Spine and Spinal Cord Emergencies: Vascular and Infectious Causes

Vincent Y. Wang[a], Dean Chou[a] and Cynthia Chin[b],*

EPIDURAL ABSCESS

Spinal epidural abscess can develop primarily or after spinal surgery. *Primary* spinal epidural abscess is a rare entity, with an estimated prevalence of 0.2 to 2.0 cases per 10,000 hospital admissions.[1] However, its incidence has increased over the last 20 years as a result of better diagnostic methods, an aging population, an increase in the number of patients with medical comorbidities, and an increase in intravenous drug use.[1,2] Most cases occur in middle-aged to elderly adults, with a predilection for men.[1–3] Risk factors include diabetes, intravenous drug use, previous invasive procedures to the spine, superficial infections of the back, immunocompromised state, and infections at distant sites.[1–4] *Staphylococcus aureus* is the most common pathogen and is found in approximately 70% of cases.[1–3,5] Gram-negative rods were previously reported to be associated with a significant number of spinal epidural abscesses and vertebral osteomyelitis; however, its prevalence has been decreasing over the years.[1,6] In rare cases, other pathogens, such as tuberculosis, fungal, and parasitic agents, can also be associated with a spinal epidural abscess.[1,7]

The classic clinical triad of fever, back pain, and neurologic deficits is found in only a small fraction of patients.[2] Almost all patients have back pain, but fever occurs in only about two-thirds of patients, and neurologic deficits occur in 25% to 60% of patients.[1,3,8] Therefore, many patients do not seek medical treatment with the onset of symptoms and a delay in diagnosis is not uncommon.[4,9]

In terms of location, epidural abscess can occur in all 3 regions of the spine (cervical, thoracic, and lumbosacral). Of the 3 regions of the spine, the thoracic and the lumbosacral regions are affected more commonly than the cervical spine.[1,5,8] There are also cases of a spinal epidural abscess involving the entire spine.[8,10]

The spinal epidural abscess can cause neurologic deficits by either direct compression or by vascular compromise through thrombophlebitis or thrombosis.[11] Thus, sudden neurologic deterioration can occur and prompt diagnosis is important. MR imaging is the initial study of choice for identifying a spinal epidural process of any etiology. In the case of an abscess, the diagnosis is often straightforward: isointense or hypointense on T1-weighted images and hyperintense on T2-weighted images.[3,8] The collection usually partially enhances with gadolinium-based intravenous contrast media (Gad).[3,8] The fluid portion of the abscess is typically hyperintense on T2-weighted images.[12] The enhancement pattern sometimes helps to define the consistency of the collection. Specifically, liquid purulent material is associated with a central area of low T1 signal intensity; whereas, a rim of tissue that enhances after Gad represents granulation tissue.[12] Recently, diffusion-weighted imaging (DWI) has been used

[a] Department of Neurological Surgery, University of California, 505 Parnassus Avenue, MS112, San Francisco, CA 94143, USA
[b] Section of Neuroradiology, Department of Radiology, University of California, San Francisco, San Francisco, CA 94143, USA
* Corresponding author.
E-mail address: cynthia.chin@radiology.ucsf.edu

in the spine; on DWI, the abscess is markedly hyperintense to the surrounding tissue and hypointense on the apparent diffusion coefficient map (Fig. 1).[13] More than 80% of patients with spinal epidural abscess have concomitant osteomyelitis.[2,3] Therefore, in patients with a suspected spinal epidural abscess, it is important to look for additional findings of osteomyelitis or diskitis.

Patients who are unable to undergo MR imaging can be studied with CT myelography (CTM), which has been shown to have similar sensitivity to MR imaging for detecting an epidural abscess.[14] MR imaging has the advantage of being more specific in its ability to differentiate the epidural mass from other pathologies. In addition, it is superior for evaluating the adjacent paraspinal soft tissues. Moreover, CTM requires intrathecal injection of contrast material and therefore has the theoretical risk of introducing the infectious agents into the intrathecal space in cases of lumbosacral abscess. Nevertheless, CTM may be an alternative diagnostic method for patients who are unable to undergo an MR imaging examination and for patients in whom MR imaging may be difficult to interpret because of previous spinal instrumentation. Although MR imaging, and to a lesser extent CTM, is a key diagnostic study to establish the diagnosis of a spinal epidural abscess, plain radiographs or CT of the spine are often the first diagnostic tests that are obtained, especially in patients who are afebrile without neurologic deficits. Surgical decompression with antibiotic treatment have historically been the mainstay of treatment for spinal epidural abscess.[1–5,8] Recently, nonsurgical treatment has been used to treat patients with no neurologic deficits, significant comorbidities, or complete spinal cord injury at the affected level below the level of epidural abscess.[2,3]

EPIDURAL HEMATOMA

Spinal epidural hematoma (EDH) can compress the spinal cord or nerve roots and result in neurologic deficits. Spinal EDHs occur most commonly after spinal surgery.[15] After discectomy or decompression, EDH can be identified in 33% to 100% of the patients when evaluated by postoperative CT or MR imaging.[15] However, despite the radiographic appearance of compression by many of these lesions, neurologic symptoms associated with postoperative EDH are extremely rare.[15] It is estimated that the incidence of symptomatic EDH after spinal surgery is only 0.1% to 0.2%.[15] Risk factors associated with EDH formation include advanced patient age, multilevel surgery,
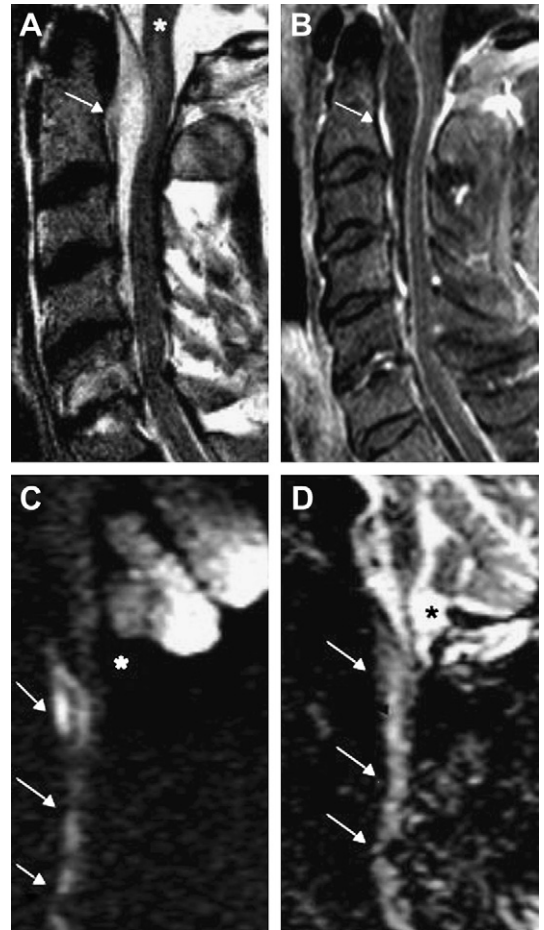

