REVIEW ARTICLE

Vascular Myelopathies

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ABSTRACT

PURPOSE OF REVIEW: Neurologists should be able to identify clinical and neuroimaging features that distinguish vascular disorders from other causes of myelopathy.

RECENT FINDINGS: Although certain clinical features suggest a vascular etiology in acute and chronic myelopathy settings, accurate MRI interpretation within the clinical context is key. Recent studies have shown vascular myelopathies are frequently misdiagnosed as transverse myelitis, and recognition of this diagnostic pitfall is important. Many different vascular mechanisms can cause myelopathy; this article provides a comprehensive review that simplifies disease categories into arterial ischemia, venous congestion/ischemia, hematomyelia, and extraparenchymal hemorrhage.

SUMMARY: It is important to recognize and manage vascular disorders of the spinal cord as significant causes of acute, subacute, and progressive myelopathy.

INTRODUCTION

Two large retrospective studies recently showed that patients initially diagnosed with idiopathic transverse myelitis frequently had alternative myelopathy diagnoses, with vascular etiologies among the most common.[1,2] Vascular disorders of the spinal cord have important time-to-treatment considerations as delays in diagnosis can be associated with worse outcomes,[3] highlighting the importance of considering vascular causes early. Physicians should also be aware that treatments used for inflammatory disorders can potentially worsen symptoms and outcomes in vascular myelopathies.[4–7] Although certain features strongly suggest a vascular myelopathy in acute (severe deficits developing within ≤12 hours with pain) and chronic (exertional/Valsalva worsening) settings, accurate MRI interpretation within the clinical context is most critical. Many different vascular mechanisms exist; to simplify organization, this article divides vascular myelopathies into the categories of arterial ischemia, venous congestion/ischemia, hematomyelia, and extraparenchymal hemorrhage.

Myelopathy should be presumed as acute and a potential neurologic emergency at initial presentation until proven otherwise. Practically speaking, if a patient presents with rapid accumulation of deficits within approximately 24 hours or the acuity is indeterminate, a time-sensitive five-step, five-category approach is recommended (TABLE 2-1). This approach provides an important framework for compressive and noncompressive diagnoses and management considerations in the acute setting.

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RELATIONSHIP DISCLOSURE:
Dr Zalewski reports no disclosure.

UNLABELED USE OF PRODUCTS/INVESTIGATIONAL USE DISCLOSURE:
Dr Zalewski discusses the unlabeled/investigational use of IV alteplase for the treatment of spinal cord infarction.

VASCULAR ANATOMY OF THE SPINAL CORD

An understanding of the basic framework of spinal cord vascular anatomy helps provide important insights into clinical features and mechanisms. The vascular anatomy of the spinal cord consists of a single anterior spinal artery and paired posterior spinal arteries that run along the length of the spinal cord. The anterior spinal artery typically forms from two branches of intracranial V4 segments of the vertebral arteries (or posterior inferior cerebellar arteries [PICAs] or cervical segmental branches), whereas the paired posterior spinal arteries (often forming a plexus) originate from PICAs or the proximal vertebral arteries. The anterior spinal artery is often noncontinuous in the midthoracic region, referred to as the watershed area.[8]

Conceptualizing the pathway of extrinsic vasculature to reinforce the anterior spinal artery and posterior spinal arteries provides insight into localization of pathology that could contribute to ischemia (FIGURE 2-1[9]). This can be visualized as starting at the central venous return of the caval system, to the right heart, to the pulmonary vasculature, to the left heart, to the aortic arch, to the multiple reinforcing artery branches. These reinforcing artery branches are (rostral to caudal): the vertebral arteries (and PICA branches), subclavian arteries (with ascending cervical and deep cervical artery branches), posterior intercostal arteries, lumbar arteries, and iliac arteries (with lateral sacral and hypogastric artery branches). These arteries feed into spinal branches (entering the neural

Approach to Acute Myelopathy (Rapid Deficits, Approximately 24 Hours) TABLE 2-1

Airway, breathing, and circulation (and optimize)

Trauma?

- **Trauma protocol (eg, cervical collar), emergent spine surgery consult**

Aortic dissection?

- **CT angiography (eg, risk factors, chest/back pain, vital sign/pulse changes)**

Emergent MRI spine, with diffusion-weighted imaging of cervical, thoracic, and lumbar levels, unless clear localization (eg, quadriplegia). Assess for:

- **Cord compression**
  - Structural/spondylosis (rarely acute, unless severe compression with or without new trauma or fall)
  - Tumor/mass
  - Hematoma
  - Infection (epidural abscess/space-occupying infection)
- **Blood**
- **Flow voids**
- **Cord lesion (noncompressive and no blood or flow voids)**
- **Normal/equivocal**

Stroke laboratory studies, with or without head CT (especially consider if MRI spine within normal limits and no sensory level or pain)

CT = computed tomography; MRI = magnetic resonance imaging.

foramina) that divide into paired anterior and posterior radicular arteries at each spinal segmental level (FIGURE 2-2). However, only a portion of radicular arteries provide significant contributions to the spinal cord, termed anterior and posterior radiculomedullary arteries, whereas the rest terminate within the nerve root, dura, or pial plexus. Anterior radiculomedullary arteries provide significant reinforcement to the anterior spinal artery, whereas posterior radiculomedullary arteries supply the posterior spinal arteries. Wide variability exists in the number of radiculomedullary arteries, with anywhere from two to 17 unpaired arteries.[8,10,11] A main cervical radiculomedullary artery is usually present at C3 and another at C6-C7.[8] The thoracic cord is supplied by deep cervical and intercostal arteries. The great anterior radiculomedullary artery (artery of Adamkiewicz) is most often located between T9 and T12 (range T5 through L3); some patients may even lack this artery. Great posterior radiculomedullary arteries have been demonstrated as well.[12] Notably, the anterior spinal artery can facilitate anterograde or retrograde flow, depending on demand and variable anatomy. The spinal cord also receives extrinsic vascular reinforcement from adjacent paraspinal muscles and paravertebral tissue, creating a longitudinally connected flexible system relatively resistant to ischemia.[10,13] This is supported in everyday practice when vascular surgeons occlude the aorta's segmental branches with large grafts, yet patients most often do not experience spinal cord ischemia.

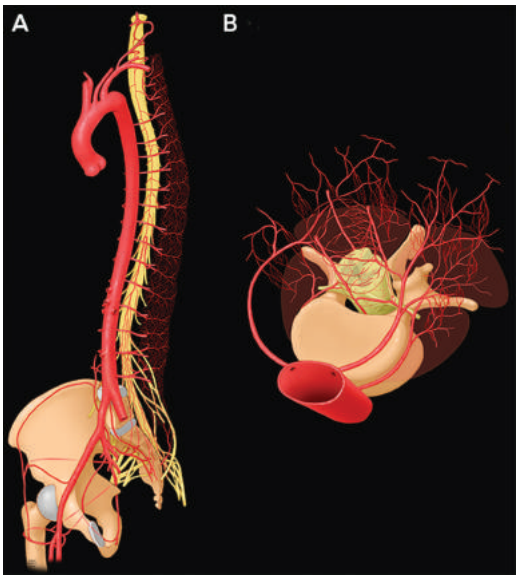

FIGURE 2-1
Extensive and flexible collateral vascular supply to the spinal cord. *A*, Longitudinal view of segmental arteries branching off the aorta with supplemental perfusion to the spinal cord. *B*, Anastomotic network depicted showing vascular supply of the spinal cord, paraspinal muscles, and paravertebral tissue creating a longitudinally connected flexible system.
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Intrinsically, the anterior spinal artery supplies the anterior two-thirds of the spinal cord through deep penetrating branches of central sulcal arteries, running deep to superficial.[14] Smaller posterior spinal arteries supply intrinsic vasculature to the posterior one-third of the spinal cord and are a predominant contribution to the vasocorona surrounding the cord, with superficial radial penetrating arteries supplying superficial to deep.[14] At the tip of the conus, an anastomotic loop exists between the anterior spinal artery and posterior spinal arteries.[15,16] Proximal nerve roots also receive vascular supply from branches of the anterior spinal artery and radiculomedullary arteries,[17] highlighting root involvement in many patients with spinal cord infarction.[7]

The venous system drains the spinal cord through radially oriented intramedullary veins that connect to a pial venous network on the surface

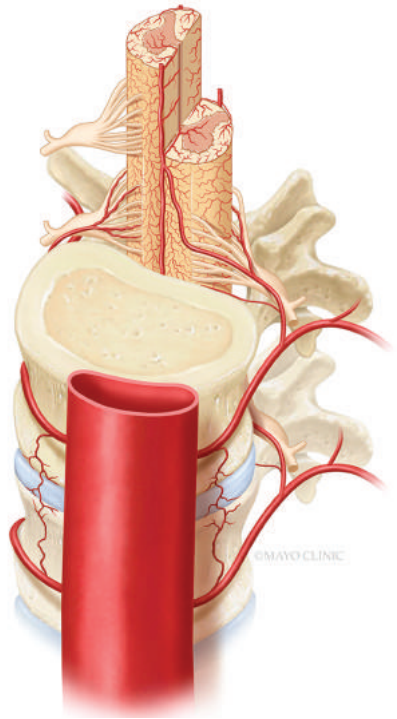

FIGURE 2-2
Representative view of the extrinsic vascular supply to a vertebral level of the spinal cord. The aorta is shown supplying posterior intercostal arteries, traversing closely to vertebral bodies, with supply of a spinal branch in the neural foramen dividing into anterior and posterior radiculomedullary artery branches supplying reinforcement to the anterior and posterior spinal arteries.

of the cord into both anterior and posterior spinal veins and subsequently to the internal venous plexus of the cord. This venous system located within the subarachnoid space joins epidural veins (internal vertebral plexus) via a radiculomedullary vein in neural foramina, which then subsequently exits to the external vertebral plexus. The venous system then converges through ascending lumbar (lumbar region), azygos (thoracic region), and innominate (cervical region) veins before returning to the caval system.[18]

ARTERIAL ISCHEMIA

Arterial ischemia affecting the spinal cord is classified as periprocedural spinal cord infarction, spontaneous spinal cord infarction, or spinal cord transient ischemic attack (TIA).

