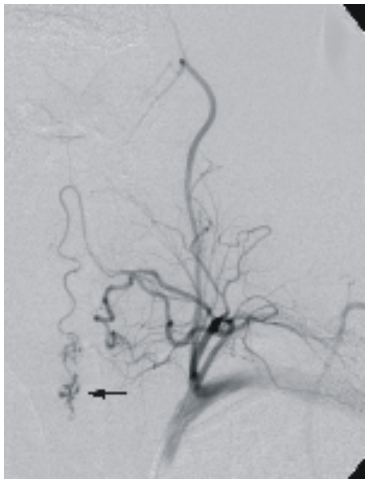
Intradural AVMs are also known as type I, glomus, or classic AVMs. They resemble AVMs found in the brain. These lesions usually manifest with hemorrhage within the spinal cord or with acute, nonhemorrhagic deterioration possibly related to spontaneous venous thrombosis.[11] They can also become symptomatic with progressive neurologic deterioration related to vascular steal or with spinal cord compression caused by engorged veins.

These lesions often compromise the anterior spinal artery. Therefore, surgery is usually required to obtain a definitive cure. Frequently, intradural AVMs can be resected from a posterior approach.[12]

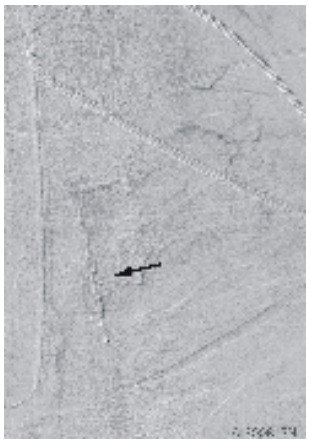
Based on the angioarchitecture of the nidus, these lesions can be subdivided into compact and diffuse types. Those with compact niduses (Fig. 3A) are most amenable to curative embolization (Fig. 3B and C) or surgical resection. They can be approached through a standard laminotomy. A small myelotomy is performed by following the posterior median sulcus, and the spinal cord is split between the two posterior columns. If instead the lesion is close to the lateral surface of the spinal cord, a posterolateral myelotomy is performed through the dorsal root entry zone (DREZ) between two or more nerve roots. Superselective catheterization is very helpful to identify any associated aneurysms.[8] Preoperative embolization of major feeders is advantageous during surgery. [5,26]



Gonzalez 3A



Gonzalez 3B



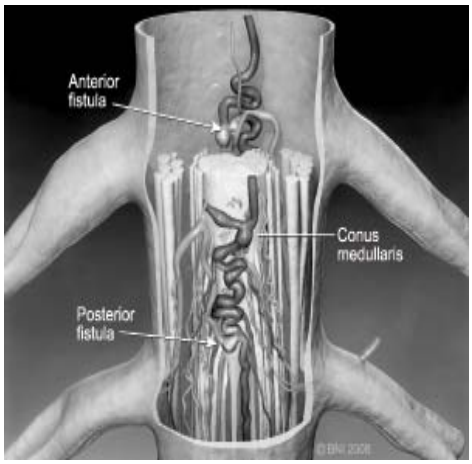
Gonzalez 3C

Figure 3. (A) Illustration showing an intradural AVM with its compact nidus within the spinal cord. (B) Left subclavian angiographic injection showing a compact AVM fed by the anterior spinal artery in a patient who became symptomatic with progressive quadriparesis. Arrow points at nidus. (C) The Onyx cast (arrow) is visible after embolization. Used with permission from Barrow Neurological Institute.

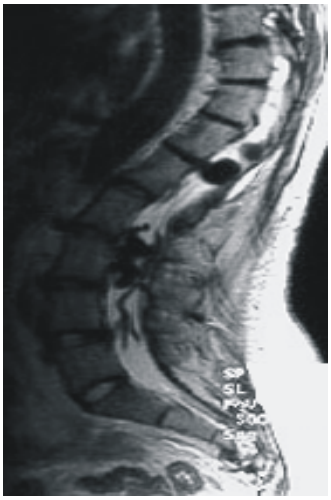
Complete obliteration of the AVM is possible with standard endovascular techniques. During spinal embolization in patients under general anesthesia, we routinely monitor motor evoked potentials [35] and somatosensory evoked potentials. A selective injection of amobarbital (spinal Wada test) [14] and cardiac lidocaine, as close to the AVM as possible, helps predict the effect of occluding a particular feeding artery on the normal blood supply of the spinal cord. In some cases, we have observed lidocaine and amytal to cause a significant decrease in the motor evoked potentials whereas no change was seen on somatosensory evoked potentials.