Fig. 1. Methicillin-resistant *Staphylococcus aureus* spinal epidural abscess. 58-year-old man with diabetes with progressive weakness. Sagittal fast spin echo T2 (*A*) and postgadolinium T1 with fat saturation (*B*) sequences through the cervical spine demonstrate extensive circumferential peripherally enhancing epidural abscess (*arrow*) with cord compression and associated cord edema (*asterisk*). Sagittal diffusion-weighted imaging (*C*) demonstrates increased signal, and apparent diffusion coefficient map (*D*) demonstrates decreased signal (*arrows*) relative to cerebrospinal fluid (*asterisk*) compatible with reduced diffusion within the epidural abscess.

and coagulopathy (increased international normalized ratio).

In contrast, *spontaneous* EDHs of the spine are rare. The incidence of spontaneous spinal epidural hematomas is estimated to be 0.1/100,000 population per year.[16] They usually occur in older patients (aged 50–80 years) with a slight predominance of male patients.[16] Limited evidence suggests that the spontaneous spinal EDH is caused by bleeding from the valveless venous plexus, although some investigators postulate

that arterial rupture may be the cause.[16] In most cases, there is no clear source of hemorrhage or a causative event. There have been several case reports of spinal epidural hematomas occurring after chiropractic manipulation, epidural anesthesia, and steroid injection.[17,18] The incidence of spinal EDH after chiropractic manipulation is extremely low, with fewer than 10 cases reported in the literature.[17] The risk of spinal EDH after epidural anesthesia is also low, estimated to be about 1:220,000.[19] It is thought that coagulopathy increases the risk of spinal EDH in these latter cases.[17,18]

The classic clinical presentation of a spinal EDH consists of the sudden onset of back or neck pain. Neurologic symptoms related to the compression of nerve roots or spinal cord may also occur.[20] Many pathologic conditions, including intervertebral disc herniation, infection, pathologic fractures associated with tumor, transverse myelitis, and other vascular malformations, may mimic the clinical presentation of a spinal EDH. Therefore, prompt evaluation with MR imaging is important to establish the diagnosis.

The typical imaging appearance of an acute spinal EDH is that of a homogenously isointense to hyperintense mass on T1-weighted images, and hyperintensity on T2-weighted images.[20] The hematoma can have homogenous or heterogeneous hyperintensity on T2-weighted images.[20] Atypical appearance of the hematoma usually involves a shift from isointense to hyperintense on T1-weighted images.[20] Importantly, the acute spinal EDH *should not* enhance with gadolinium-based intravenous contrast media.[20] In one series of 19 subjects with a diagnosis of spinal EDH, 2 cases showed Gad enhancement on MR imaging and the intraoperative findings revealed that neither case was an EDH.[20] Most hematomas occur in the cervical or the thoracic region (**Fig. 2**).[18]

In the majority of cases, emergent surgical evacuation of the hematoma is the treatment of choice. Patients with small hematomas and without neurologic deficit may be managed conservatively.[17,20] For patients with neurologic deficits, it is important to remove the hematoma. Further, because neurologic improvement has been observed even in patients presenting with complete injury, prompt recognition, diagnosis, and treatment are essential for the optimal management of the spinal EDH.[20]

ARTERIOVENOUS SHUNTS

Arteriovenous shunts and vascular malformations in the spinal cord region are classified based on
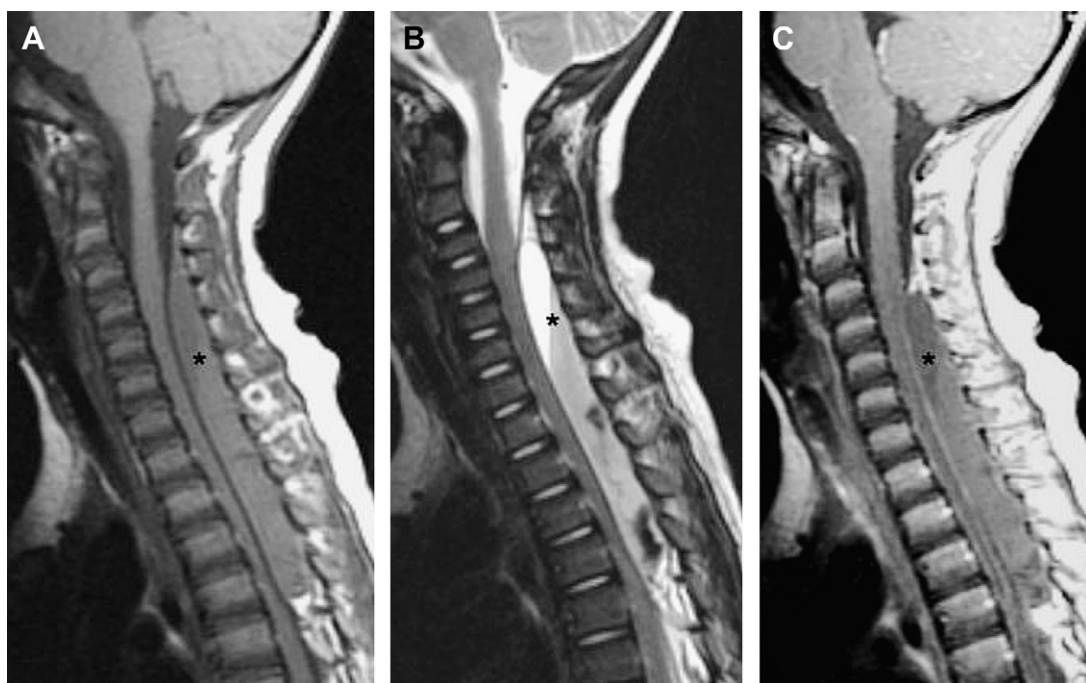