Periprocedural Spinal Cord Infarction

Periprocedural spinal cord infarction was first described by Sir Astley Cooper in 1825 in a patient with a large traumatic aneurysm of the iliac artery extending into the lower aorta. Since then, many different operations and medical procedures have been associated with spinal cord infarction.[19] Open or endovascular thoracic aortic aneurysm repair is the most common procedure associated with spinal cord infarction, representing approximately 50% of cases of periprocedural spinal cord infarction.[19] Spinal cord infarction has also been associated with other aortic operations (15%) and an array of other procedures (eg, cardiac surgery, spinal decompression, epidural injection, angiography, nerve block, embolization, other vascular surgery, and thoracic surgery).[19]

The majority of patients (80%) with periprocedural spinal cord infarction wake up from their procedure with maximum deficits, whereas others develop symptoms several hours or days after the procedure and may accumulate deficits over many hours. Although flaccid paraplegia is common, a number of patients (>30%) have less severe deficits. Pain is much less frequent (15% of cases) than in spontaneous spinal cord infarction (approximately 60% to 70% of cases); this may signify an association with mechanisms of spinal cord infarction (eg, arterial dissection, fibrocartilaginous embolism), although analgesics and other factors could confound this. Emergent spinal cord imaging is important to rule out an alternative treatable myelopathy mechanism (eg, epidural hematoma). Also, typical imaging patterns can confirm the diagnosis of spinal cord infarction. Periprocedural spinal cord infarction has provided insights into the diversity of

KEY POINTS

- **Two large retrospective studies recently showed that patients initially diagnosed with idiopathic transverse myelitis frequently had alternative myelopathy diagnoses, with vascular etiologies among the most common.**
- **Vascular disorders of the spinal cord have important time-to-treatment considerations as delays in diagnosis can be associated with worse outcomes, highlighting the importance of considering vascular causes early.**
- **The vascular anatomy of the spinal cord consists of a single anterior spinal artery and paired posterior spinal arteries that run along the length of the spinal cord.**
- **Open or endovascular thoracic aortic aneurysm repair is the most common procedure associated with spinal cord infarction, representing approximately 50% of periprocedural spinal cord infarction cases. Spinal cord infarction has also been associated with other aortic surgeries and an array of other procedures (eg, cardiac surgery, spinal decompression, epidural injection, angiography, nerve block, embolization, other vascular surgery, and thoracic surgery).**

CASE 2-1

A 66-year-old man was lifting heavy equipment when he acutely developed severe neck pain and a sense of general weakness. The following day, he suddenly developed worsening leg weakness and urinary retention. He took a brief nap and awoke developing rapid paraplegia within minutes. He was taken to the emergency department, nearly quadriplegic and in respiratory distress. He was intubated and transferred for further care.

Examination demonstrated skew deviation, severe asymmetric quadriparesis in proximal and distal muscles, and a truncal sensory level to temperature and pain on the left at C4 and the right at T4. Dorsal column function was preserved. Resting tone was flaccid with areflexia. Emergent MRI revealed T2-hyperintense signal in the upper cervical spinal cord, predominantly in the ventral gray matter (FIGURE 2-3A). Characteristic owl eyes were observed on axial cuts, typical of spinal cord infarction (FIGURE 2-3B). CT angiography of the head and neck identified a left vertebral artery dissection as the etiology. Brain MRI confirmed multifocal strokes in the cerebellum contributing to skew deviation (FIGURE 2-3C). The patient was treated with mean arterial blood pressure augmentation and a lumbar drain for 48 hours, then placed on aspirin for long-term treatment. Follow-up several months later revealed subtle residual motor and sensory deficits.

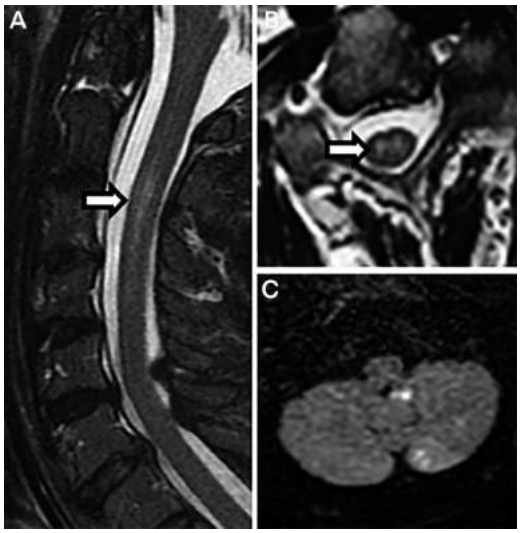

FIGURE 2-3
Imaging of the patient in CASE 2-1. *A*, Sagittal T2-weighted MRI shows T2-hyperintense signal in the upper cervical spinal cord, predominantly in the ventral gray matter (*arrow*). *B*, Axial T2-weighted MRI shows characteristic owl eyes (*arrow*), typical of spinal cord infarction. *C*, Axial brain diffusion-weighted MRI confirms multifocal strokes in the cerebellum contributing to skew deviation.

COMMENT

This case highlights several points. Despite the patient's reported earlier symptoms of pain and generalized weakness, the sudden change with rapid severe myelopathy was an essential clue to suspect spinal cord infarction; examination suggesting deficits in the anterior spinal artery territory raised additional suspicion for spinal cord infarction. Typical imaging findings were seen on T2-weighted imaging, with owl eyes and severe deficits out of proportion to a smaller lesion, and specific findings confirmed spinal cord infarction (dissection, concurrent cerebral stroke). Treatment was pursued, and the patient had an excellent outcome.

clinical and imaging findings that can be seen in spontaneous spinal cord infarction, in which diagnosis can be more challenging.

During spinal cord ischemia, the goal of treatment is to increase spinal cord perfusion pressure through collaterals. This is accomplished by lowering pressure within the spinal canal via CSF drainage (eg, drain 10 mL every 2 to 4 hours; goal CSF pressure 8 mm Hg to 12 mm Hg, depending on response) or mean arterial blood pressure (MAP) augmentation (eg, goal MAP >90 mm Hg or increase by 10 mm Hg to 20 mm Hg increments, observing for benefit), or both. Potential benefit needs to be weighed against risks (eg, overdrainage). Although two randomized clinical trials and multiple meta-analyses have shown benefit using prophylactic lumbar drains,[20] data are limited regarding treatment benefit once ischemia has occurred. When ischemia occurs, early poor prognostication is generally not recommended, as many patients have substantial improvements despite significant deficits.[19,21] Factors associated with poor outcomes include a larger lesion on MRI, clinical severity,[22] flaccid areflexia, absence of a Babinski reflex,[21] age, and comorbid medical factors.

Spontaneous Spinal Cord Infarction

The incidence of spinal cord infarction has recently been estimated at 3.1 per 100,000 people; in this small study, six were spontaneous and two periprocedural.[23] Other studies have published similarly low estimates, representing approximately 1% of strokes and 5% to 8% of cases of acute myelopathy.[16,22] However, two large studies highlighted the frequent misdiagnosis of spinal cord infarction as "transverse myelitis" in approximately 15% of referred cases.[2,7] The recent introduction of diagnostic criteria may further increase our understanding of the true incidence. Although an older patient population with vascular risk factors is common, mechanisms affecting younger patients also occur (eg, fibrocartilaginous embolism, vertebral dissection), highlighting that spinal cord infarction can occur at any age.

CLINICAL FEATURES. From the earliest days, it has been recognized that spinal cord infarction frequently results in acute deficits localized to the anterior spinal artery territory (bilateral corticospinal tract, lower motor neuron at lesion level, and pain/temperature loss) or, less frequently, the posterior spinal artery territory (dorsal column dysfunction); these deficits may distinguish spinal cord infarction from other myelopathies. Over time, other clinical syndromes, such as Brown-Séquard syndrome, central cord syndrome, and complete cord syndrome, were also described.[24] Although identifying a classic vascular territory is helpful, absence of a classic pattern is frequent and other premorbid conditions (eg, peripheral neuropathy) can confound.

Diagnosis has shifted to a focus on establishing severe nontraumatic myelopathy deficits within 12 hours. Although labeling deficits as severe can be challenging retrospectively, the timeline of severe paresis/plegia (usually bilateral) within hours is usually well delineated. In posterior spinal artery infarctions, this may manifest as a severe objective sensory deficit (eg, sensory ataxia). Approximately 25% of patients reach nadir after 12 hours but still display a component of severe deficits within a 12-hour time frame of their acute stepwise/stuttering decline. The faster and more severe the deficits, the easier to diagnose spinal cord infarction. Severe acute pain (back, chest, neck, limb) at or before onset is another helpful feature that is reported in approximately 70% of patients[7] but is atypical acutely in myelitis (CASE 2-1). Pain may be an insight

KEY POINTS

- During spinal cord ischemia, the goal of treatment is to increase spinal cord perfusion pressure through collaterals by lowering pressure within the spinal canal via CSF drainage or mean arterial blood pressure augmentation.
- Two large studies highlighted the frequent misdiagnosis of spinal cord infarction as "transverse myelitis" in approximately 15% of referred cases.
- Although an older patient population with vascular risk factors is common in spinal cord infarction, mechanisms affecting younger patients also occur (eg, fibrocartilaginous embolism, vertebral dissection), highlighting that spinal cord infarction can occur at any age.
- From the earliest days, it has been recognized that spinal cord infarction frequently results in acute deficits localized to an anterior spinal artery territory (bilateral corticospinal tract, lower motor neuron at lesion level, and pain/temperature loss) or, less frequently, a posterior spinal artery territory (dorsal column dysfunction); these deficits may distinguish spinal cord infarction from other myelopathies.
- Severe acute pain (back, chest, neck, limb) at or before onset is another helpful feature that is reported in approximately 70% of patients with spinal cord infarction but is atypical acutely in myelitis.

into the mechanism (eg, dissection, fibrocartilaginous embolism), given its higher frequency in spontaneous spinal cord infarction than periprocedural cases.[19] A truncal sensory level or localized pain, or both, is helpful in localization of spinal cord infarction; clinicians should consider alternative localizations if these features are absent (bilateral anterior cerebral artery territory, rapid peripheral disorder). Strictly unilateral presentations can occur but are rare and raise suspicion for cerebral localization (eg, contralateral anterior cerebral artery territory with leg weakness). A physical maneuver (eg, lifting, back hyperextension, neck movement, Valsalva) is frequently reported at or before onset. This serves as a clue to the diagnosis and potential mechanism (eg, arterial dissection, fibrocartilaginous embolism, radicular artery compression from disk, or other mechanical mechanism).

NEUROIMAGING. Once spontaneous spinal cord infarction is suspected, it is important to understand the typical MRI appearance in acute, subacute, and chronic settings.

