Spinal Cord Vascular Malformations in Children

Debbie Song, MD[a], Hugh J.L. Garton, MD, MHSc[a], Daniel K. Fahim, MD[b], Cormac O. Maher, MD[a,*]

KEYWORDS

- Arteriovenous malformation • Arteriovenous fistula
- Cavernous malformation • Spinal cord
- Vascular malformation

Spinal vascular malformations comprise a diverse group of abnormalities, including arteriovenous malformations (AVMs), cavernous malformations, dural arteriovenous fistulas (AVFs), and capillary telangiectasias. These conditions each have distinct causes, presentations, radiologic appearances, natural histories, and treatments and are best considered separately. Although these lesions have been associated with a poor prognosis in the past, improvements in diagnosis and treatment have resulted in significantly better outcomes over the past 2 decades.[1,2] All of these lesion types may be found in the pediatric age range. This article explores the presentation, natural history, investigation, and treatment of spinal AVMs, spinal AVFs, and spinal cavernous malformations.

Various classification schemes exist to characterize arteriovenous lesions in the spinal cord. In any system of classification, a distinction should be made between AVFs and true AVMs. In 1993, Anson and Spetzler[3] attempted to classify spinal AVMs into 4 types. In this classification system, a type I AVM is actually a dural AVF, and types II, III, and IV are intradural lesions. A type II spinal AVM is an intramedullary glomus AVM characterized by a compact nidus of abnormal blood vessels within the spinal cord. Juvenile or metameric AVMs, referred to as type III lesions, are more extensive vascular lesions that can occupy the entire spinal canal at multiple levels and often extend into the paraspinal space.[4] Type IV malformations are perimedullary AVFs in which the transition from feeding artery to draining vein occurs without an intervening nidus of abnormal vessels.[4,5] Perimedullary fistulas can be further divided into 3 subtypes: a type IVa lesion, which is a small extramedullary fistula fed by a single arterial branch; a type IVb lesion, which is an intermediate-sized lesion supplied by multiple dilated arterial feeders; and a type IVc fistula, which is a high-flow, large-caliber, multipediculated fistula associated with dilated and tortuous vessels and a large shunt volume.[6]

The tendency for the classification system of Anson and Spetzler[3] to link AVFs with AVMs led Spetzler and colleagues[7] to propose a new classification system that draws a clearer distinction between these 2 distinct entities. This new classification system emphasizes the distinction between lesion type (eg, AVF or AVM) as well as location of the lesion within the spinal axis (eg, extradural, intradural extramedullary, and intradural intramedullary).[5] Intramedullary lesions may be further divided according to nidus type (eg, compact or diffuse). Intradural extramedullary fistulae are divided according to location (eg, ventral or dorsal).

[a] Department of Neurosurgery, University of Michigan, 1500 East Medical Center Drive, Room 3552, Ann Arbor, MI 48109-5338, USA
[b] Department of Neurosurgery, Baylor College of Medicine, One Baylor Plaza, Mail Stop BCM650, Houston, TX 77030, USA
* Corresponding author.
E-mail address: cmaher@med.umich.edu

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SPINAL CORD AVMS

Pial spinal AVMs are lesions with a vascular nidus present within the spinal cord parenchyma. These lesions can be compact or diffuse. Compact glomus AVMs account for approximately 90% of all intramedullary spinal AVMs and are supplied by multiple feeders from the anterior and posterior spinal arteries.[3–5,8] The venous outflow of these lesions is usually diffuse, occurring in the rostral and caudal directions and on the dorsal and ventral surfaces of the spinal cord.[3] Glomus AVMs are most commonly seen at the dorsal cervicomedullary junction.[8,9] As with cerebral AVMs, associated aneurysms may increase the risk of hemorrhage.[3,4] Surgical resection is the mainstay of treatment of such lesions, and preoperative embolization is useful in select cases.