Fig. 2. **Spontaneous epidural hematoma.** 3-year-old healthy girl with 4-day history of back pain progressing to lower extremity weakness and inability to ambulate. Sagittal T1 (*A*) fast spin echo T2 (*B*) and postgadolinium T1 with fat saturation (*C*) images of the cervical spine demonstrate extensive nonenhancing dorsal epidural hematoma compressing the spinal cord. A hematocrit level is present within the epidural hematoma (*asterisks*).

their nidus location, vascular supply, and drainage pattern. In one of the most commonly used classifications, developed by Oldfield and Doppman, the lesions are divided into 4 types:[21]

> Type I lesions are the typical dural arteriovenous fistula (DAVF), and usually arise near nerve roots.
> Type II lesions are glomus arteriovenous malformations (AVM), with a mass of dysmorphic arteries and veins without an intervening capillary bed. These lesions can be partial or entirely intramedullary.
> Type III lesions are juvenile AVMs that are composed of a network of arteries and veins, without an obvious nidus. These lesions can be extensive and involve the spinal cord, spine, and paravertebral tissue.
> Type IV lesions are perimedullary DAVF.

Combined, these 4 types of arteriovenous shunts or vascular malformations account for 3% to 16% of all space-occupying lesions of the spinal cord, with the Type 1 DAVF being the most common.[22] Each of these types are subsequently discussed.

Spinal vascular malformations/arteriovenous shunts can cause neurologic symptoms by 3 mechanisms:

1. These lesions can cause acute neurologic symptoms by hemorrhage, and the risk of hemorrhage varies for the 4 types of lesions.
2. Spinal vascular malformations and arteriovenous shunts can result in venous hypertension. Venous hypertension, in turn, can lead to reduced perfusion, and thus ischemia.
3. They can cause symptoms by direct mass effect, resulting in a progressive myelopathy or radiculopathy.

In one study of 78 subjects with myelopathy of unknown cause, 22 subjects had an AVM on angiography.[23] Importantly, 5 subjects in this series had previous MR imaging that was interpreted as normal.[23]

Type I: Dural Arterial Venous Fistula

The spinal dural arterial venous fistula is an acquired arteriovenous shunt in the dura.[22] It is most commonly located near the exiting nerve root.[22] In some cases, the fistulous vessel may penetrate the dura at a more distant site and have a significant extradural component.[24] Spinal DAVFs usually present in adults after the fourth or the fifth decade, have a significant male predominance, and most commonly affect the thoracic spine.[22] Patients often present with vague complaints of back pain or radicular pain, followed by progressive lower-extremity paresis, and bowel and bladder sphincter dysfunction.[22,25,26] Hemorrhage is extremely rare.[22,26] Histologically, spinal DAVFs are supplied by a normal dural branch artery and drain into a single, dilated vein.[27] This drainage results in retrograde flow into the venous plexus surrounding the spinal cord. Physiologic studies have shown that venous pressure is approximately 75% of systemic arterial pressure.[25] The resultant venous hypertension and venous congestion cause a reduction in the arterial-venous pressure gradient, and thus a reduction in tissue perfusion pressure. A reduction in tissue perfusion pressure then leads to hypoxia, and further vasodilation may occur as a result of autoregulation.[22] Eventually, compensatory mechanisms from autoregulation are exhausted, the spinal cord tissue becomes ischemic. Cord edema and progressive loss of function then develops.

Although most patients present with *progressive myelopathy*, acute worsening of motor or sphincter function can also occur.[24] It is important to evaluate patients with an unclear cause of myelopathy, because 50% of untreated patients with a spinal DAVF can become disabled in 3 years.[28] MR imaging is the initial screening test of choice, and it is important to obtain a *whole-spine MR imaging survey* because there can be a discrepancy between the location of the spinal DAVF and the spinal level as suggested by clinical signs and symptoms.[28] The most common finding on MR imaging in patients with DAVF is abnormal T2 signal hyperintensity within the cord.[22,28] There can also be enlargement of the cord, consistent with swelling from cord edema.[29] The T2 signal changes can be extensive, extending over 6 to 7 vertebral levels in some cases.[30] Occasionally, prominent, lacelike tortuous and ectatic flow voids may be seen.[22,31] There can also be nonspecific enhancement with Gad contrast (**Fig. 3**).[22]

None of the aforementioned imaging findings are specific for DAVF. The current gold standard for imaging the DAVF is with digital subtraction angiography (DSA). Angiography allows a clear definition of the anatomy of the DAVF, including its location, arterial supply, and venous drainage pattern.[22,24–26] In some cases, the DAVF can obtain arterial supply from 2 arteries located in adjacent nerve roots.[22] One of the most important angiographic details to assess is the *origin of the anterior spinal artery*. If the anterior spinal artery shares a common origin with the supply to the DAVF, then the DAVF is not amendable to endovascular treatment.[25]

Fig. 3. Spinal dural arterial venous fistula. 65-year-old man with progressive lower extremity weakness. Sagittal fast spin echo T2 (*A*) images of the thoracic cord demonstrate extensive cord edema (*asterisk*) with flow voids along the dorsal spinal cord (*black arrows*). There is associated cord enhancement demonstrated on sagittal postgadolinium T1 fat saturated sequences (*B*).