ACUTE. Refer to **TABLE 2-1** for the emergent protocol for acute myelopathy. A low threshold for MRI of the entire spine should exist, unless the localization is clear (eg, cervical spinal cord in quadriplegia). Diffusion-weighted imaging (DWI)/apparent diffusion coefficient (ADC) should be performed, but the sensitivity is incomplete (50% to 70%),[7,19] and sometimes takes days to evolve.[25] In the initial hours of symptoms, imaging is likely normal or equivocal.[7] Early

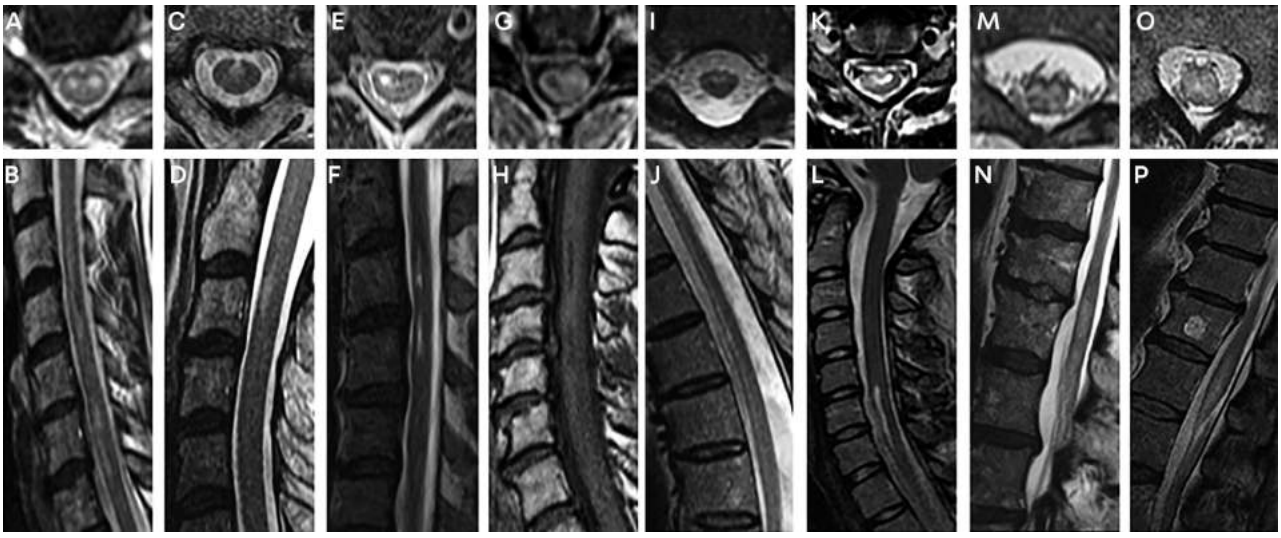

FIGURE 2-4

MRI findings in spinal cord infarctions. Typical patterns of T2-hyperintense signal seen in spinal cord infarction include owl eyes (*A*), associated with noncontiguous anterior pencillike hyperintensity (*B*); anteromedial spot (*C*), confirmed with short anterior pencillike hyperintensity on sagittal view (*D*); residual cystic myelomalacia with very bright T2 hyperintensity (*E, F*) and associated T1 hypointensity (*G, H*), seen more than 1 month after spinal cord infarction; anteromedial T2 hyperintensity (*I*) with associated anterior pencillike hyperintensity (*J*); anterior U or V shape (*K*) with associated anterior pencillike hyperintensity on sagittal view (*L*); hologray (entire gray matter) pattern (*M*) with associated edematous T2 hyperintensity on sagittal view (*N*); and holocord (entire spinal cord) pattern (*O*) with associated edematous T2 hyperintensity extending through the conus on sagittal view (*P*).
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T2-hyperintense signal typical of spinal cord infarction (FIGURE 2-4) may be seen. Significant edema/swelling or enhancement is unusual acutely. Adjacent dissection/occlusion or concurrent cerebral infarction may also be identified.

SUBACUTE. A variety of axial T2-hyperintensity patterns can be seen in the day(s) after spinal cord infarction (FIGURE 2-5). The lower thoracic cord into the conus frequently demonstrates larger cross-sectional lesion areas that do not respect classic vascular territories. Although identifying typical T2-hyperintense imaging patterns is helpful, the spectrum can be diverse and atypical. However, some T2-hyperintensity findings raise a red flag for an etiology other than spinal cord infarction, including a large lesion out of proportion to mild deficits, signal extending the entire length of the cord, separate well-defined lesions (although noncontiguous lesions are common), and a small lesion with severe edema. When present (in approximately 40% of cases), gadolinium enhancement in spinal cord infarction is usually a linear craniocaudal strip (FIGURE 2-6); resolution of enhancement is typically seen within months.[7] Adjacent nerve root enhancement (non-nodular) can be seen subacutely (FIGURE 2-6). Specific findings confirming spinal cord infarction include diffusion restriction, vertebral body infarction, and adjacent arterial dissection/occlusion. Well-defined DWI hyperintensity should be confirmed on ADC, especially given the technical difficulties inherent in DWI of the cord. At times, T2-hyperintense signal in the anterior spinal artery is seen, suggesting slow flow or thrombus,[19] but the specificity is unclear. It is important to correctly identify vertebral body infarctions; new well-defined posterior or anterior T2-hyperintense signal, often in a wedge or triangular shape on axial views, may enhance subacutely. However, misinterpreting vertebral body hemangiomas and degenerative changes could lead to misdiagnosis. Rib and muscle infarctions can also rarely be seen.[26,27] Dissections can be difficult to find; therefore, a dissection protocol (eg, cervical MRI with T1-weighted fat saturation) should be used. Dissections and other mechanical pathology affecting smaller feeding arteries may be responsible for some idiopathic cases. Magnetic resonance angiography (MRA) of the spinal canal is currently of limited value in spinal cord infarction, unless a rare acute presentation of spinal dural arteriovenous fistula/arteriovenous malformation (AVM) is suspected. Rarely, large artery[27] or long anterior spinal artery occlusions can be found. The cost and risk of digital subtraction angiography (DSA) likely outweigh its utility in assessing spinal cord infarction, unless strong concern for an alternative diagnosis (eg, spinal dural arteriovenous fistula) or a rare etiology (vasculitis) remains.

CHRONIC. Several weeks to months after a spinal cord infarction, differentiating imaging findings from other causes of myelopathy becomes more difficult. Although, at times, classic findings of myelomalacia (very bright focal T2 hyperintensity with T1 hypointensity and volume loss) (FIGURE 2-4),[7] cord atrophy, or residual T2 hyperintensity respecting a vascular territory can be seen, many cases are more difficult to distinguish.

ADDITIONAL TESTS. Many patients with spinal cord infarction are initially suspected to have Guillain-Barré syndrome. When nerve conduction studies/EMG and CSF evaluation are obtained, it is important to recognize frequent abnormalities attributable to spinal cord infarction. In one series, 13 of 19 patients with spinal cord infarction had EMG abnormalities corresponding to the area of infarction.[7,28]

KEY POINTS

- Once spontaneous spinal cord infarction is suspected, it is important to understand the typical MRI appearance in acute, subacute, and chronic settings.
- A low threshold for MRI of the entire spine should exist, unless the localization is clear (eg, cervical spinal cord in quadriplegia). Diffusion-weighted imaging/apparent diffusion coefficient should be performed, but the sensitivity is incomplete and sometimes takes days to evolve. In the initial hours of symptoms, imaging is likely normal or equivocal.

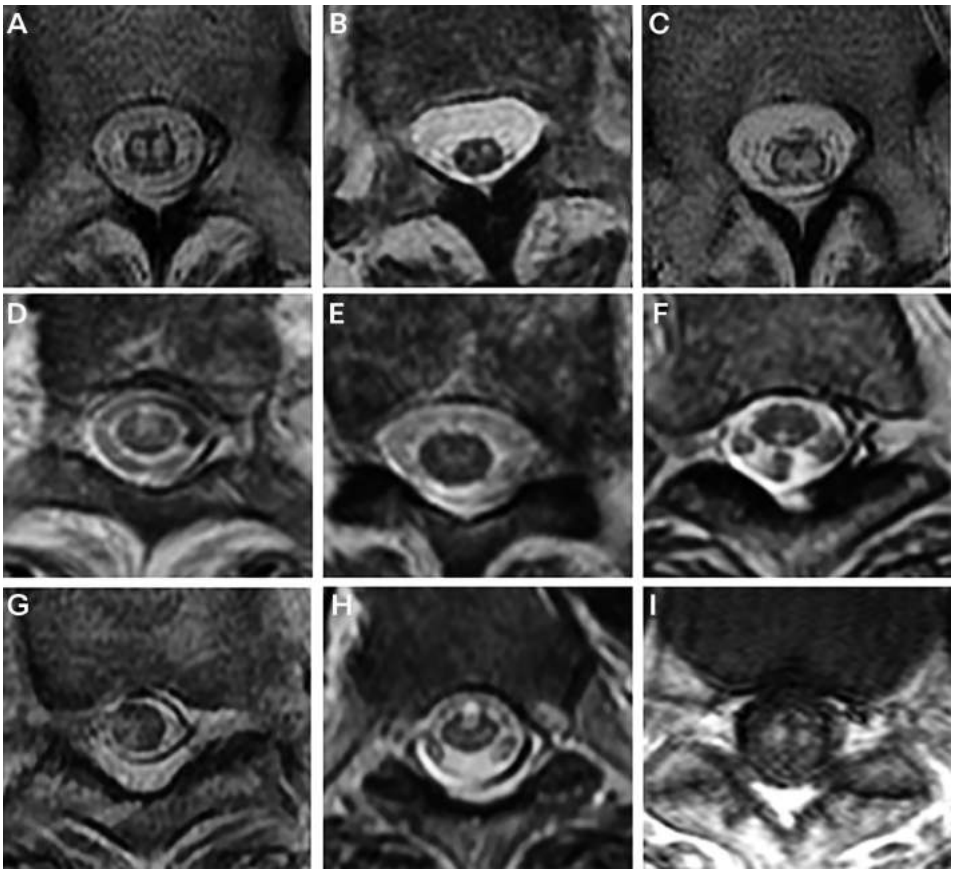

FIGURE 2-5

Axial MRI findings of spinal cord infarctions. A variety of axial T2-hyperintense MRI patterns can be seen in spinal cord infarctions, which are often incomplete, asymmetric, or noncontiguous, including owl eyes (large [*A*] or small [*B*]), hologray (entire gray matter) (*C*), anteromedial spot (*D*), and anterior U or V (*E*). Infarcts often involve territories other than the typical anterior two-thirds of the spinal cord, including holocord (entire spinal cord), dorsomedial (*F*), and lateral (*G*). A small focus of bright T2-hyperintense signal (*F*) accompanied by focal T1 hypointensity (*not shown*) represents residual cystic myelomalacia as a chronic finding after infarction. Anterior spinal artery T2 hyperintensity (*H*) (demonstrated ventral to an anteromedial spot) is observed in some cases, supporting the presence of a thrombus or slow flow contributing to ischemia. T1-weighted contrast enhancement highlights the predominant area of ischemia, which is often an owl-eye pattern (*I*), and in this example demonstrates associated ventral root enhancement.