Conus Medullaris AVMs

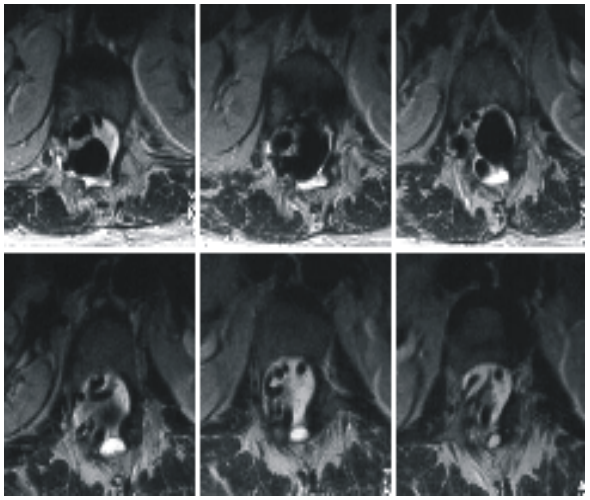
This special category of lesion is characterized by the presence of multiple simultaneous fistulas, including anterior and dorsal components with multiple feeders, and by the presence of an intramedullary AVM (Fig. 4). An abnormality during neurulation has been proposed as responsible for the predilection of these lesions to develop at the conus medullaris [22] and might be associated with tethered cord. The extensive nature of these lesions, with multiple fistulas, makes them challenging to treat by endovascular means, but endovascular treatment can be successful in some cases (Fig. 4B-E). An initial embolization followed by surgical resection is ideal.



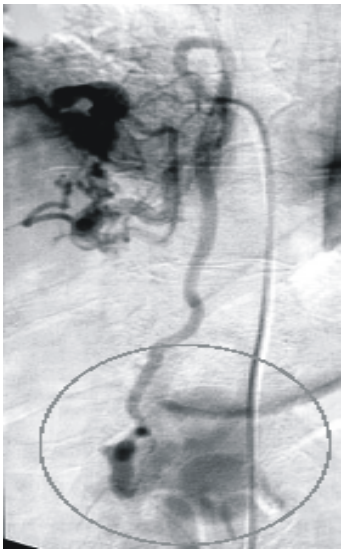
Gonzalez 4A



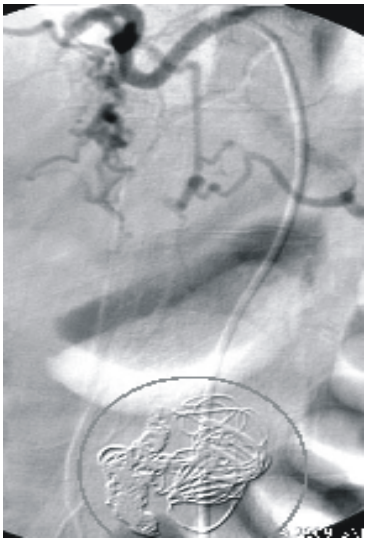
Gonzalez 4b



Gonzalez 4C



Gonzalez 4D



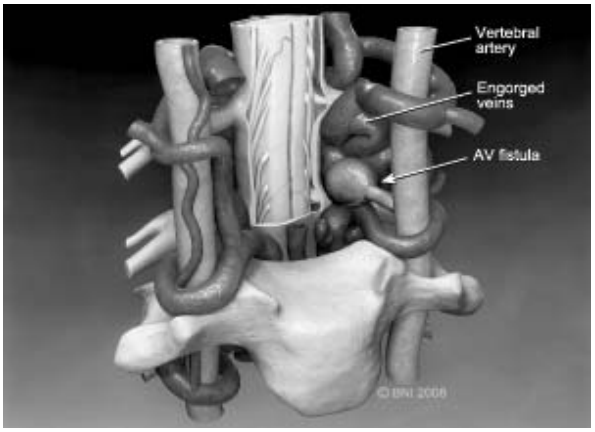
Gonzalez 4E

Figure 4. (A) Illustration showing a complex vascular malformation, including two AVFs, at the level of the conus medullaris. Note the two arterial feeders. (B) Sagittal MR image showing dilated varices. (C) Sequence of axial MR images showing the complex varices. (D) Angiogram showing the arterial feeder draining into the large venous pouch (circle). (E) Angiogram showing coil embolization of the pouch (circle). Used with permission from Barrow Neurological Institute.