Although DSA provides high temporal and spatial resolution imaging information of the DAVF, it is not without its drawbacks. DSA is often time consuming, as many as 40 injections can be needed to catheterize the bilateral intercostal, lumbar, and sacral arteries.[30] This procedure requires a high dose of iodinated contrast and increased radiation exposure. In addition, each catheterization also poses a small risk of vascular injury and can cause transient neurologic symptoms.[31] Thus, there is interest in developing noninvasive imaging technology to optimally evaluate the spinal DAVF. Two main technologies, CT angiography and MR angiography, have shown some promising developments. Lai and colleagues[32] reported their experience of 8 subjects with 16-row multidetector CT (MDCT) angiography for DAVF. In all 8 subjects, MDCT angiography correctly identified the enlarged radiculomedullary draining vein, the fistula, and the feeding artery.[32] Only 1 subject had an additional feeding artery identified on the subsequent DSA that was not identified on the MDCT angiography.[32] Similarly, Yamaguchi and colleagues[33] reported the successful identification of 2 out of 3 subjects with a DAVF. In the one case where MDCT angiography failed to identify the fistula, DSA also failed to identify the DAVF,

and the fistula was only identified at surgery.[33] In a more recent report, Si-jia and colleagues[31] noted that the spinal DAVF could be readily identified by MDCT angiography using a 64-row detector. In their series of 9 subjects, all of the draining veins were identified, and in 7 of 9 subjects, the feeding artery was also found.[31] Another recent study by Yamaguchi and colleagues[34] evaluated 10 subjects with 16-row detector MDCT angiography and DSA. Dilated perimedullary veins were found in all 10 subjects, focal enhancement of nerve root and intradural draining veins were noted in 8 subjects, and localization of the feeding artery was correctly depicted in 6 subjects. In 2 subjects, there were additional feeding arteries from the contralateral side that were not depicted on the MDCT angiography but were visualized on conventional angiography.[34] Although these findings are promising in that MDCT angiography can reveal a spinal DAVF, the temporal and spatial resolution of MDCT is still inferior to that of DSA. Therefore, the anterior or posterior spinal artery cannot be distinguished easily from that of the feeding artery.[29,31] In addition, the field of view is often more limited and it is difficult separating the arterial from venous phase.[29] MDCT angiography also exposes patients to radiation.

MR angiography (MRA) has been gaining attention in the diagnosis of spinal DAVFs. Rapid multiphase dynamic MRA with gadolinium and parallel imaging can achieve better temporal and spatial resolution than MDCT angiography.[29] However, increase in temporal resolution may come at an expense of the spatial resolution and signal-to-noise ratio is reduced.[30] Using time-resolved MRA, Ali and colleagues[30] studied 12 subjects with suspected DAVF based on clinical symptoms and MR imaging findings. In 6 subjects with a DAVF confirmed by DSA, all of them also demonstrated arteriovenous shunting on the time-resolved MRA.[30] Furthermore, the location of the arteriovenous shunting identified on MRA was within one vertebral level as identified on DSA.[30] Others have used contrast-enhanced MRA to study DAVFs. Using elliptic centric contrast-enhanced MRA, Luetmer and colleagues[35] depicted DAVFs in 20 out of 22 subjects who had DAVFs identified on conventional angiography. In 14 subjects, the level of the fistula was within the volume of imaging, and MRA correctly located the fistula to within one level of the spine in 13 subjects.[35] However, MRA does not always correctly identify the type of lesion. In one case, Sharma and Westesson reported that although the MRA showed a convincing type I DAVF, subsequent conventional angiography revealed a type IV perimedullary AVF.[36]

Although both MDCT angiography and MRA show promising results, they are still considered to be inferior to conventional angiography; however, both techniques help demonstrate the location of the fistula, which may minimize the number of catheterizations during DSA. Furthermore, newer techniques will continue to improve the temporal and spatial resolution of MDCT and MRA. These developments may one day enable these noninvasive techniques to become the primary diagnostic modality for studying spinal DAVFs.

Because of the potential for neurologic deterioration over time, prompt treatment of the spinal DAVF is advocated. Microsurgery has been the traditional method of treatment. In one meta-analysis, Steinmetz and colleagues[37] showed a 98% success rate of fistula obliteration with microsurgery. During recent years, endovascular treatment has also been used to treat spinal DAVFs. Although early experience demonstrated only limited success, with an overall success rate of only 46%, recent advances in embolization material and the possibility of being able to perform the treatment during the same setting of the diagnostic angiogram make endovascular therapy an option employed as a first line of treatment.[25,37,38] Surgical treatment can then be performed if endovascular therapy is unsuccessful or contraindicated. In addition, endovascular treatment is not advised for patients with a feeding artery that shares a common origin with the anterior or posterior spinal artery.[25]

Type II: Glomus Arteriovenous Malformation

Type II lesions, or glomus AVMs, have a nidus that is located completely or partially *within the spinal cord*.[25] These AVMs can be supplied by either the ventral radiculomedullary or anterior spinal arteries, or the dorsal radiculopial or posterior spinal arteries.[22] In 20% to 40% of cases, there is an associated arterial or venous aneurysm.[25] In 20% of cases, the AVMs are supplied by multiple shunts.[22]

Compared with the Type I spinal DAVF, the Type II glomus AVM tends to present in *younger* age, usually in young adults aged 20 to 30 years.[22,25] In 20% of cases, it affects pediatric patients.[22] In one series of pediatric subjects with spinal vascular malformations/arteriovenous shunts, AVMs were found in 44% of the subjects, representing the most common vascular malformation in children.[39] Also, unlike the DAVF, AVM most commonly presents with hemorrhage and *sudden onset* of neurologic symptoms.[22] There is a high

rebleeding rate if the AVM is not treated; therefore, prompt recognition and treatment is warranted.[22]

MR imaging is currently the imaging modality of choice for the initial evaluation of spinal AVMs. MR imaging may show areas of low signal intensity in the center of the cord (representing either vascular flow voids or blood products) and hyperintensity on T2-weighted images.[22] Moreover, MR imaging can show the relationship between the lesion and the spinal cord and dura, identify recent or remote hemorrhage and thrombosis, and reveal any paravertebral involvement (**Fig. 4**).[22]

DSA remains the definitive test for the diagnosis and treatment planning of spinal AVMs. The goal of angiography is to define the normal vasculature around the lesion as well as the diseased vasculature of the lesion. Features to look for include perimedullary and intramedullary anastomoses, direct and indirect AVM supply, collateral recruitment, associated aneurysms and pseudoaneurysms, venous drainage, and normal spinal cord vascular supply.