Juvenile or metameric AVMs are extensive lesions that typically have an intramedullary component and extend into the extramedullary and extradural space. These complex vascular lesions can occupy the entire spinal canal and extend into the paraspinal space.[4,5] They may involve multiple spinal levels and are supplied by several large medullary arterial feeders.[4] Diffuse intramedullary AVMs can involve the subarachnoid space, pia, and parenchyma of the spinal cord. Normal intervening neural tissue may be contained between the intraparenchymal portions of the AVM.[5] Secondary bony remodeling changes that can be seen in patients with juvenile AVMs include enlargement of the spinal canal with a widened interpedicular distance and erosion of the laminae and pedicles.[9] These lesions typically affect children and young adults and may involve the bone, muscle, skin, spinal canal, spinal cord, and nerve roots of an entire somite level, as seen in disorders such as Cobb syndrome.[5,8] These extensive lesions are difficult to treat, with the goals of therapy being to reduce mass effect, venous hypertension, and vascular steal. Endovascular embolization is the treatment of choice, but open surgical treatment may be required for decompressive purposes as well.[5] Surgical resection of diffuse lesions involves ligating the vascular loops at the pial surface rather than following the lesions into the parenchyma of the spinal cord.[5]

Demographics and Incidence

Spinal AVMs are one-tenth as common as intracranial AVMs.[8] Spinal AVMs are rare entities in children. In a recent review of 267 patients who presented with a spinal AVM in a single year, 22% of patients were 18 years or younger.[10] Intradural spinal AVMs are more likely to affect children and young adults, whereas dural AVFs are more likely to occur in patients older than 40 years.[4] In a study of 81 patients with spinal AVMs, the average age of patients with a symptomatic dural AVF was 49 years, whereas the average age for patients with symptomatic spinal cord AVM was 27 years.[4] Almost two-thirds of patients with an intradural spinal AVM were younger than 25 years.[4] Among intradural spinal AVMs, glomus-type AVMs are most common.[3,11] Syndromes associated with spinal cord AVMs include hereditary hemorrhagic telangiectasia, neurofibromatosis type I, Klippel-Trénaunay-Weber syndrome, and Cobb syndrome.[3,11,12]

Clinical Presentation and Pathophysiology

Children with spinal AVMs can present with acute, subacute, or chronic symptoms of back or radicular pain, sensorimotor deterioration, bowel or bladder dysfunction, and myelopathy. Most patients with a spinal AVM are diagnosed as young adults; however, patients often become symptomatic during childhood.[4,13] Thus, a spinal AVM should be included in the differential diagnosis for any child who presents with slowly progressive or acute symptoms of radiculopathy or myelopathy.

Sudden focal pain with a neurologic deficit is thought to reflect an acute hemorrhage and is the most common presentation of a spinal AVM in children; 40% of patients younger than 14 years present with acute symptoms.[6,14] Children may also present in a subacute fashion with recurrent events associated with a neurologic deficit from which there is some element of recovery; with time, however, there is a gradual deterioration in baseline neurologic function because of repeated hemorrhages and alterations of blood flow associated with the malformation.[14]

Subarachnoid and/or intramedullary hemorrhage is associated with intradural AVMs and produces acute symptoms. In the series reported by Rosenblum and colleagues,[4] 52% of patients with an intradural spinal AVM experienced a subarachnoid hemorrhage (SAH), whereas none of the patients with a dural AVF suffered SAH. SAH was the initial presenting symptom in 31% of patients with an intradural spinal AVM; among intradural AVMs, SAH most commonly occurred in patients with glomus malformations.[4] Thus, for patients who present with nontraumatic SAH and who have a negative cerebral angiogram, obtaining cervical spine magnetic resonance imaging (MRI) is imperative for evaluation of a spinal cord vascular malformation. An acute onset of neurologic deficit is more often found in children with glomus lesions, reflecting the higher

incidence of SAH in these patients.[4] Less commonly, patients with glomus AVMs may present with progressive myelopathy due to mechanical compression or venous obstruction.[8]

High-pressure, high-volume, turbulent blood flow through intradural spinal AVMs can also lead to an arterial steal phenomenon that diverts blood away from the spinal cord parenchyma and results in ischemia.[4,14] Venous hypertension can also contribute to spinal cord ischemia, associated with progressive distention and engorgement of veins that drain intramedullary AVMs.[4,14] Progressive weakness is the most common initial symptom in patients with juvenile AVMs and perimedullary AVFs.[4,13] A sensory level may also be present in patients with spinal AVMs and reflects the spinal localization of the AVM nidus.[4] High-flow AVMs may also be associated with a spinal bruit or heart failure.[3,4]