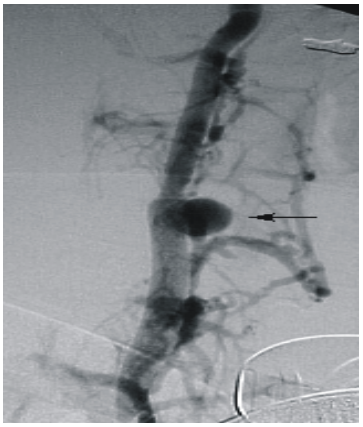
AVFs

Extradural AVFs

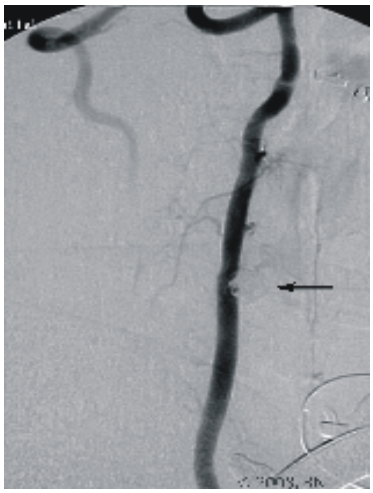
A direct fistula between an extradural artery and the epidural venous system causes a high-flow lesion that produces engorgement of the epidural veins and causes compression of the spinal cord. Shunting arterial blood into the venous system can result in vascular steal from the spinal cord and cause symptoms related to ischemia. This type of AVF rarely requires surgery because it can be treated effectively with endovascular techniques (Fig. 5).[9] In rare cases extradural fistulas may be the cause of a spinal epidural hematoma, and spinal angiography should be considered during the patient's evaluation. [25,28]



Gonzalez 5A



Gonzalez 5B



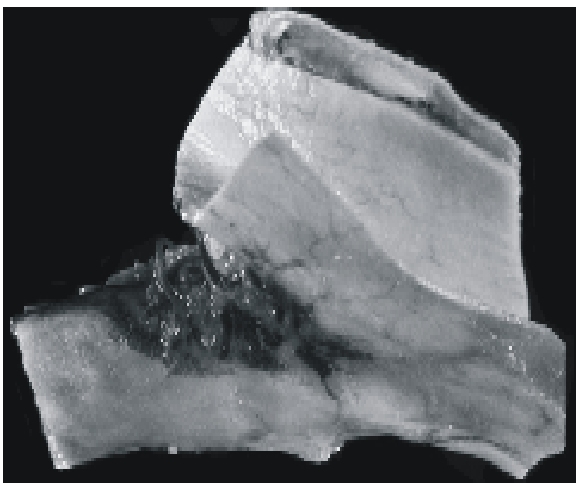
Gonzalez 5C

Figure 5. (A) Illustration showing the connection between the vertebral artery and epidural venous plexus. Note the engorgement of the epidural veins. (B) Vertebral artery angiographic injection showing an intradural fistula caused by a gun shot wound that left the patient quadriplegic. Note the presence of an aneurysm (arrow). (C) The vertebral artery was treated with a stent, and the aneurysm was coiled (arrow). Used with permission from Barrow Neurological Institute.

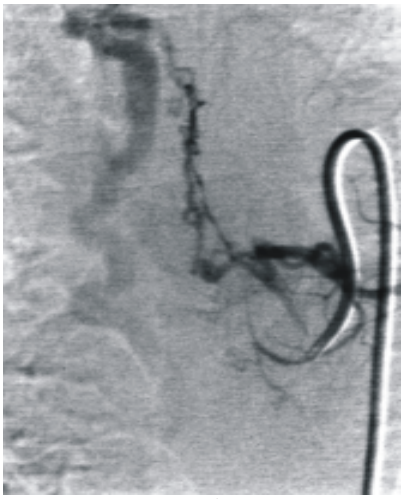
Intradural Dorsal AVFs

Many names, such as Type I, angioma racemosum, angioma racemosum venosum, long dorsal AVFs, and dorsal extramedullary AVFs, have been used to describe intradural dorsal AVFs. These AVFs are the most common type of vascular lesion to affect the spinal cord.[21,37] For unknown reasons, intradural dorsal AVFs typically occur in the thoracic and lumbar regions.