The goals of treatment include preservation of neurologic function and minimization of rebleeding. Treatment options include surgical excision, endovascular obliteration, or conservative management. Spetzler and colleagues[40] reported that 92% of subjects who underwent surgery had complete resection, with 68% of subjects experiencing neurologic improvement and 29% of subjects remaining neurologically unchanged. Others have reported favorable experience with endovascular treatment. For example, Da Costa and colleagues[22] reported that in a series of 47 subjects with spinal AVMs treated with endovascular obliteration, 77% of subjects had a favorable result, with about half of the subjects achieving complete obliteration. However, 22% of subjects experienced procedure-related complications, with 50% being permanent.[22] Conservative therapy includes physical therapy, pain control, and possible anticoagulation in patients with thrombosis.[22]

Type III: Juvenile Metameric Vascular Malformation

Type III lesions, also known as juvenile vascular malformations or metameric vascular malformations, are large, high-flow lesions with involvement of the *paraspinal tissues*.[25,40] These are uncommon lesions; among the 48 AVMs treated surgically by Spetzler and colleagues,[40] only 5 were metameric vascular malformations. Similarly, in a series of 72 pediatric subjects with spinal vascular malformations/arteriovenous shunts, only 3 had paravertebral tissue involvement.[39] As
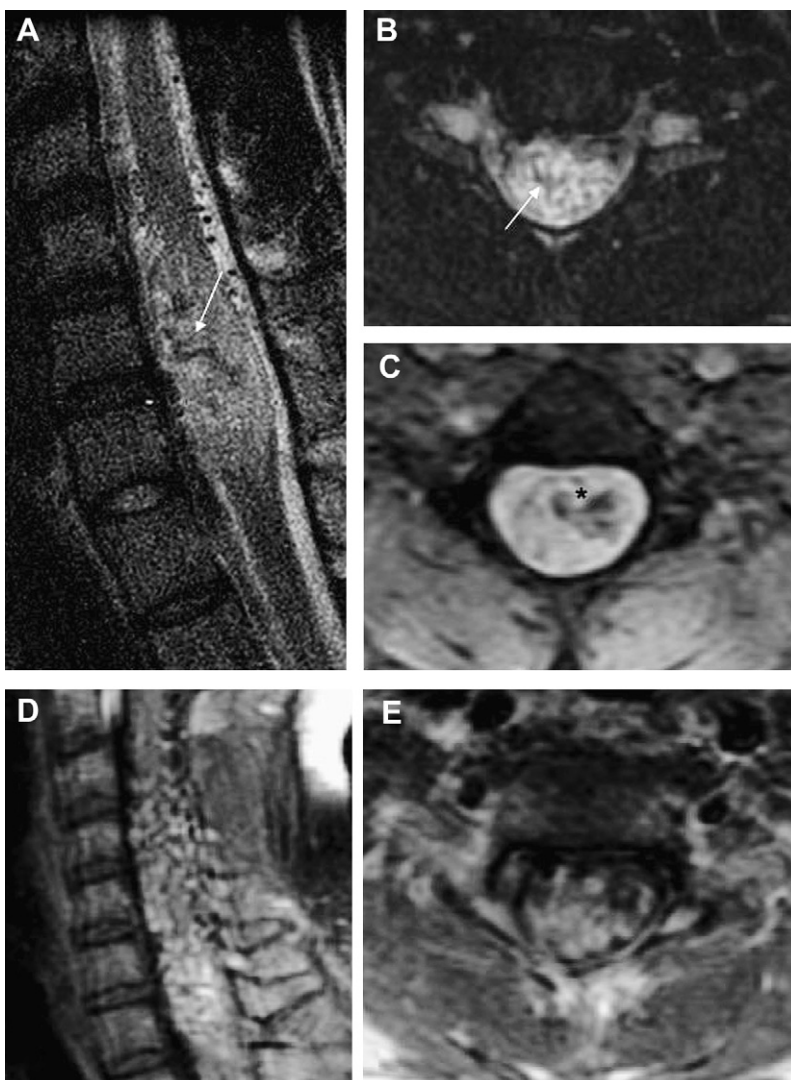
Fig. 4. **Glomus arteriovenous malformation.** 21-year-old man with right leg weakness. Sagittal (*A*) and axial (*B*) fast spin echo T2 sequences of the cervical spine demonstrate cord expansion, intramedullary edema, and flow voids (*arrows*) with associated enhancement on postgadolinium sagittal (*D*) and axial (*E*) T1 fat saturated images. Axial gradient echo sequence (*C*) shows evidence of cord hemorrhage (*asterisk*).

the name implies, these lesions commonly present in the pediatric population. Neurologic deficits occur from compression, hemorrhage, or vascular steal phenomenon.[40] MR imaging remains the initial diagnostic modality of choice, because it nicely reveals the extent of the lesion in the paravertebral tissue. DSA reveals the flow dynamics and feeder pattern, which are essential for treatment planning. A multidisciplinary approach is often required to address these lesions, with a combination of embolization and surgical resection; however, complete resection and obliteration is often difficult without incurring significant neurologic morbidity.[40]

Type IV: Perimedullary Arteriovenous Fistula

The perimedullary AVF is usually located in the *midline* and *ventrally* in the subarachnoid space.[40] The lesion consists of an abnormal connection between the anterior spinal artery and an enlarged venous network.[25,40] Blood flow through the lesion can be rapid, and therefore flow-related acquired lesions, such as aneurysms or ectatic venous malformations, can develop.[25,40] They most commonly present in young adulthood, but they can also present in the pediatric population.[39,41] The most common presentation is a progressive myelopathy, but rupture of an associated

aneurysm resulting in hemorrhage can also be the first sign and symptom.[41]

Perimedullary AVFs have been further subclassified into 3 subtypes. Type I perimedullary AVF are small, with a single feeder and low flow fistula, and mild to moderate venous hypertension.[25,41] Type II perimedullary AVFs are larger, supplied by 1 or 2 spinal medullary arteries, and have a more rapid transit time.[41] Type III perimedullary AVFs are the most common. These are giant lesions with multiple dilated feeding arteries and rapid transiting. The fistula drains into greatly dilated and dysplastic veins.[25,41]

As with all other vascular malformations, MR imaging and conventional angiography remain the primary mode of diagnostics. MR imaging may demonstrate T2-hyperintensity within the spinal cord caused by venous hypertension and edema. Intradural signal voids can often be seen, which represent dilated vascular structures (**Fig. 5**).[41] More recently, MDCT angiography has also been shown to be effective in correctly diagnosing the perimedullary AVF.[31] The gold standard of evaluation, however, is still DSA to best evaluate the anatomy and flow dynamics of the arterial feeders, fistulas, draining veins, and associated aneurysms.