Imaging

MRI is a sensitive modality for detecting spinal AVMs. Common findings include intradural signal voids reflecting dilated arteries or ectatic veins in or on the surface of the spinal cord, thrombosed veins, hematomyelia, spinal cord edema, syringomyelia, or spinal cord myelomalacia.[8] Intramedullary flow voids that are present in spinal cord AVMs appear as areas of hypointensity within the center of the spinal cord on axial T1-weighted images and as areas of hyperintensity on T2-weighted images (**Fig. 1**).[15] In addition, ectatic perimedullary veins and prominent flow voids in the subarachnoid space around the spinal cord are often visualized.[8]

Spinal angiography is the gold standard for diagnosing and defining the anatomy and angioarchitecture of spinal AVMs. The nidi of intradural AVMs are most commonly present within the spinal cord (80% of cases) but may less frequently be present on either the dorsal or ventral surface of the spinal cord.[3] Intradural spinal AVMs are often fed by multiple arterial feeders, are high-flow lesions, and can have single or multiple aneurysms associated with the arterial feeders or draining veins.

Treatment and Outcomes

Given the risk of hemorrhage and the cumulative risk of progressive neurologic deficit, treatment of spinal AVMs in the pediatric population is indicated in cases in which the estimated morbidity from treatment is acceptably low. Neurologic outcome after treatment has been directly correlated to preoperative motor function.[4] Other variables such as the age at symptom onset, degree of preoperative sensory loss, and rate of progression of neurologic deficit have not been shown to correlate with outcome.[4]

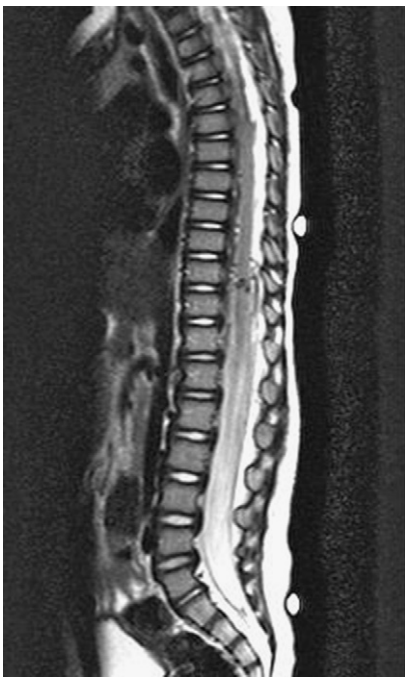

Fig. 1. Sagittal T2-weighted MRI illustrating the typical appearance of an intramedullary pial AVM of the spinal cord with dilated surface veins.

Surgical removal of glomus AVMs may be performed with similar principles that guide resection of intracranial AVMs.[16] Specifically, care is taken to identify and preserve draining veins until after the nidus is separated from the feeding vessels and surrounding parenchyma.[3] Intraoperative angiography can be technically challenging to perform for spinal malformations but may have a role in carefully selected cases.[17] Preoperative endovascular embolization may have a role in select cases.[18] Arterial side embolization as a treatment of intramedullary spinal AVMs is usually palliative at best and is rarely recommended as a primary treatment.[19] For endovascular treatment of spinal cord malformations, liquid embolic agents such as *n*-butylcyanoacrylate (NBCA) are preferred because they allow for easier filling of the distal nidus of a vascular malformation, they can be deposited in a more precise fashion, and they have a lower recanalization rate.[8] Complications from endovascular therapy can arise from inadvertent occlusion of arterial feeders supplying the spinal cord or venous branches draining the spinal cord. Intraoperative monitoring of somatosensory evoked potentials and motor evoked potentials is routinely

performed during both open microsurgical excision and endovascular embolization of spinal cord AVMs.