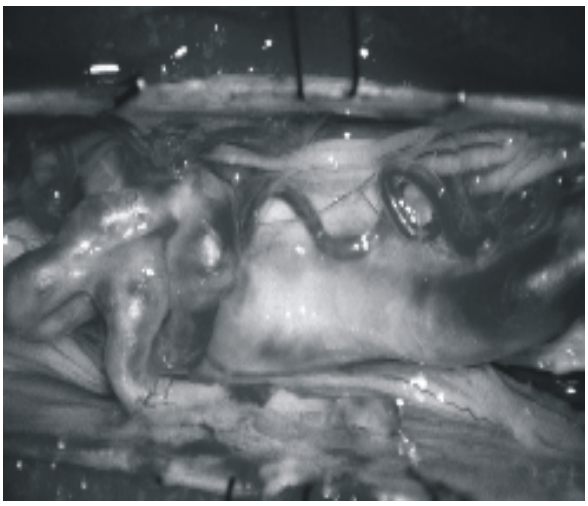
The connection between a radiculomedullary artery and a radicular vein occurs intradurally at the dural root sleeve causing venous hypertension at the coronal plexus (Fig. 6).[15] The absence of valves between the coronal and radicular veins facilitates the development of venous congestion.[33] Venous hypertension decreases spinal cord perfusion pressure, thereby leading to ischemia as the primary cause of symptoms. Symptoms are usually exacerbated by a Valsalva maneuver, which further increases venous pressure and decreases blood flow in the spinal cord.



Gonzalez 6A



Gonzalez 6B



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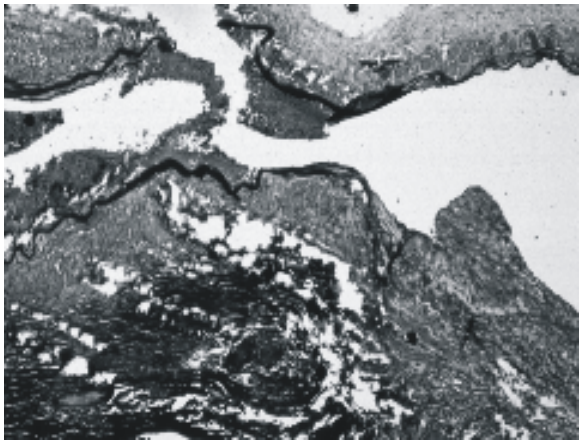
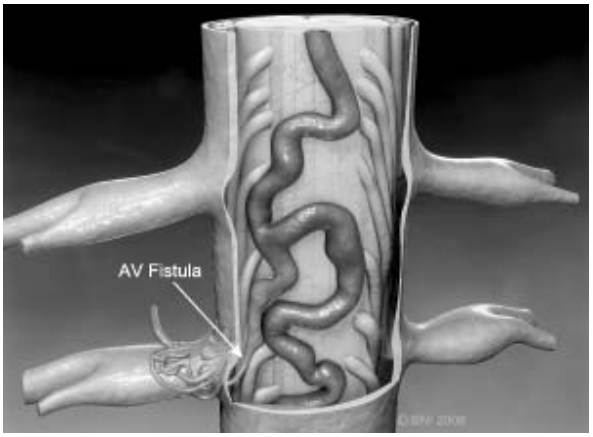


Gonzalez 6D



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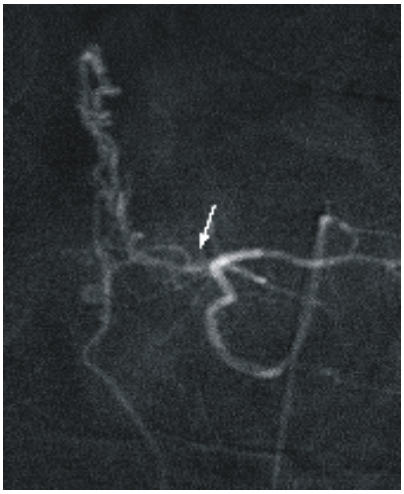