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CSF often shows mild to moderate protein elevation, frequently confused for the albuminocytologic dissociation seen in Guillain-Barré syndrome. In some cases (<10%),[7] mild elevation in inflammatory markers is seen, raising caution for alternatives, but it is important to note that mild abnormalities can still be consistent with spinal cord infarction; significant elevations of greater than 25 cells/mm^3 would be unlikely.

DIAGNOSIS. Spinal cord infarction diagnostic criteria are outlined in TABLE 2-2.[7] Clinicians should err on the side of using a working diagnosis of "possible spinal cord infarction" acutely, as criterion 4 highlights a workup ensuring alternative diagnoses are not likely.

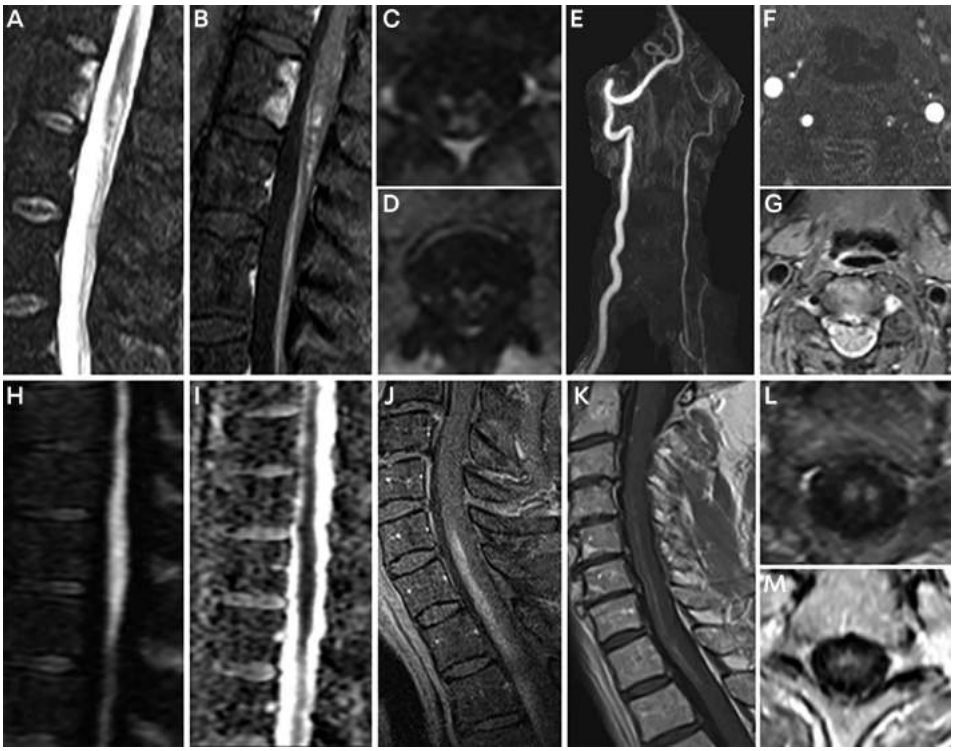

FIGURE 2-6

Confirmatory MRI findings and typical gadolinium enhancement pattern in spinal cord infarctions. Vertebral body infarction is seen on short tau inversion recovery (STIR) images (*A*) with associated gadolinium enhancement (*B*) of the vertebral body infarct, spinal cord infarction (*C*), and anterior cauda equina (*D*); T1-weighted fat-suppressed images show cervical artery dissection with significantly decreased left vertebral flow (*E*, *F*), with confirmed intramural hematoma (*G*) adjacent to spinal cord infarction. Diffusion-weighted image shows diffusion restriction (*H*) with correlation on apparent diffusion coefficient (*I*) image. Gadolinium enhancement of spinal cord infarction demonstrated with a typical craniocaudal linear strip on sagittal views (*J*, *K*) and corresponding anterior predominant gray matter (*L*) and anteromedial spot (*M*) patterns on axial views, highlighting the predominant areas of ischemia.

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KEY POINT

- **It is reasonable to discuss risks and benefits of IV recombinant tissue plasminogen activator within the first 4.5 hours after onset if suspicion of spinal cord infarction is high and the patient understands the limited evidence.**

TREATMENT. Data on treatment for spinal cord infarction are limited. It is reasonable to discuss risks and benefits of IV recombinant tissue plasminogen activator (rtPA) within the first 4.5 hours after onset once the acute myelopathy algorithm (TABLE 2-1) has been followed and if suspicion of spinal cord infarction is high and the patient understands the limited evidence. At least 13 cases of the use of rtPA in spinal cord infarction had been published at the time this article was written,[7,29] many of which described benefit and no significant complications. If rtPA is not administered, MAP augmentation with or without lumbar drainage can be considered, as discussed in the periprocedural spinal cord infarction section.

Secondary stroke prevention should focus on the suspected mechanism (TABLE 2-3). In many cases, the mechanism is not clear, and the focus is on rehabilitation, reducing vascular risk factors, and maintaining a healthy lifestyle. Most patients receive antiplatelet therapy, but no evidence has established its use unless supported by a specific mechanism. In some cases with atherosclerotic risk factors and stuttering decline, dual-antiplatelet therapy and a high-intensity statin could be considered.[30]

MECHANISM. Atherosclerosis, arterial dissection, and fibrocartilaginous embolism are the most common presumed mechanisms of spontaneous spinal cord infarction. Patients frequently have thorough evaluations with no clear mechanism identified. Given the frequent history of various activities occurring in the context in many cases, mechanical mechanisms should be closely considered (eg, fibrocartilaginous embolism, dissection, compression of radicular artery by a disk). Other more diverse mechanisms that are not currently understood may also exist (eg, arterial vasospasm). Some studies have demonstrated compressive intersegmental artery mechanisms suspected in spinal cord infarction and spinal TIAs (eg, diaphragmatic crus syndrome, endothoracic fascia compression, and disko-osteo-arterial conflict)[31]; symptoms related to such lesions could also be related to vasospasm or dissection.[31] Many additional mechanisms of spinal cord infarction have been seen (TABLE 2-3), and investigations can be individually tailored (TABLE 2-4).

VASCULITIS. Primary angiitis of the central nervous system (CNS), systemic autoimmune vasculitis (eg, antineutrophil cytoplasmic antibody [ANCA], giant cell arteritis), infectious vasculitis with or without meningitis

TABLE 2-2 Spinal Cord Infarction Diagnostic Criteria[a]

Criteria

1 Acute nontraumatic myelopathy (no preceding progressive myelopathy)

Onset to nadir of severe deficits[b] 12 hours or less

If stuttering course is more than 12 hours, severe deficits rapidly develop over 12 hours or less

2 MRI

A No spinal cord compression

B Supportive: intramedullary T2-hyperintense spinal cord lesion

C Specific (one of): diffusion-weighted imaging/apparent diffusion coefficient restriction, associated vertebral body infarction, arterial dissection/occlusion adjacent to lesion

3 CSF

Noninflammatory (normal cell count and IgG index and no oligoclonal bands)

4 Alternative diagnoses

Alternative diagnosis is not more likely

Type and diagnostic certainty of spinal cord infarction

- Definite spontaneous spinal cord infarction (requires all of the following: 1, 2A, 2B, 2C, 4)
- Probable spontaneous spinal cord infarction (requires all of the following: 1, 2A, 2B, 3, 4)
- Possible spontaneous spinal cord infarction (requires all of the following: 1, 4)
- Definite periprocedural spinal cord infarction (requires all of the following: 1, 2A, 2B, 4)
- Probable periprocedural spinal cord infarction (requires all of the following: 1, 4)

CSF = cerebrospinal fluid; IgG = immunoglobulin G; MRI = magnetic resonance imaging.
[a] Modified with permission from Zalewski NL, et al, JAMA Neurol.[7] © 2019 American Medical Association.
[b] A severe acute deficit (motor and/or sensory) typically consists of loss of antigravity strength or worsened severe objective sensory loss impairing function (eg, severe sensory ataxia).

(eg, varicella-zoster virus, syphilis, hepatitis), and paraneoplastic vasculitis (eg, Hodgkin lymphoma) can rarely affect spinal cord vasculature, with spinal cord infarction, subarachnoid hemorrhage, or hematomyelia. The spinal cord is usually affected as part of a more widespread presentation (with or without systemic features), leading to suspicion. A large series of primary angiitis of the CNS showed spinal cord involvement in 5% of cases; all had cerebral involvement, one initially presented with myelopathy, and all had significant CSF pleocytosis.[32] In a large series of spontaneous spinal cord infarction, 1.5% of cases were secondary to vasculitis.[7]

Etiologies of Spinal Cord Infarction[a] TABLE 2-3

Atherosclerosis

Dissection

- Aortic dissection
- Vertebral artery dissection
- Subclavian artery dissection

Mechanical

- Fibrocartilaginous embolism
- Surfer's myelopathy
- Arterial compression (disk, other)

Embolic

- Fibrocartilaginous embolism
- Aortic embolism (eg, atheroma, calcium)
- Cardioembolic (eg, atrial fibrillation, endocarditis, myxoma, fat, paradoxical)
- Fungal embolus

Vasculitis

- Primary angiitis of the central nervous system
- Systemic vasculitis (eg, giant cell arteritis)
- Infectious (eg, varicella-zoster virus, syphilis, fungal, schistosomiasis, Lyme disease, tuberculous meningitis, hepatitis)

Hypotension

Hypercoagulability

- Lupus anticoagulant/antiphospholipid antibody syndrome
- Malignancy
- Sickle cell disease, hereditary spherocytosis, polycythemia vera
- Disseminated intravascular coagulation

Pharmacologic

- Medications (sildenafil, oral contraceptives, phentermine, others)
- Drugs of abuse (eg, cocaine, cannabis)

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TABLE 2-4

Evaluation of a Spinal Cord Infarction[a]

Standard evaluation

- **Imaging**
  - MRI cervical/thoracic spine (with diffusion-weighted imaging/apparent diffusion coefficient and gadolinium, with or without brain MRI)
  - Magnetic resonance angiography (MRA) cervical (if cervical level affected, with T1 fat saturation for dissection)
- **Blood tests**
  - Hemoglobin A_{1c}, fasting lipids, glucose
  - Complete blood cell count, prothrombin time/activated partial thromboplastin time
- CSF (can consider deferring if clear diagnosis)
  - Cell count, glucose, protein, IgG index, oligoclonal bands

Additional testing to consider

- **Imaging**
  - MRI brain (evaluate for concurrent stroke, alternative lesions)
  - MRA spinal canal or angiogram (clinical/radiographic concern for spinal dural arteriovenous fistula/arteriovenous malformation, or vasculitis)
  - CT chest/abdomen/pelvis (malignancy concern)
- **Blood**
  - Aquaporin-4-IgG, myelin oligodendrocyte glycoprotein (MOG)-IgG
  - Syphilis and Lyme serology
  - Hypercoagulable profile
  - Erythrocyte sedimentation rate, C-reactive protein
  - Antinuclear antibody, extractable nuclear antigen, antineutrophil cytoplasmic antibody panel, dsDNA
  - Paraneoplastic autoantibody evaluation
  - Toxicology screen (with urine)
- **CSF**
  - Varicella-zoster virus polymerase chain reaction (PCR)
  - Venereal Disease Research Laboratory test
  - Lyme serology/PCR
  - *Cryptococcus* antigen
- **Cardiac evaluation**
  - Transesophageal echocardiogram
  - Holter monitoring
- **Other**
  - EMG (suspicion of Guillain-Barré syndrome)
  - Temporal artery biopsy (concern of giant cell arteritis)

CSF = cerebrospinal fluid; CT = computed tomography; dsDNA = double-stranded deoxyribonucleic acid; EMG = electromyography; IgG = immunoglobulin G; MRI = magnetic resonance imaging.