SPINAL AVFS

Spinal AVFs, which lack an intervening nidus of vessels in the transition between arterial feeders and draining veins, can be further divided into extradural and intradural AVFs. An extradural AVF is a rare, abnormal communication between an extradural arterial branch that arises from a radicular artery and an epidural venous plexus.[5] This can result in venous engorgement with subsequent mass effect on adjacent nerve roots and the spinal cord (**Figs. 2** and **3**). On spinal angiography, slow retrograde venous drainage of the lesion can be demonstrated in association with enlarged medullary veins.[4] Endovascular embolization is often an effective therapy.[5]

Intradural AVFs can be further divided into dorsal or ventral AVFs. Intradural dorsal AVFs have a radicular feeding artery that communicates with an intradural medullary vein in the dural sleeve of a proximal nerve root and adjacent spinal dura.[4,5] These lesions are most often fed by a single arterial feeder originating in the thoracic or lumbar region. Dilated tortuous veins that drain in a rostral direction are usually seen on the dorsal aspect of the spinal cord, and in some cases, along the ventral aspect of the spinal cord as well.[4] Patients often present with myelopathy that is due in part to venous hypertension and vascular steal. Progressive sensorimotor myelopathy with lower-extremity weakness is the most common presentation.[5] Symptoms may be exacerbated by activity.[3,4] Chronic venous hypertension is thought to play a critical role in producing neurologic deterioration. Elevated venous pressures occur as arterial blood passes through the