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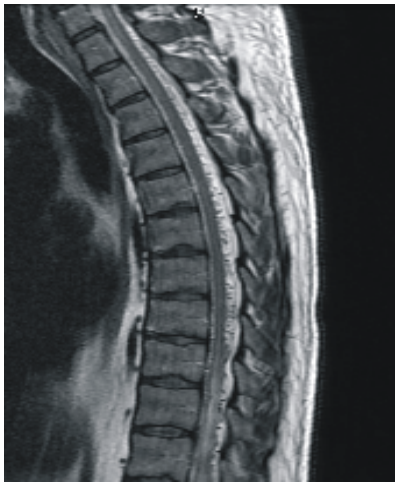
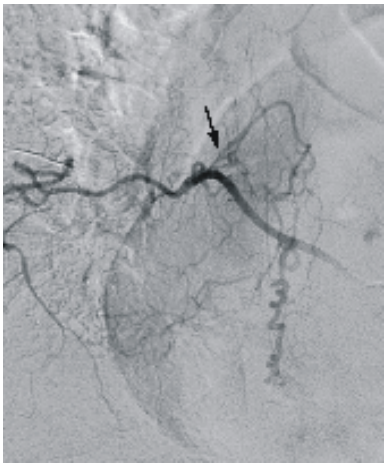
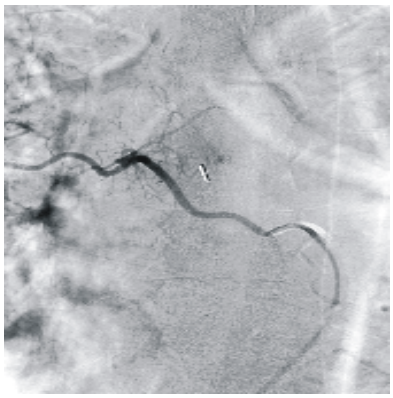


Gonzalez 6 H



Gonzalez 6I



Gonzalez 6J

Figure 6. (A) Anatomical specimen showing the normal vascular plexus around the dural sleeve. (B) Angiogram showing the AVF with (C) an intraoperative photograph from a surgical case showing the same findings. (D) Photomicrograph showing the transition from artery to vein after resection of the fistula. (E) Illustration showing an intradural dorsal AVF. The fistulous site occurs in the intradural compartment just after the vessels enter the dura. (F) Spinal angiogram at left T6 from a patient with a dorsal AVF. Arrow shows the fistulous site. (G) The nBCA cast and the coil occluding the intercostal artery to facilitate occlusion of the fistula. (H) Sagittal T2-weighted MR image from a 55-year-old patient who presented with paraparesis and urinary incontinence showing abnormal flow voids within the spinal canal. (I) Spinal angiogram from the same patient showing a dorsal fistula (arrow). (J) Postoperative spinal angiogram demonstrates no early filling. Figure 6A courtesy of Professor Rob Groen Department of Neurosurgery, University Hospital Groningen, Groningen, The Netherlands. Figure 6B and D from Spetzler RF, Koos WT: Color Atlas of Microneurosurgery, Vol 3. New York: Thieme, 2000, p 407. Used with permission from Thieme. Figure 6C from Anson JA, Spetzler RF: Spinal arteriovenous malformations: Surgical treatment. In Carter LP, Spetzler RF, Hamilton MG (eds.) Neurovascular Surgery. New York, McGraw-Hill, 1995, p. 1203. Used with permission from McGraw-Hill. Figures 6E-J Used with permission from Barrow Neurological Institute.

Clinically, patients present with early fecal and urinary incontinence and progressive and incapacitating myelopathy.[37] Aminoff and Logue reported that 19% of their patients were disabled within 6 months and 50% were disabled within 3 years. [1]

When the fistula is occluded during surgery, venous pressure decreases significantly in the intradural space, demonstrating the presence of the fistula in the intradural compartment. [19]

That the pressure does not decrease to the level of normal central venous pressure

indicates some degree of obstruction in venous drainage. [7]

These lesions have been subclassified in two groups according to their number of feeders. Type A lesions have a single feeder, and type B lesions have multiple feeders. [2,3]

Surgery provides the most definitive treatment, although excellent outcomes have been associated with endovascular techniques using n-butyl cyanoacrylate (nBCA) rather than particles, which are associated with a higher incidence of recurrences. [9,15]