Treatment is often advised for patients with perimedullary AVFs because untreated lesions may lead to progressive myelopathy and eventually paralysis.[41] Type I perimedullary AVFs are small and are bested treated with surgery.[25,41] For Type II lesions with multiple feeders, a combination of endovascular therapy and surgery should be used.[25,41] For the giant Type III lesions, it is recommended that these lesions be first treated endovascularly , and reserve surgery for patients who fail endovascular treatment.[25,41]

SPINAL CORD INFARCT

Spinal cord infarction is rare in comparison to infarction of brain tissue, comprising only 1% of all strokes.[42] It is a disease that typically affects older adults.[42–44] Most patients experience *pain* before the onset of neurologic symptoms.[42,43] Symptoms usually develop quickly, although some patients may experience a transient

Fig. 5. Perimedullary arteriovenous malformation. 13-year-old boy with abrupt onset of back pain and lower extremity weakness. Sagittal and axial fast spin echo T2 (*A, C*) and postgadolinium T1 fat saturated (*B, D*) sequences of the lumbar spine demonstrate focal hemorrhage within the conus (*asterisk*) with numerous prominent, dilated enhancing vessels (*arrows*).

ischemic attack before the actual spinal cord infarct.[42,43] There are 2 potential pathophysiological mechanisms for spinal cord infarction: (1) hypoperfusion from arterial insufficiency and hypotension, and (2) occlusion of a specific arterial branch (anterior spinal artery or, sometimes, posterior spinal artery).[42]

The most common risk factors include aortic disease and atherosclerosis.[45,46] Aortic dissections are associated with an overall 4.2% chance of having paraparesis or paraplegia as a result of spinal cord infarction.[47] Furthermore, aortic surgery also carries a significant risk of spinal cord ischemia, ranging from 1% in the upper and lower levels of the aorta to 10% in the midsection of the aorta, corresponding to the watershed area of the spinal cord.[48] Others have proposed a mechanical cause, as disk protrusions, foraminal fibrosis, and other degenerative spinal disease may put mechanical stress on a radicular artery.[42] It is unclear whether degenerative spinal conditions, such as disk protrusions, can directly cause radicular artery thrombosis/occlusion, because the prevalence of degenerative spine disease is high; whereas, spinal cord infarction is so rare. Degenerative spinal changes may potentially predispose patients to a spinal infarct, but other factors likely also contribute, such as a traumatic event.

Spinal cord infarcts occur at about equal frequency in the cervical and thoracolumbar spine, but they are extremely rare in the upper thoracic spine.[42,43] The most common pattern involves the anterior spinal artery (unilateral or bilateral). Other infarct patterns include the posterior spinal artery territory, a central cord infarct that affects bilateral spinothalamic tracts without any effect on the motor tracts, and a transverse infarct that results in a complete infarct of the level, resulting in bilateral motor function deficits and complete sensory deficits.[42,43] MR imaging is the imaging tool of choice for evaluation of patients with suspected spinal cord infarct. On sagittal T2-weighted images, a pencil-like hyperintensity can be seen in most patients.[44] Cord enlargement caused by swelling may also be seen.[44] In patients with an anterior spinal artery infarct, the owl's sign (T2 signal hyperintensity within anterior horns bilaterally) may be identified (**Fig. 6**).[49] Another imaging finding pointing to a cord infarct as the cause for an intramedullary signal abnormality is the presence of an adjacent *vertebral body* infarct.[42,43] Recently, diffusion weighted imaging has been used to evaluate spinal cord infarction.[44] Similar to ischemic infarction of the brain, acute spinal cord infarction also shows restricted diffusion with marked hyperintensity on the DWI sequence and a reduction in the apparent diffusion coefficient.[44] Because DWI changes develop before T2 signal changes, the addition of a diffusion sequence may expedite the diagnosis of spinal cord infarction (**Fig. 7**).[44] In some cases, DSA is
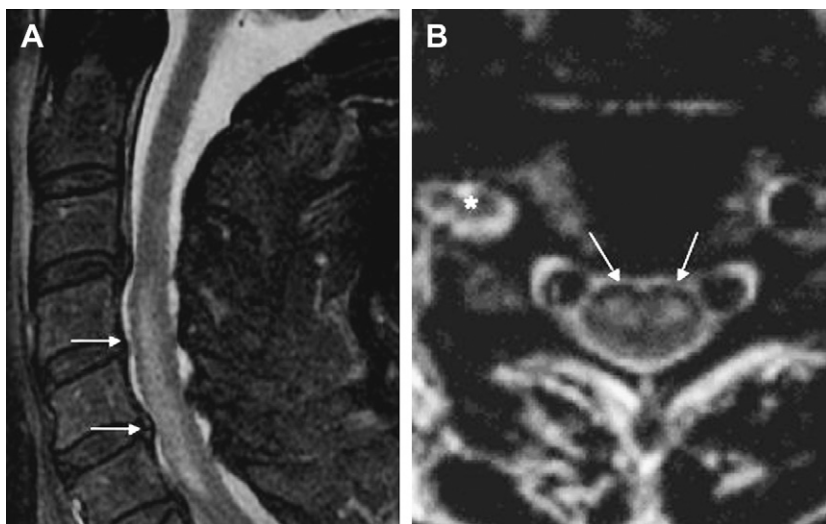