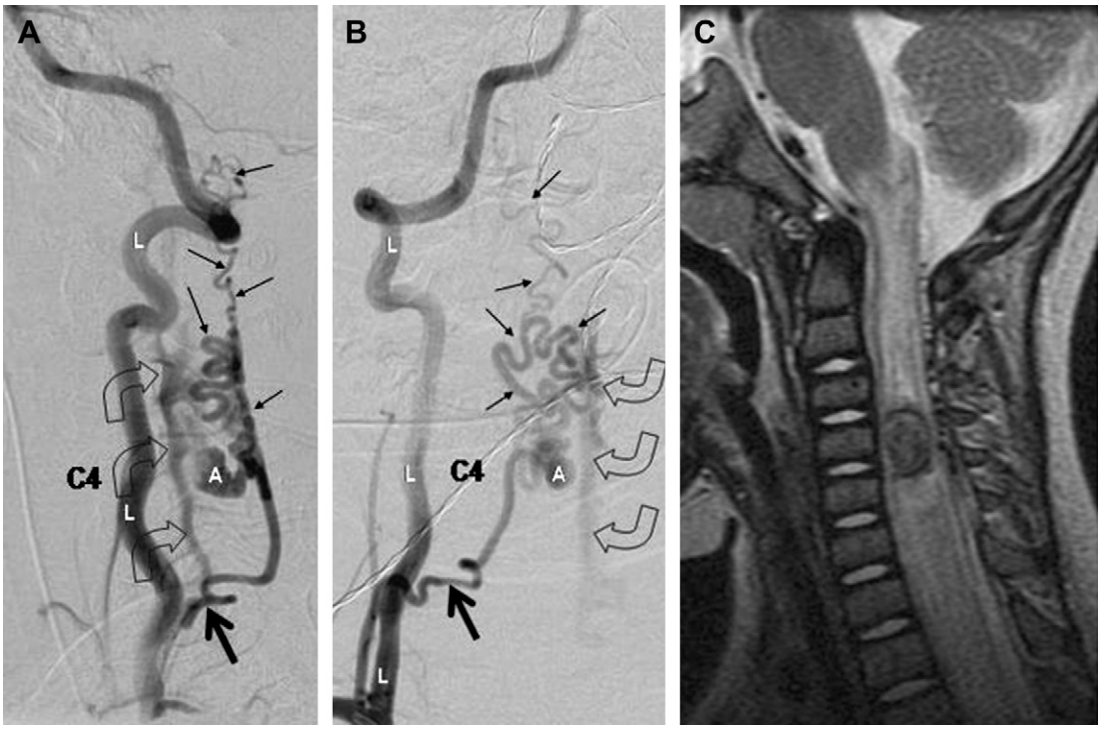

Fig. 2. Lateral (*A*) and anteroposterior (*B*) left subclavian or thyrocervical trunk angiograms (subtracted images; early arterial phase) reveal an enlarged radiculomeningeal branch of the left thyrocervical trunk entering the spinal canal at the C6-7 level (*large arrow*), which ascends to the C4-5 level. At the C4-5 level, an anteriorly oriented aneurysm (A) is identified. Further extending superiorly from the C4-5 level to the level of the foramen magnum are multiple serpiginous vessels representing engorged arterialized veins (*small arrows*). Finally, early filling of normal-appearing epidural venous structures is seen initially at the C3 and C4 levels with caudal drainage (*curved arrows*). The observed constellation of findings suggest an atypical perimedullary fistula at the C4 level giving rise to an aneurysm, arterialized veins extending cephalad, and early filling of epidural venous structures. Incidentally noted is transient reflux of contrast material into the left vertebral artery (L). Sagittal MRI (*C*) illustrating an aneurysm within the cervical cord.

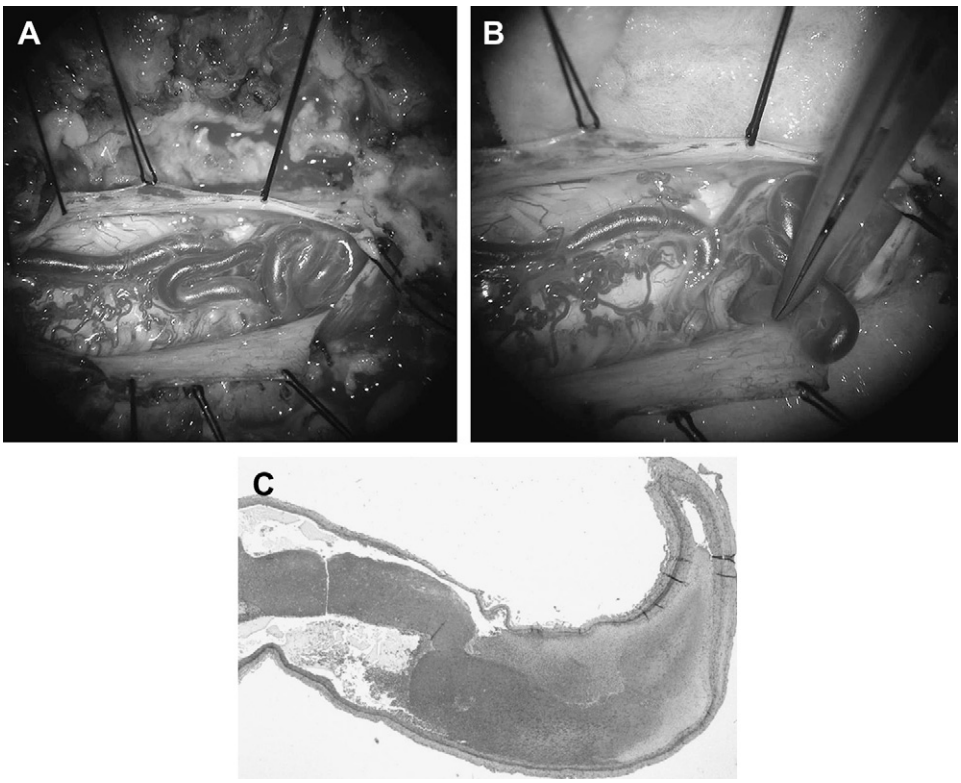

Fig. 3. Intraoperative views (*A* and *B*) of the patient represented in **Fig. 2**. The large aneurysm is removed and the arterialized vein is disconnected. The junction between the arterial and venous segments may be appreciated in the histologic specimen (*C*).

fistula into a draining medullary vein and transmits relatively high pressure to the valveless venous plexus and radial veins associated with the spinal cord.[4] These lesions may arise spontaneously or after trauma.[4,8,10]