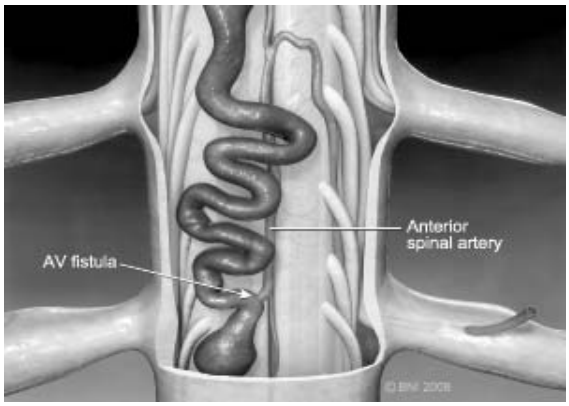
In the presence of amenable feeding vessels, diagnosis and treatment can be achieved during the same endovascular procedure (Fig. 6B, C). During surgery a small laminotomy and a midline dural incision offer access to the nerve root where the fistula is located. Intraoperative indocyanine green angiography (ICG) shows the early and rapid filling of compromised veins. It also shows the absence of rapid filling once the fistula is temporarily occluded, thereby confirming the fistulous site. Once the site is confirmed, definitive coagulation and section can be performed (Fig. 6D-F).

We recommend limiting the surgical procedure to ligation of the feeding vessel at the fistulous site, without stripping the dilated veins [29] that drain normal spinal cord. Doing so may cause clinical deterioration.

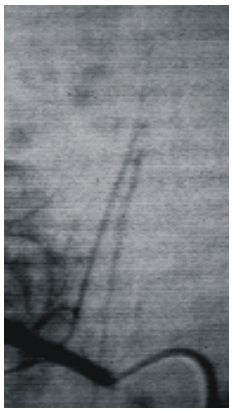
Intradural Ventral AVFs

These AVFs, initially described by Djindjian, [13] are also known as Type IV [20] or perimedullary fistulas. As their name implies, these ventral

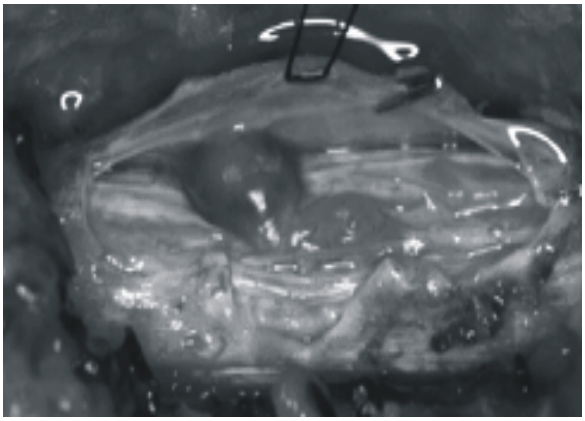
AVFs usually occur anterior to the spinal cord and cause symptoms related to compression or hemorrhage (spinal subarachnoid hemorrhage) (Fig. 7). They can manifest as part of the Rendu-Osler-Weber or Cobb's syndrome. [18]