Fig. 6. Cervical spinal cord infarct and right vertebral artery dissection. 61-year-old woman with right shoulder weakness progressing to bilateral shoulder and upper extremity weakness. Sagittal (*A*) and axial (*B*) fast spin echo T2 sequences demonstrate mild cord expansion with increased signal within the bilateral anterior horns involving the anterior spinal artery territory (*arrows*). There is absence of the normal right vertebral artery flow void (*asterisk*).
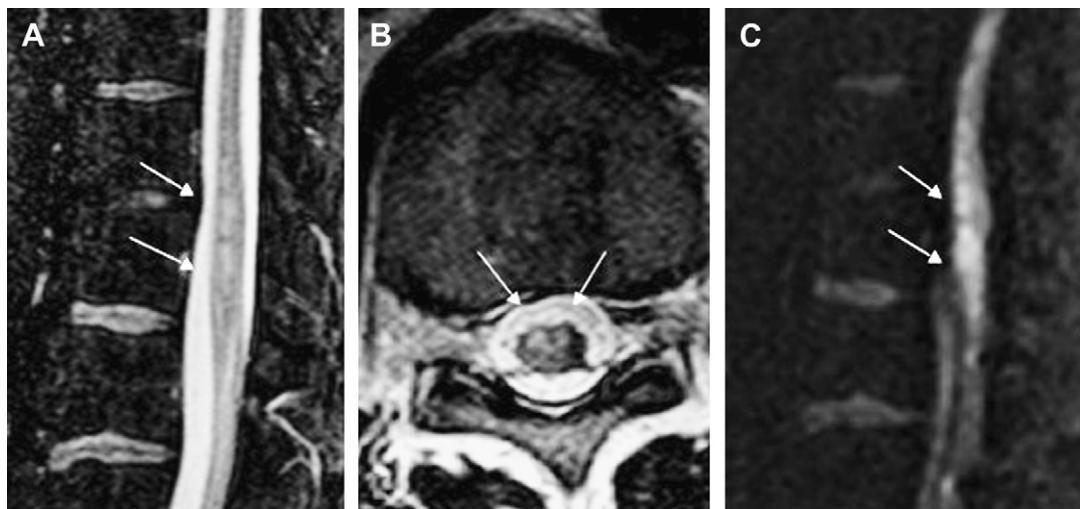
Fig. 7. **Spinal cord infarct.** 49-year-old man with acute back pain, nausea, and lower extremity weakness. Sagittal (*A*) and axial (*B*) fast spin echo T2 images through the thoracic cord demonstrate increased signal within the bilateral anterior horns involving the anterior spinal artery territory. (*C*) Sagittal diffusion-weighted imaging demonstrates reduced diffusion compatible with ischemia (*arrows*).

used to evaluate for specific arterial occlusions. In addition, DSA can be useful for ruling out treatable conditions, such as the DAVF, which also frequently shows abnormal intramedullary T2 hyperintensity on MR imaging.

The treatment of patients with spinal cord infarction remains supportive. Thrombolytics have not been used. Some patients may receive antiplatelet therapy, such as aspirin.[42] Most patients will have some recovery with supportive care and physical therapy. However, sphincter functions do not recover as well as motor functions.[42,43]

SUMMARY

Like the brain, the spinal cord is composed of neuronal and glial tissue that is vulnerable to trauma, infection, inflammation, ischemia, hemorrhage, and compression. This wide variety of conditions can damage spinal cord tissue and result in permanent paralysis or paraplegia. Certain conditions, such as the epidural abscess, can be treated, and therefore, prompt diagnosis is important. Advances in neuroimaging, especially with MR imaging, allow for improved evaluation of the spine and spinal cord. Noninvasive imaging, including MDCT angiography and MRA, are also likely to become more important in the evaluation of spinal vascular lesions. The development of new neuroradiological techniques and technology will continue to improve the diagnosis and treatment options for patients with spinal cord pathology.

REFERENCES

1. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 2000;23:175—204 [discussion: 205].
2. Darouiche RO. Spinal epidural abscess. N Engl J Med 2006;355:2012—20.
3. Chen WC, Wang JL, Wang JT, et al. Spinal epidural abscess due to staphylococcus aureus: clinical manifestations and outcomes. J Microbiol Immunol Infect 2008;41:215—21.
4. Chuo CY, Fu YC, Lu YM, et al. Spinal infection in intravenous drug abusers. J Spinal Disord Tech 2007;20:324—8.
5. Pereira CE, Lynch JC. Spinal epidural abscess: an analysis of 24 cases. Surg Neurol 2005;1(Suppl 63):S26—9.
6. Chen WH, Jiang LS, Dai LY. Surgical treatment of pyogenic vertebral osteomyelitis with spinal instrumentation. Eur Spine J 2007;16:1307—16.
7. Alg VS, Demetriades AK, Naik S, et al. Isolated subacute tuberculous spinal epidural abscess of the cervical spine: a brief report of a special case. Acta Neurochir (Wien) 2009;151:695—6.
8. Curry WT Jr, Hoh BL, Amin-Hanjani S, et al. Spinal epidural abscess: clinical presentation, management, and outcome. Surg Neurol 2005;63:364—71 [discussion: 371].
9. Gonzalez-Lopez JJ, Gorgolas M, Muniz J, et al. Spontaneous epidural abscess: analysis of 15 cases with emphasis on diagnostic and prognostic factors. Eur J Intern Med 2009;20:514—7.
10. Gorchynski J, Hwang J, McLaughlin T. A methicillin-resistant Staphylococcus aureus-positive holospinal

epidural abscess. Am J Emerg Med 2009;27(514): e517—9.

11. Lohr M, Reithmeier T, Ernestus RI, et al. Spinal epidural abscess: prognostic factors and comparison of different surgical treatment strategies. Acta Neurochir (Wien) 2005;147:159—66 [discussion: 166].

12. Parkinson JF, Sekhon LH. Spinal epidural abscess: appearance on magnetic resonance imaging as a guide to surgical management. Report of five cases. Neurosurg Focus 2004;17:E12.

13. Eastwood JD, Vollmer RT, Provenzale JM. Diffusion-weighted imaging in a patient with vertebral and epidural abscesses. AJNR Am J Neuroradiol 2002; 23:496—8.

14. Hlavin ML, Kaminski HJ, Ross JS, et al. Spinal epidural abscess: a ten-year perspective. Neurosurgery 1990;27:177—84.

15. Sokolowski MJ, Garvey TA, Perl J 2nd, et al. Prospective study of postoperative lumbar epidural hematoma: incidence and risk factors. Spine (Phila Pa) 1976;2008(33):108—13.

16. Kunizawa A, Fujioka M, Suzuki S, et al. Spontaneous spinal epidural hematoma inducing acute anterior spinal cord syndrome. J Neurosurg Spine 2009;10: 574—7.

17. Heiner JD. Cervical epidural hematoma after chiropractic spinal manipulation. Am J Emerg Med 2009;27(1023):e1021—2.

18. Xu R, Bydon M, Gokaslan ZL, et al. Epidural steroid injection resulting in epidural hematoma in a patient despite strict adherence to anticoagulation guidelines. J Neurosurg Spine 2009;11:358—64.