Although spinal angiography remains the gold standard, magnetic resonance angiography is useful for predicting the level of the fistula before angiography, leading to decreased radiation and contrast dose.[20] For these reasons, preangiographic magnetic resonance investigation is especially useful in the pediatric age group. Intradural dorsal AVFs are best treated via surgical disruption of the fistula.[5,21] From a posterior approach, the arterialized vein should be isolated, cauterized, and ligated at its exit point from the margin on the dural nerve root sleeve.[5] This operation is straightforward, curative, and associated with minimal morbidity.[21]

An intradural ventral AVF is usually located in the midline and consists of a fistulous communication between the anterior spinal artery and an enlarged venous network in the subarachnoid space.[5] Intradural ventral AVFs can be small with a single feeder or they can be giant lesions with large, multipediculated feeders from the anterior spinal artery that communicates with dilated venous channels.[5,10] Smaller-sized lesions may be treated surgically via anterior or posterior approaches, with care taken to preserve anterior spinal artery branches.[5]

PARASPINAL AVMs

Paraspinal AVMs in children are rare, with less than 2 dozen reported cases in the literature.[22–24] These lesions can involve the paravertebral musculature, intervertebral foramen, or paravertebral region. Although such lesions may occur at any spinal cord level, in children they are most commonly found in the thoracic spine.[24] They can cause spinal cord dysfunction by spinal venous hypertension that leads to progressive myelopathy or by hemorrhage into the spinal canal that is associated with acute paraplegia. Paraspinal AVMs may manifest with a bruit or murmur, weakness, congestive heart failure secondary to arteriovenous shunting, myelopathy from mass effect of enlarged venous varices, or low back pain.[24] These lesions may be congenital or have

a posttraumatic cause. Among children with paraspinal AVMs, presentation is bimodal, with most children presenting either before the age of 4 years or after 10 years.[24] Spinal angiography is the modality of choice in evaluating paraspinal AVMs, with MRI being a useful adjunct that can reveal engorged intrathecal and paraspinal veins (**Fig. 4**).[24] Successful obliteration of paraspinal AVMs in children can be performed via endovascular embolization with liquid embolic agents such as NBCA.[22–24]

CAVERNOUS MALFORMATIONS

Spinal cord cavernous malformations are rare lesions that are thought to account for 3% to 5% of all central nervous system cavernomas.[3] They are even more unusual in children, with only a handful of cases of spinal cord cavernous malformations in the pediatric population reported in the literature.[25–29] Histologically, cavernous malformations are characterized by thin-walled, dilated, sinusoidal endothelial channels lined with a subendothelial stroma that lacks smooth muscle and elastic tissue.[8,28] There is no intervening spinal cord parenchyma between the vascular channels in such lesions.[28] Cavernomas can occur throughout the neuraxis and often contain hemosiderin-laden macrophages, indicating hemorrhages of varying ages.

Despite the scarcity of cases of spinal cord cavernomas in children reported in the literature, differences exist in the clinical presentations of adults and children with such lesions. Whereas spinal cord cavernomas have a 2:1 predilection for women among adults, they are more common in boys than girls in the pediatric population.[3,28] Among children with spinal cord cavernous malformations, the mean age at which an initial symptomatic hemorrhage occurs is around 13 years.[28] Spinal cord cavernous malformations in children are evenly distributed throughout the thoracic and lumbar spinal cord, whereas they are most commonly located in the thoracic spinal cord in adults.[28] Adults with spinal cord cavernous malformations present with a slow, progressive myelopathy or with an acute deficit followed by a stuttering pattern of deterioration with intervening periods of clinical improvement. This is thought to reflect multiple hemorrhages or thromboses within the cavernoma.[28] In contrast, children with symptomatic spinal cord cavernomas often present with a severe and acute neurologic deficit followed by a rapid decline.[10,26] Common symptoms include radiating low back pain with weakness and sensory changes. Although these clinical features have been reported in a series of pediatric spinal cavernous malformations, it is difficult to draw definitive conclusions from the paucity of available data.