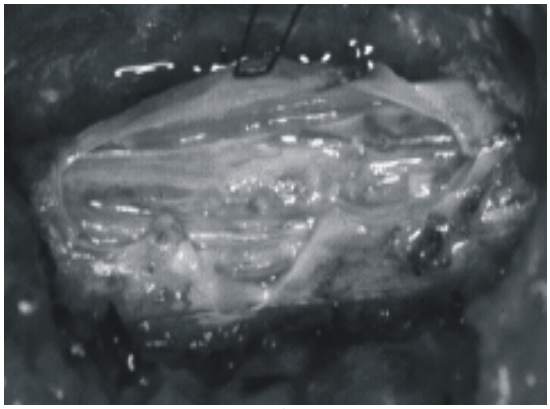
Gonzalez 7A



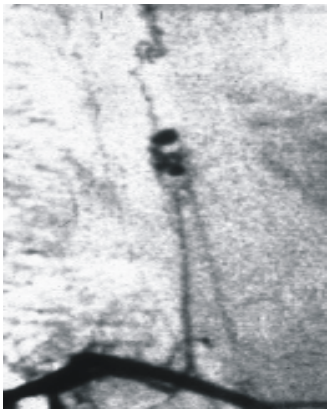
Gonzalez 7B



Gonzalez 7C



Gonzalez 7D



Gonzalez 7E

Figure 7. (A) Illustration of an intradural ventral AVF. The anterior spinal artery is involved in the fistula. (B) Spinal angiogram from a 15-year-old patient who presented with back pain and paraplegia showing the anterior fistula involved with the anterior spinal artery. (C) Intraoperative photograph showing an anterolateral AVF associated with an aneurysm. (D) Intraoperative photograph after resection of the aneurysm. (E) Postoperative spinal angiogram showing no evidence of the AVM. Figure 7A used with permission from Barrow Neurological Institute. Figures 7B-E from Anson JA, Spetzler RF: Spinal arteriovenous malformations: Surgical treatment. In Carter LP, Spetzler RF, Hamilton MG (eds.) Neurovascular Surgery. New York, McGraw-Hill, 1995, p. 1209. Used with permission from McGraw-Hill.

Angiographically, these AVFs are high-flow lesions. They frequently involve the anterior spinal artery. The fistula occurs within the spinal canal and outside the spinal cord.[17] The communication occurs between the anterior spinal artery and the perimedullary veins.[27] These are the main differences compared to dorsal AVFs, which are fed by segmental arteries and occur dorsal to the spinal cord.

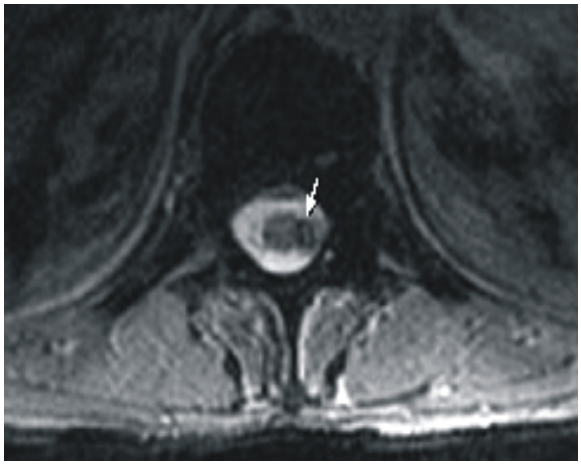
Intradural ventral AVFs can be subdivided in types 1, 2 or 3 based on the size of the fistula.[17] Good outcomes are usually obtained with endovascular treatment. If, however, endovascular therapy is unsuccessful, the fistula can be interrupted surgically. [6] The lack of nidus involving the spinal cord [27] constitutes an advantage that favors surgical treatment. In the case of larger fistulas, endovascular treatment is typically used as a surgical adjunct.[9] Precise identification of the angioarchitecture of the fistula, especially to understand the contribution from the anterior spinal artery, is key to avoid compromising blood flow distal to the fistula.

Aneurysms

Spinal aneurysms are rare.[31] They can manifest as compressive lesions but typically rupture causing spinal subarachnoid hemorrhage. They are a rare cause of intracranial subarachnoid hemorrhage [16] but should be considered when no other sources of subarachnoid hemorrhage are identified or when the subarachnoid hemorrhage is limited to the spine.

Spinal aneurysms differ from intracranial aneurysms.[23] They usually occur along the course of an artery as a fusiform dilatation. Partial thrombosis of spinal aneurysms, which is a frequent surgical finding, probably is the reason why they become symptomatic in some cases.

Treatment is planned on the basis of flow distal to the aneurysm on the parent vessel. If the aneurysm is distal in the circulation or thrombosed, the parent vessel can be sacrificed and the aneurysm resected. If there is flow distal to the aneurysm, the lesion can be wrapped to create a fibrotic scar that surrounds it and protects it from further bleeding.[38] In some cases when distal flow is present, the aneurysm can be resected and the parent vessel can be reconstructed with an end-to-end anastomosis (Fig. 8).[16]



Gonzalez 8

Figure 8. Axial T2-weighted MRI shows the extra-axial mass with a flow-void signal inside the lesion (arrow). From Gonzalez LF, Zabramski JM, Tabrizi P, et al: Spontaneous spinal subarachnoid hemorrhage secondary to spinal aneurysms: diagnosis and treatment paradigm. Neurosurgery 57:1127-1131, 2005. Used with permission from Lippincott Williams & Wilkins.

Endovascular techniques are limited by the small diameter of these vessels and by the fusiform and thrombosed features that are often present. In such cases, the only alternative is parent vessel occlusion with nBCA or coils.

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