19. Horlocker TT, Wedel DJ, Benzon H, et al. Regional anesthesia in the anticoagulated patient: defining the risks (the second ASRA Consensus Conference on Neuraxial Anesthesia and Anticoagulation). Reg Anesth Pain Med 2003;28:172—97.

20. Liao CC, Hsieh PC, Lin TK, et al. Surgical treatment of spontaneous spinal epidural hematoma: a 5-year experience. J Neurosurg Spine 2009;11:480—6.

21. Oldfield EH, Doppman JL. Spinal arteriovenous malformations. Clin Neurosurg 1988;34:161—83.

22. da Costa L, Dehdashti AR, terBrugge KG. Spinal cord vascular shunts: spinal cord vascular malformations and dural arteriovenous fistulas. Neurosurg Focus 2009;26:E6.

23. Strom RG, Derdeyn CP, Moran CJ, et al. Frequency of spinal arteriovenous malformations in patients with unexplained myelopathy. Neurology 2006;66:928—31.

24. Clarke MJ, Patrick TA, White JB, et al. Spinal extradural arteriovenous malformations with parenchymal drainage: venous drainage variability and implications in clinical manifestations. Neurosurg Focus 2009;26:E5.

25. Medel R, Crowley RW, Dumont AS. Endovascular management of spinal vascular malformations:

history and literature review. Neurosurg Focus 2009;26:E7.

26. Rosenblum B, Oldfield EH, Doppman JL, et al. Spinal arteriovenous malformations: a comparison of dural arteriovenous fistulas and intradural AVM's in 81 patients. J Neurosurg 1987;67:795—802.

27. Benhaiem N, Poirier J, Hurth M. Arteriovenous fistulae of the meninges draining into the spinal veins. A histological study of 28 cases. Acta Neuropathol 1983;62:103—11.

28. Andres RH, Barth A, Guzman R, et al. Endovascular and surgical treatment of spinal dural arteriovenous fistulas. Neuroradiology 2008;50:869—76.

29. Eddleman CS, Jeong H, Cashen TA, et al. Advanced noninvasive imaging of spinal vascular malformations. Neurosurg Focus 2009;26:E9.

30. Ali S, Cashen TA, Carroll TJ, et al. Time-resolved spinal MR angiography: initial clinical experience in the evaluation of spinal arteriovenous shunts. AJNR Am J Neuroradiol 2007;28:1806—10.

31. Si-jia G, Meng-wei Z, Xi-ping L, et al. The clinical application studies of CT spinal angiography with 64-detector row spiral CT in diagnosing spinal vascular malformations. Eur J Radiol 2009;71: 22—8.

32. Lai PH, Pan HB, Yang CF, et al. Multi-detector row computed tomography angiography in diagnosing spinal dural arteriovenous fistula: initial experience. Stroke 2005;36:1562—4.

33. Yamaguchi S, Eguchi K, Kiura Y, et al. Multi-detector-row CT angiography as a preoperative evaluation for spinal arteriovenous fistulae. Neurosurg Rev 2007;30:321—6 [discussion: 327].

34. Yamaguchi S, Nagayama T, Eguchi K, et al. Accuracy and pitfalls of multidetector-row computed tomography in detecting spinal dural arteriovenous fistulas. J Neurosurg Spine 2010;12:243—8.

35. Luetmer PH, Lane JI, Gilbertson JR, et al. Preangiographic evaluation of spinal dural arteriovenous fistulas with elliptic centric contrast-enhanced MR angiography and effect on radiation dose and volume of iodinated contrast material. AJNR Am J Neuroradiol 2005;26:711—8.

36. Sharma AK, Westesson PL. Preoperative evaluation of spinal vascular malformation by MR angiography: how reliable is the technique: case report and review of literature. Clin Neurol Neurosurg 2008;110:521—4.

37. Steinmetz MP, Chow MM, Krishnaney AA, et al. Outcome after the treatment of spinal dural arteriovenous fistulae: a contemporary single-institution series and meta-analysis. Neurosurgery 2004;55: 77—87 [discussion: 87—8].

38. Eskandar EN, Borges LF, Budzik RF Jr, et al. Spinal dural arteriovenous fistulas: experience with endovascular and surgical therapy. J Neurosurg 2002; 96:162—7.

39. Du J, Ling F, Chen M, et al. Clinical characteristic of spinal vascular malformation in pediatric patients. Childs Nerv Syst 2009;25:473–8.

40. Spetzler RF, Detwiler PW, Riina HA, et al. Modified classification of spinal cord vascular lesions. J Neurosurg 2002;96:145–56.

41. Halbach VV, Higashida RT, Dowd CF, et al. Treatment of giant intradural (perimedullary) arteriovenous fistulas. Neurosurgery 1993;33:972–9 [discussion: 979–80].

42. Novy J, Carruzzo A, Maeder P, et al. Spinal cord ischemia: clinical and imaging patterns, pathogenesis, and outcomes in 27 patients. Arch Neurol 2006;63:1113–20.

43. Cheng MY, Lyu RK, Chang YJ, et al. Spinal cord infarction in Chinese patients. Clinical features, risk factors, imaging and prognosis. Cerebrovasc Dis 2008;26:502–8.

44. Thurnher MM, Bammer R. Diffusion-weighted MR imaging (DWI) in spinal cord ischemia. Neuroradiology 2006;48:795–801.

45. Cheshire WP, Santos CC, Massey EW, et al. Spinal cord infarction: etiology and outcome. Neurology 1996;47:321–30.

46. Nedeltchev K, Loher TJ, Stepper F, et al. Long-term outcome of acute spinal cord ischemia syndrome. Stroke 2004;35:560–5.

47. Zull DN, Cydulka R. Acute paraplegia: a presenting manifestation of aortic dissection. Am J Med 1988;84:765–70.

48. Connolly JE. Hume memorial lecture. Prevention of spinal cord complications in aortic surgery. Am J Surg 1998;176:92–101.

49. Mawad ME, Rivera V, Crawford S, et al. Spinal cord ischemia after resection of thoracoabdominal aortic aneurysms: MR findings in 24 patients. AJR Am J Roentgenol 1990;155:1303–7.