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LONG-TERM FOLLOW-UP AND PROGNOSIS. Outcomes after spontaneous spinal cord infarction are variable. Despite severe deficits, approximately 50% of patients ultimately ambulate without a gait aid.[7] Residual neurogenic bowel/bladder and neuropathic pain are common. Examination may demonstrate lower motor neuron dysfunction (eg, atrophy), depending on lesion location.[27] Long-term recurrence of spontaneous spinal cord infarction (outside of an initial stuttering presentation) is exceedingly rare.[7]

Spinal Cord Transient Ischemic Attack

Spinal cord TIAs are questioned at times in patients with spells affecting bilateral limbs. TIAs can be difficult to prove, even in cerebrovascular disease, with a number of mimickers. Working around limitations, a recent study found 4 of 133 patients (3%) who ultimately had a spontaneous spinal cord infarction and initially experienced spinal cord TIAs.[33] Traditional vascular risk factors were seen in 75% of patients; the patients developed spinal cord infarction in the same distribution as their spinal cord TIAs, and all had experienced multiple spinal cord TIAs. Symptoms lasted from seconds to minutes before return to baseline. No patients had pain as a feature. Spinal cord TIA is different than the stuttering decline frequently seen in spinal cord infarction (23% of cases), in which deficits fluctuate but accumulate. Given the rarity of spinal cord TIAs, physicians should carefully consider alternatives.

VENOUS ISCHEMIA

Venous ischemia of the spinal cord is predominantly caused by spinal dural arteriovenous fistula, but other mechanisms should also be considered.

Spinal Dural Arteriovenous Fistula

Spinal dural arteriovenous fistulas (Anson-Spetzler type I AVMs) comprise 70% of spinal AVMs. This arteriovenous shunt is composed of a connection between a radiculomeningeal artery and single radicular vein, typically in the neural foramen, leading to arterialization of the vein, venous hypertension, spinal cord congestion, and venous ischemia.[34,35] The incidence of spinal dural arteriovenous fistula is 5 to 10 cases per million per year.[36-40] An older population is typical (40 to 80 years), with male predominance (80%), and patients frequently report previous back surgeries or trauma that may contribute to the development of a fistula.[36,37] A number of cases of spinal dural arteriovenous fistula have been demonstrated less than 2 years after spine surgery (cervical, thoracic), in which clear evidence of a structural spine disease initially contributing and no preoperative evidence of spinal dural arteriovenous fistula were seen.[41]

CLINICAL FEATURES. Clinical presentation typically includes a gradually progressive thoracic myelopathy with leg weakness/numbness, bowel/bladder dysfunction, symptoms frequently referable to the conus/roots, and episodic worsening with exertion/Valsalva (CASE 2-2). Pain is frequent (back or radicular).[37] Acute paraplegia (in approximately 2% to 5% of cases) and stepwise deterioration (in 11% to 32% of cases) can occur.[22,24,36,37,42-44] A sensory level is reported in 18% to 37% of cases, most frequently at L1.[43,45] The strongest clinical clue is dramatic worsening of deficits with activities elevating venous pressure, such as exertion, Valsalva, or lumbar puncture.[46] Patients are

KEY POINTS

- Atherosclerosis, arterial dissection, and fibrocartilaginous embolism are the most common presumed mechanisms of spontaneous spinal cord infarction.
- Outcomes after spontaneous spinal cord infarction are variable. Despite severe deficits, approximately 50% of patients ultimately ambulate without a gait aid.
- The incidence of spinal dural arteriovenous fistula is 5 to 10 cases per million per year. An older population is typical (40 to 80 years), with male predominance (80%), and patients frequently report previous back surgeries or trauma that may contribute to the development of a fistula.
- The clinical presentation of spinal dural arteriovenous fistula typically includes a gradually progressive thoracic myelopathy with leg weakness/numbness, bowel/bladder dysfunction, symptoms frequently referable to the conus/roots, and episodic worsening with exertion/Valsalva. The strongest clinical clue is dramatic worsening of deficits with activities elevating venous pressure, such as exertion, Valsalva, or lumbar puncture.

frequently given corticosteroids for suspected alternatives, and 50% of patients can have significant worsening[4] (venous hypertension)[4,47]; this is a strong diagnostic clue. Inadvertent improvement with plasma exchange has also been described.[48] Features are often mistaken for peripheral localization (eg, cauda equina), relating to a frequent lack of upper motor neuron signs[36,44] with progressive lower motor neuron injury at the level of the conus/roots. At diagnosis, 70% of patients have both upper motor neuron and lower motor findings.[49] Cervical myelopathy presentations with similar radiographic and clinical features can be seen (2% of cases).[36,41,50] Rarely, spinal dural arteriovenous fistula can be an incidental finding.[51]

CASE 2-2

A 62-year-old man was referred for evaluation of "transverse myelitis." He reported 9 months of progressive weakness and sensory loss of the right more than left leg. Walking resulted in dramatic worsening of weakness. His past medical history included hypertension, hyperlipidemia, and lumbar spine surgery 5 years earlier.

Examination demonstrated moderate asymmetric right more than left leg weakness, with severe sensory loss to all modalities in the lower extremities and no truncal sensory level. Reflexes were reduced, and plantar responses were mute. Nerve conduction studies and EMG were performed, revealing a lumbosacral polyradiculopathy; however, MRI of the lumbar spine revealed T2-hyperintense signal extending into the conus, with further imaging demonstrating T2-hyperintense signal throughout the lower thoracic cord (FIGURE 2-7A). Multiple small T2-hypointense flow voids were seen surrounding the cord (FIGURES 2-7A and 2-7B), and T1-weighted imaging with gadolinium demonstrated abrupt missing pieces within a confluent area of enhancement (FIGURES 2-7C and 2-7D); these findings were suspicious for spinal dural arteriovenous fistula. Digital subtraction angiography demonstrated a fistula at the right T10 intercostal artery. Surgical obliteration was successful, and the patient had clinical stabilization.

COMMENT

This case highlights the importance of recognizing exertional or Valsalva-induced worsening of deficits and concurrent findings of lower motor neuron involvement in spinal dural arteriovenous fistula. Expansile T2-hyperintense signal into the conus with accompanying flow voids and the missing piece sign are characteristic features of spinal dural arteriovenous fistula. A diagnosis is confirmed with digital subtraction angiography, and treatment includes endovascular or surgical obliteration.

NEUROIMAGING. Spinal cord T2-hyperintense signal is present in approximately 95% of cases,[45] is often longitudinally extensive (three or more vertebral body segments), and frequently extends to the conus (90% of cases) (FIGURE 2-8). T2 hyperintensity can be faint, but a large lesion with edema is typical; atrophy can be seen in delayed diagnoses. Flow voids (engorged perimedullary veins) are seen on the dorsal more than the ventral surface of the cord in approximately 80% of cases. They are typically demonstrated on T2-weighted imaging but can also be seen better, at times, on T1-weighted imaging with gadolinium[45,52,53]; heavily T2-weighted myelographic sequences (eg, phase-cycled fast imaging employing steady state acquisition [PC-FIESTA], three-dimensional constructive

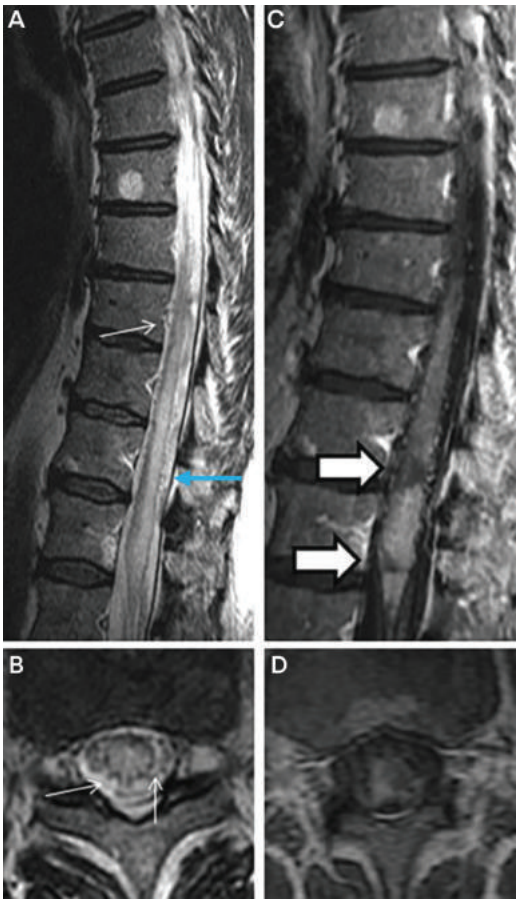

FIGURE 2-7
Imaging of the patient in CASE 2-2. Sagittal (*A*) and axial (*B*) T2-weighted MRIs show T2-hyperintense signal and edema throughout the lower thoracic cord. Multiple small T2-hypointense flow voids are seen surrounding the cord (*A, blue arrow*; *B, arrows*). Sagittal (*C*) and axial (*D*) T1-weighted images with gadolinium show abrupt missing pieces (*C, arrows*) within a confluent area of enhancement. These findings are suspicious for spinal dural arteriovenous fistula.