Spinal cord cavernomas are best visualized on MRI, as they are occult on spinal angiography. They appear as well-defined, intramedullary masses of varying sizes and mixed signal intensity on T1- and T2-weighted spin echo sequences.[3,8,28] Cavernous malformations have hyperintense areas on T1- and T2-weighted images with a peripheral rim of hypointensity on gradient echo sequences, which reflects the hemosiderin ring. Spinal cord cavernous malformations may be associated with multiple cavernomas elsewhere in the neuraxis or with familial cavernoma syndromes.[30] Thus, MRI of the entire neuraxis is indicated when a spinal cord cavernous malformation is identified in isolation.

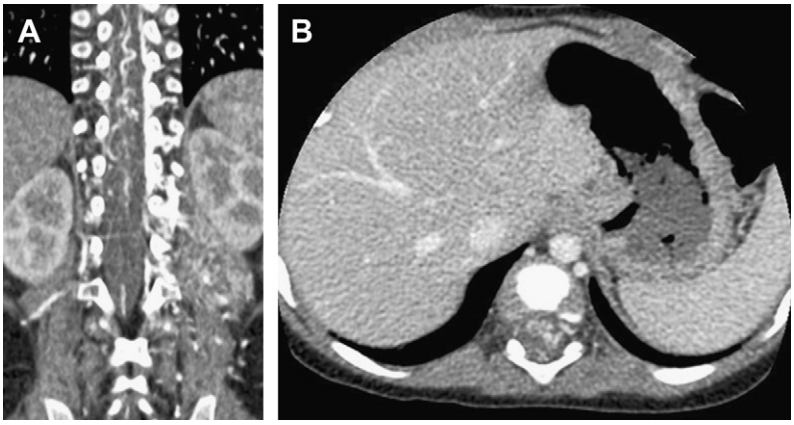

Fig. 4. Coronal (*A*) and axial (*B*) CT angiography illustrating a paraspinal AVM with multiple arterial feeders from the abdominal aorta and a nidus in the paraspinal musculature. The perimedullary venous plexus is dilated as a consequence of the fistula.

Spinal cord cavernous malformations have a higher risk of hemorrhage that has a greater potential for causing significant morbidity compared with supratentorial cavernomas. The actuarial risk of hemorrhage for a supratentorial cavernous malformation has been estimated to be 0.2% to 0.7% per year; the risk is 1.6% per year for spinal cord cavernomas and 2.7% per year for brainstem cavernomas.[3,28] Given the cumulative risk and greater morbidity of hemorrhage that is faced by children with spinal cord cavernous malformations, treatment via microsurgical excision is typically favored for those lesions that have bled or for asymptomatic lesions that present with an accessible pial surface. Surgical treatment is generally not recommended for asymptomatic spinal cord cavernomas that do not present to a pial surface. Typically, such lesions may be followed expectantly, with regular clinical and imaging surveillance. Microsurgical excision is the only means of curing a cavernous malformation. Radiosurgery is not an acceptable method of treatment. Most lesions can be surgically approached via dorsal laminectomy, but a posterolateral transpedicular approach may be required for more ventrally located lesions.[3] In children, laminoplasty with replacement of the bone and preservation of the supraspinous ligament is preferred to prevent long-term postoperative spinal deformities. For lesions that present at the pial surface, dissection can be performed directly over the lesion. For deeper lesions, a midline myelotomy or myelotomy in the dorsal root entry zone is performed. Dissection is performed in the thin plane of hemosiderin-laden gliosis around the cavernoma to limit trauma to the normal surrounding spinal cord tissue. Grossly, cavernous malformations have a distinctive purple mulberry appearance with a surrounding rim of yellow-brown discoloration from hemosiderin deposits. In contrast to cerebral cavernomas, the thin rim of hemosiderin-stained perilesional gliosis should not be removed in spinal cord cavernous malformations.[3,28] Complete excision of spinal cord cavernous malformations can usually be achieved safely, and generally, it has a good long-term outcome. As with spinal cord AVMs, the most predictive factor of surgical outcome is the patient's pretreatment neurologic status.[3]

SUMMARY

Spinal cord and paraspinal vascular malformations are rare entities in the pediatric population. Each of these lesion types has distinct clinical presentations and treatments. In general, the most common predictor of neurologic outcome after treatment is the pretreatment neurologic function.

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