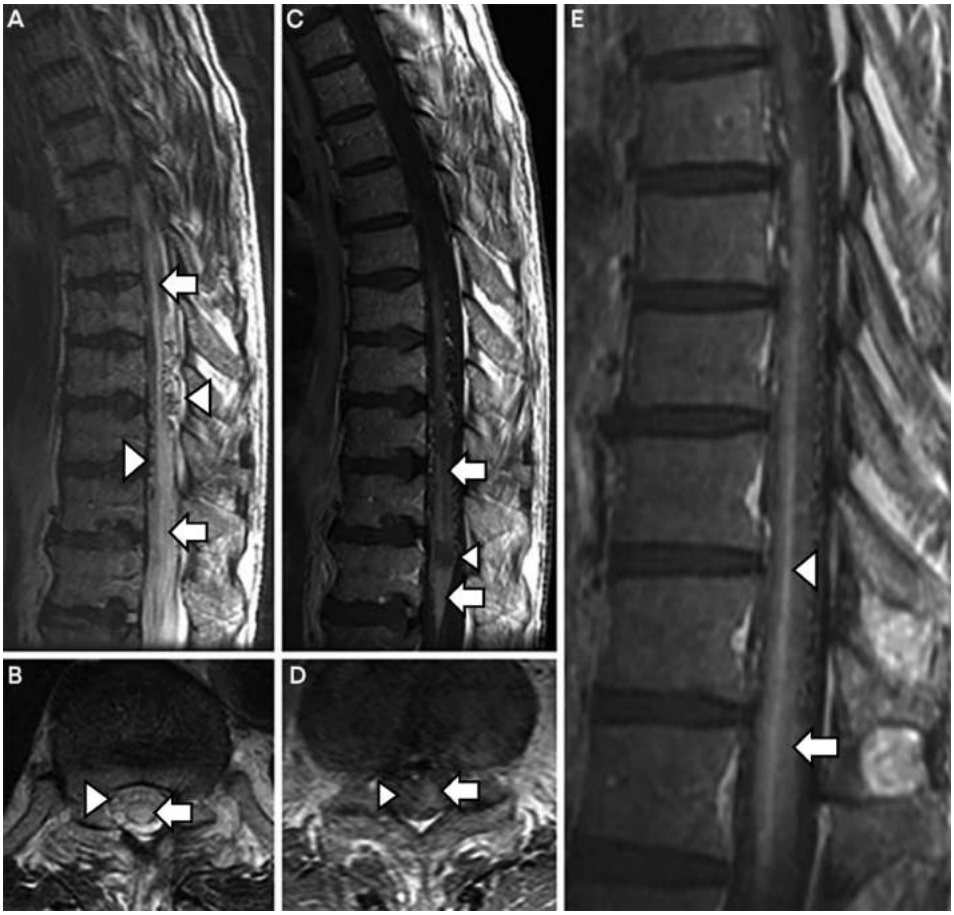

FIGURE 2-8

MRI findings in spinal dural arteriovenous fistula. Sagittal (*A*) and axial (*B*) T2-weighted thoracic spine images reveal longitudinally extensive holocord (entire spinal cord) T2 hyperintensity (*A*, *B*, *arrows*) extending to the conus with associated flow voids along the ventral and dorsal cord surface (*A*, *B*, *arrowheads*). Sagittal (*C*) and axial (*D*) postcontrast T1-weighted images show the missing piece sign (*C*, *D*, *arrowheads*), with a well-defined abrupt segment of gadolinium enhancement missing amid an otherwise homogenous, avid enhancing area (*C*, *D*, *arrows*) Sagittal postcontrast T1-weighted image (*E*) reveals another frequent enhancement pattern with hazy enhancement (*arrow*) and central canal predominance (*arrowhead*).

interference in steady state [CISS], or T2 sampling perfection with application-optimized contrasts using different flip-angle evolution [SPACE]) should be requested as they may improve the yield of demonstrating flow voids. As a general rule, high-quality MRI of the entire spine without T2-hyperintense signal or flow voids reliably excludes spinal dural arteriovenous fistula.[52,54] Flow voids can also be seen with tumors (eg, hemangioblastoma, paraganglioma) and other AVMs. Mild prominence of nontortuous vasculature in the lower thoracolumbar cord is common and not pathologic. Gadolinium enhancement of the spinal cord is common (65% to 85% of cases), and clinicians should not be misled to suspect an inflammatory or neoplastic etiology when features are suspicious for spinal dural arteriovenous fistula. A variety of gadolinium enhancement patterns can be seen, commonly referred to as hazy heterogeneous patterns throughout, although the missing piece sign or hazy with central canal prominence pattern is helpful, when present.[44,55]

Although DSA is the gold standard to identify a fistula, a noninvasive spinal MRA can initially be considered. The typical detection on MRA of the spinal canal may be as high as 80% to 95% and can help localize the fistula level within one to two segments in approximately 95% of cases, but updated technology and an experienced neuroradiologist can increase the sensitivity and specificity.[56-58] DSA has a very high sensitivity in detecting spinal dural arteriovenous fistula with an experienced angiographer; however, evaluation is not perfect, as seen in a review of 53 cases with delayed diagnosis that showed 19% had previously "normal" angiograms (incomplete angiogram or retrospectively demonstrated). Some cases had multiple negative angiograms and discovery of a fistula after durotomy.[55] Fistula visualization may be compromised by atherosclerosis of segmental arteries or thrombosis of veins.[59] Intracranial localization with four-vessel angiography should be considered,[60,61] particularly for cervical cases. Feeder vessels from the pelvis may also be identified.

ADDITIONAL TESTS. Lumbar puncture should be avoided when spinal dural arteriovenous fistula is strongly suspected.[46] When obtained, findings generally reveal noninflammatory CSF; however, dramatic inflammation with pleocytosis has been seen.[62] Polyradiculopathy may be seen on nerve conduction studies and EMG.[63]

TREATMENT. Treatment options for spinal dural arteriovenous fistula include embolization of the fistula via DSA (approximately 70% to 80% efficacy) or surgical disconnection of the draining vein (98% efficacy).[4,64-66] Embolization via DSA offers the advantage of avoiding surgery and treating at diagnosis; however, failure rates are higher, and complexity may be too high of a risk in some.

LONG-TERM FOLLOW-UP AND PROGNOSIS. Improvement (>40% of cases) or stabilization (>30% of cases) is expected after treatment. The strongest predictor of outcome is clinical severity before treatment.[67] Follow-up MRI is commonly obtained approximately 3 months after treatment and may show persistent T2 hyperintensity or enhancement, but enlarged perimedullary vessels should not be seen and raise concern for treatment failure. Ultimately MRI shows significant improvement or resolution of T2-hyperintense signal in 80% of patients.[67]

Spinal Epidural and Pial Arteriovenous Fistulas

Spinal epidural arteriovenous fistula results from an anomalous connection between paraspinal or paravertebral arteries and the epidural venous plexus and typically presents as a slowly progressive venous congestive myelopathy.[68] Additional features may include compression of the cord or nerve roots from venous distention. Patients may have an osseous or nonosseous spinal epidural arteriovenous fistula.[69] Lesions are commonly located in the cervical or upper thoracic level, and MRI findings mirror spinal dural arteriovenous fistula with the addition of possible vascular engorgement of the epidural space. Angiogram demonstrates involvement of the epidural venous plexus, often with a venous pouch.[68] Pial arteriovenous fistulas (superficial intradural with direct shunt between spinal cord arteries and veins) are rare AVMs typically found anteriorly near the conus that also present with venous congestive myelopathy (60% of cases), whereas others present with hemorrhage (35%).[70]

Other Arteriovenous Malformations

Other spinal and paraspinal AVMs can also present with venous congestive myelopathy mirroring spinal dural arteriovenous fistula, although other clinical

KEY POINTS

- Inappropriate corticosteroid use for suspected alternative diagnoses can lead to clinical worsening in spinal dural arteriovenous fistulas from exacerbation of venous hypertension and thus should be avoided.
- Spinal cord T2-hyperintense signal is present in approximately 95% of cases of spinal dural arteriovenous fistula, is often longitudinally extensive (three or more vertebral body segments), and frequently extends to the conus (90% of cases).
- Flow voids (engorged perimedullary veins) are seen on the dorsal more than the ventral surface of the cord in approximately 80% of cases of spinal dural arteriovenous fistula.
- Gadolinium enhancement of the spinal cord is common in spinal dural arteriovenous fistula, and clinicians should not be misled to suspect an inflammatory or neoplastic etiology when features are suspicious for spinal dural arteriovenous fistula.
- Although digital subtraction angiography is the gold standard to identify a fistula, a noninvasive spinal magnetic resonance angiogram can initially be considered to potentially help localize the fistula before the formal angiography.

presentations (eg, hematomyelia) generally predominate given the location of the arteriovenous shunt and friable vessels.

Miscellaneous Causes of Venous Ischemic Myelopathy

Behçet disease is a systemic vasculitis with significant venous involvement. Pathology/thrombus in spinal cord venous structures is postulated in some patients.

Other unusual causes of venous congestive myelopathy have been reported. Any process inhibiting venous outflow from the intrinsic veins of the spinal cord through the caval system and to the heart could result in venous congestive myelopathy. Inferior vena cava thrombosis,[71] spinal cord compression from an enlarged vena cava,[72] and caustic ingestion with mediastinitis and venous congestion have been seen as unusual mechanisms of myelopathy,[73] whereas obstruction of the inferior vena cava from congenital variants or thrombosis has also been seen with polyradiculopathy.[74-76] Cases of extraspinal arteriovenous fistulas occur as well.[77,78] Rare reports of a prothrombotic state with venous thrombosis (eg, pelvic vein thrombosis, cancer, sepsis),[79] epidural infection leading to venous thrombosis, spinal cavernous malformations with recurrent hemorrhage increasing venous congestion,[80] and nitrogen emboli of the venous system[81] are additional causes of venous ischemia. Spinal cord compression from anomalous arteries (eg, vertebral arteries) with associated syrinx has been reported.[82] A study with biopsies revealing venous congestive myelopathy ultimately demonstrated spinal dural arteriovenous fistula in only three of seven cases, suggesting difficulty in diagnosis or incompletely understood mechanisms.

HEMATOMYELIA

Intraparenchymal spinal cord hemorrhage (hematomyelia) is very rare. Trauma is the most common cause,[83] followed by intramedullary spinal cord cavernous malformation and AVMs; many additional causes are also been reported. Hematomyelia presents similarly to spinal cord infarction, frequently with severe back pain at onset (localizing the hemorrhage) followed by rapid severe myelopathy deficits; however, deficits can often progress over days instead of hours. Recognition of blood products within the spinal cord on MRI (T1 hyperintensity, dark T2 hypointensity, gradient recalled echo [GRE] hypointensity) is critical as it significantly shifts the differential diagnosis and acute management toward a focus on hematomyelia.

Cavernous Malformation

Intramedullary spinal cord cavernous malformations are likely the most common etiology of nontraumatic hematomyelia. Cerebral cavernous malformations are nonshunting vascular malformations found in about 0.5% of the general population,[84-86] whereas intramedullary spinal cord cavernous malformations represent approximately 5% of all cavernous malformations in the CNS.[87] Only 5% of patients with an intracranial cavernous malformation have an intramedullary spinal cord cavernous malformation, whereas approximately 25% of patients with intramedullary spinal cord cavernous malformations have an intracranial cavernous malformation.[86] A cavernous malformation is a cluster of tightly packed dilated sinusoidal veins with no intervening parenchyma and no feeding artery or draining vein. Lesions occur not only in the spinal cord but also

within nerve roots. Intramedullary spinal cord cavernous malformations have a slight male predominance, with median age of presentation in the fourth decade.[86,88] Cavernous malformations are now generally understood to develop over a lifetime. Most cases of intramedullary spinal cord cavernous malformations are spontaneous, but a familial history is present in approximately 10% of cases or as high as 50% in patients with multiple cavernous malformations[89]; radiation and trauma are other common predisposing factors.

Intramedullary spinal cord cavernous malformation most commonly presents clinically with acute hematomyelia[86] and carries an average hemorrhage risk of approximately 1% to 5% per year per cavernous malformation. Annual hemorrhage rates as high as 10% can be seen with localizing symptoms or prior episodes of hemorrhage,[86] and lesions larger than 1 cm in size are also at higher risk of bleeding. Hemorrhage rates may be as low as 0.8% with incidental intramedullary spinal cord cavernous malformation.[86] Significant back or radicular pain at the site of hemorrhage is reported in 20% to 30% of patients[86,88]; isolated episodes of pain without neurologic deficits may be seen (**CASE 2-3**). Strenuous activity before onset is reported in approximately 30% of patients.[86] Myelopathy deficits can have a range of severity, depending on the extent of hemorrhage. Some patients have progressive deficits due to venous microhemorrhage with compression of the surrounding spinal cord, a stepwise presentation with recurrent hemorrhage, or a mixture of acute and progressive decline. Symptoms may worsen during pregnancy or the puerperium or with trauma.[8]

It is important to understand the radiographic characteristics of intramedullary spinal cord cavernous malformation and findings of recent hemorrhage to further define the likelihood of intramedullary spinal cord cavernous malformation contributing to a patient's symptoms. A well-defined lobulated masslike lesion within the parenchyma of the spinal cord with heterogeneous T1- and T2-weighted signal intensity surrounded by a well-defined dark T2-hypointense rim (hemosiderin deposition) (**FIGURE 2-10**) with a classic popcorn appearance is typical, which can be best appreciated with GRE and susceptibility-weighted imaging (SWI). Usually little perilesional edema and minimal/absent gadolinium enhancement are seen in stable lesions. Calcifications are less common in intramedullary spinal cord cavernous malformations than in cerebral cavernous malformations. Size varies from 2 mm to 3 mm to several in centimeters in diameter.[90] The presence of bright T1-hyperintense signal and perilesional edema are helpful radiographic features suggesting recent hemorrhage in cavernous malformations,[91] with less distinctive features specifically for cavernous malformations in the acute setting, which can make initial diagnosis difficult. No strong predisposition for the cervical or thoracic spinal cord is seen. Lesions are angiographically occult, but DSA is still often performed for assessment of alternatives.

Management of intramedullary spinal cord cavernous malformation is focused on avoiding further deterioration with recurrent hemorrhage; observation is typically recommended for asymptomatic cavernous malformations or those with minimal symptoms.[92] In patients with progressive neurologic deterioration, surgical resection is recommended. Symptomatic improvement (in approximately 30% of patients) is possible with careful patient selection for surgical resection,[86] but the majority of patients benefit from stabilization, even with an incomplete resection.[86,88] Favorable factors for

KEY POINTS

- Treatment options for spinal dural arteriovenous fistula include embolization of the fistula via digital subtraction angiography or surgical disconnection of the draining vein. Improvement or stabilization is expected after treatment.
- Intraparenchymal spinal cord hemorrhage (hematomyelia) is very rare. Trauma is the most common cause, followed by intramedullary spinal cord cavernous malformations and arteriovenous malformations; many additional causes have also been reported.
- A well-defined lobulated masslike lesion within the parenchyma of the spinal cord with heterogeneous T1- and T2-weighted signal intensity surrounded by a well-defined dark T2-hypointense rim (hemosiderin deposition) with classic popcorn appearance is typical of an intramedullary spinal cord cavernous malformation, which can be best appreciated with gradient recalled echo and susceptibility-weighted imaging.
- Management of intramedullary spinal cord cavernous malformation is focused on avoiding further deterioration with recurrent hemorrhage; observation is typically recommended for asymptomatic cavernous malformations or those with minimal symptoms.

surgical benefit include surgery less than 3 months after symptom onset,[88] cavernous malformation size greater than 1 cm, and predominantly motor symptoms, which along with gross total resection via hemilaminectomy can be predictive of improved neurologic outcome. Preoperative sensory symptoms and pain may be predictive of poor postoperative improvement. Various data have been reported on radiosurgery in cerebral cavernous malformations, yet it remains unclear whether it has any demonstrable benefit in the management of intramedullary spinal cord cavernous malformations.[90]

Arteriovenous Malformation

An arteriovenous shunt comprises a direct connection between feeding arteries and vein(s) without an intervening capillary network. When an arteriolovenular network (nidus) transitions between feeding artery(s) and vein(s), it is referred to as an AVM. When a direct connection exists between the main arterial feeder(s) and a single vein, it is termed an arteriovenous fistula. Although spinal dural arteriovenous fistula (type I) comprises 70% of spinal arteriovenous shunts, 30% of cases are secondary to types II through V: intramedullary glomus AVM (type

CASE 2-3

A 50-year-old woman presented with abrupt-onset neck pain and upper extremity weakness. Her pain became progressively more severe over days, extending into her shoulders. Within a few more days, the pain extended into her left arm and was accompanied by a loss of strength. She also developed left-sided ptosis. An MRI of the cervical spine was obtained and revealed blood in the left anterior spinal cord between C6 and C8. Symptoms were observed over several weeks, and her ptosis resolved and left arm strength returned to normal.

Over the next few years of observation, she developed many episodes of recurrent neck and upper extremity pain lasting hours to weeks, with altered dysesthetic sensation and contact allodynia. Neuroimaging findings were consistent with a cavernous malformation (FIGURE 2-9). Surgical resection was performed, and the patient had a good outcome, with improvement in symptoms.

COMMENT

Hematomyelia is an important cause of acute myelopathy that frequently presents with significant pain and a range in severity of accompanying neurologic deficits. Of note, this patient had an accompanying Horner syndrome, important to recognize as an occasional feature in acute cervical myelopathy damaging descending first-order neurons in the sympathetic tracts. Intraparenchymal blood products were identified by the presence of mixed dark T2 hypointensity and T1 hyperintensity. A well-demarcated dark T2-hypointense rim around the small masslike lesion was typical of intramedullary spinal cord cavernous malformation, the most common cause of hematomyelia. Recurrent symptoms attributable to the intramedullary spinal cord cavernous malformation were an appropriate indication to pursue surgical resection with the goal of preventing further hemorrhage.

II), intramedullary juvenile AVM (type III), perimedullary AVF (type IV), and extradural AVF (type V).[93] To simplify for the purpose of this article, types II through V are referred to as *spinal AVM*, although clinical and radiographic differences exist between types, as mentioned earlier with discussion of perimedullary arteriovenous fistula. Notably, a spinal cord aneurysm is associated with 30% to 40% of spinal AVMs.[94] Brain AVMs have an incidence of 1.3 per 100,000 patient-years, 10 times more common than spinal AVMs.[95] Spinal AVMs most commonly present in the third or fourth decades, with no clear gender predilection. These malformations are associated with genetic syndromes including germinal (hereditary hemorrhagic telangiectasia, capillary malformation-AVM syndrome) and somatic (Cobb syndrome; Klippel-Trenaunay-Weber syndrome; and congenital lipomatous overgrowth, vascular malformations, and epidermal nevi syndrome) mutations.[70] Spinal arteriovenous metameric syndrome involves vascular malformations of multiple tissue layers from skin to spinal cord, likely caused by a somatic mutation. Cobb syndrome demonstrates arteriovenous abnormalities involving all tissue layers of a metameric segment.[70]

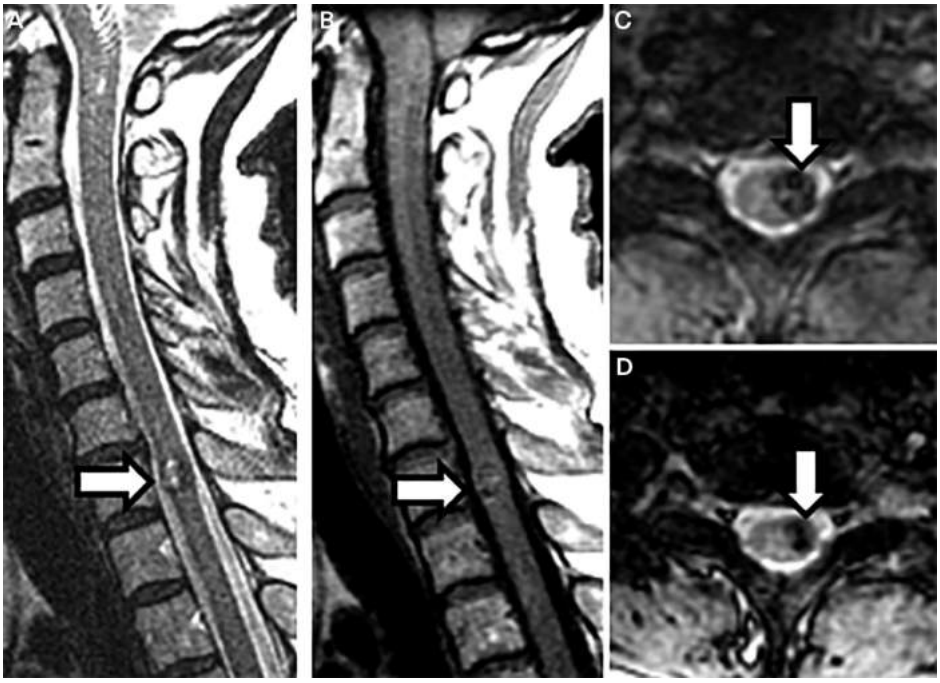

FIGURE 2-9

Imaging of the patient in CASE 2-3. Sagittal T2-weighted (*A*) and T1-weighted (*B*) MRI of the cervical spine shows hematomyelia with mixed T2 hypointensity (*A*, *arrow*) and T1 hyperintensity (*B*, *arrow*). Corresponding axial T2-weighted images (*C*, *D*) show a well-demarcated T2-hypointense rim (*C*, *arrow*; *D*, *arrow*) around the masslike lesion, typical of intramedullary spinal cord cavernous malformation.

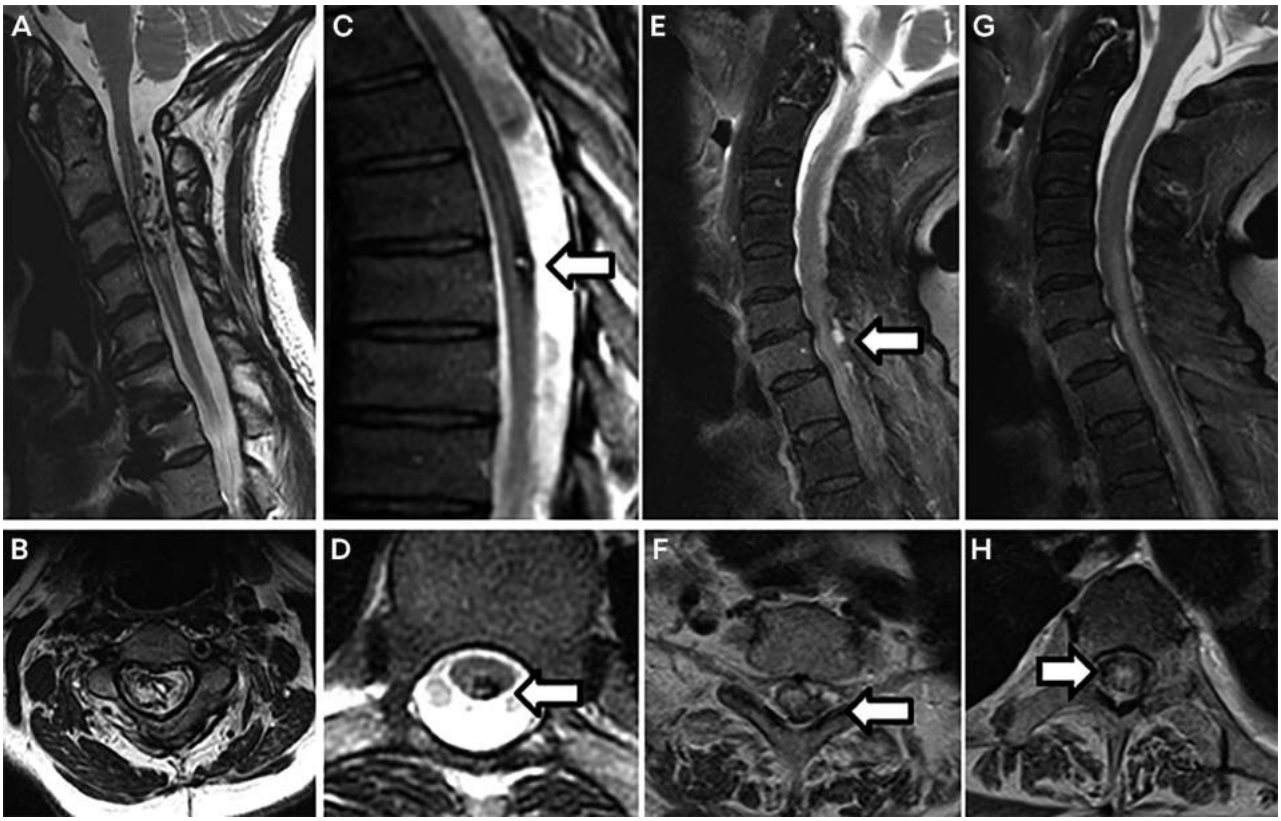

FIGURE 2-10

MRI findings in hematomyelia and extraparenchymal hemorrhage. Sagittal (*A*) and axial (*B*) T2-weighted cervical spine MRIs show intramedullary and extramedullary flow voids consistent with spinal arteriovenous malformation. Sagittal (*C*) and axial (*D*) T2-weighted thoracic spine images reveal a mixed-signal central lesion with surrounding dark T2-hypointense rim (*C, D, arrows*) typical of a cavernous malformation. Sagittal (*E, G*) and axial (*F, H*) T2-weighted MRIs of the cervical spine reveal an extramedullary mixed T2-hyperintense and T2-hypointense signal dorsal to the cord (*E, F, arrows*) representative of anticoagulation-associated subdural hematoma, with concurrent subarachnoid hemorrhage best appreciated on axial view (*H, arrow*) and associated cord edema with T2-hyperintense signal on sagittal views (*G*).
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The predominant clinical presentation of spinal AVM is acute myelopathy secondary to hematomyelia (50%).[96] Pain is frequent at onset; the deficits are typically acute and can evolve over days. Other mechanisms contributing to clinical decline include subarachnoid hemorrhage (SAH), compression from mass effect (venous dilations, pouches, or, less likely, aneurysms), arachnoiditis with scarring and occlusion of small vessels causing arterial ischemia,[8] and venous thrombosis with ischemia.[22,42,94] Most patients (>75%) will show favorable spontaneous recovery in the early period after acute symptoms of hematomyelia.[97] The annual hemorrhage risk in spinal AVM is 4%, with the rate of recurrent hemorrhage higher at 10%.[22,96] Patients can also present with radiculopathy, typically because of compression by a dilated epidural vein.[70] Vascular steal and high-output heart failure have rarely been suggested.[70]

The predominant diagnostic feature on MRI is the presence of intramedullary flow voids (FIGURE 2-10A). As discussed earlier, it is important to look for an accumulation of blood products with mixed T1 and T2 signal changes, dark T2

hypointensity, and active/recent blood products with T1-hyperintense signal. Prior hemorrhage can reveal cavitation, localized swelling, or atrophy in chronic cases. DSA confirms the presence of an AVM, defines its angioarchitecture, and helps plan treatment.

The complexity of approaching a spinal AVM should be handled by an experienced angiographer and neurosurgeon. Treatment strategies are controversial given the rarity of spinal AVMs, and disabling deficits can occur as a complication of treatment in 5% to 25% of patients.[97] Emergent surgery is often avoided because of complicating factors in acute hematomyelia and because of the natural history of early improvement in most patients.[98] The largest natural history study suggests more than 70% of patients deteriorate within 4 years (more common early), and thus earlier treatment is generally recommended.[97] Endovascular embolization, surgical resection, and radiosurgery are options. Even partial treatment can reduce hemorrhage risk; therefore, combination or staged treatment approaches are often planned.[96,99,100] After treatment, the annual hemorrhagic risk is generally absent with complete obliteration and drops to 3% for partial treatment.[96,101] Endovascular treatment is typically preferred because of the high risk for intraoperative complications.[96] Surgical resection may be preferred in posterolaterally located AVMs fed by radial arteries from the posterior spinal artery.[70] Stereotactic radiosurgery may be considered in some cases with favorable anatomy and may be used in addition to endovascular treatment.[102]

Other Causes of Hematomyelia

Beyond the two most common identifiable causes of hematomyelia (intramedullary spinal cord cavernous malformation and AVM), many other mechanisms have been reported. TABLE 2-5 provides a representative overview of categorization.

Spinal cord aneurysms present more commonly with spinal SAH than hematomyelia. Anticoagulants have been associated with hematomyelia. Bleeding diatheses (eg, von Willebrand disease, factor VIII/VII/XI deficiencies) have been noted in some cases.[103,104] Gowers intrasyringal hemorrhage is a rare syrinx-related hemorrhage.[105] Spinal dural arteriovenous fistula, vasculitis, and radiation with or without thrombocytopenia are other mechanisms.[83] A component of hemorrhagic transformation after spinal cord infarction can sometimes be seen. The question of a possible inflammatory or infectious etiology is sometimes raised in cases without an identifiable cause, but clear associations are very unusual and rarely reported. Longitudinally extensive transverse myelitis with associated hemorrhage has been reported postinfluenza,[106] in addition to cases associated with varicella-zoster virus and herpes simplex type 2 infections, more typically in immunocompromised adults.[107-109] Acute hemorrhagic leukoencephalitis has also been reported with spinal cord involvement.[110] Small asymptomatic capillary telangiectasias have reported in the spinal cord[111] but would not be expected with clinical hematomyelia. Despite extensive evaluations, a number of hematomyelia cases have an indeterminate mechanism.

EXTRAPARENCHYMAL SPINAL HEMORRHAGE

Extraparenchymal hemorrhage of the spinal canal is categorized into SAH, subdural hemorrhage, and epidural hematoma (most common).

KEY POINTS

- **Although spinal dural arteriovenous fistula (type I) comprises 70% of spinal arteriovenous shunts, 30% of cases are secondary to types II through V: intramedullary glomus arteriovenous malformation (type II), intramedullary juvenile arteriovenous malformation (type III), perimedullary arteriovenous fistula (type IV), and extradural arteriovenous fistula (type V).**
- **The predominant clinical presentation of spinal arteriovenous malformation is acute myelopathy secondary to hematomyelia.**

Epidural Hematoma

Spinal epidural hematoma is most commonly encountered in trauma or after surgery, epidural catheterization, or lumbar puncture. Epidural hematoma is estimated to be approximately 4 times more common than subdural hematoma.[8] In the postsurgical setting, deficits may be present immediately after surgery or develop in the subsequent hours to days. Symptomatic postsurgical epidural hematoma involves significant motor deficits in 80% of patients or isolated intractable pain in 20% of patients.[112] Other diagnostic possibilities should also be considered, with new severe myelopathy immediately post–spinal surgery occurring in approximately 0.2% of patients,[113] including inadequate decompression, spinal cord ischemia, graft/cage dislodgement, or presumed direct surgical trauma. A prospective study routinely evaluating for the radiographic presence of spinal epidural hematoma within 2 to 5 days of lumbar decompression demonstrated spinal epidural hematoma with thecal sac compression in 58% of patients; none had new deficits, highlighting that spinal epidural hematoma can be an asymptomatic finding.[114]

The incidence of spontaneous spinal epidural hematoma is much rarer than postsurgical spinal epidural hematoma, estimated at 0.1 per 100,000 person-years.[115] Notably, radicular pain is common at onset.[8] Attributable causes are usually secondary to coagulopathy (eg, anticoagulation, liver disease, or portal hypertension). Epidural hematomas have been described after exertion/straining and venous thoracic outlet syndrome[116] and as a rare phenomenon of dural and epidural arteriovenous malformations.[117,118]

TABLE 2-5 **Hematomyelia**

Hematomyelia etiologies	Helpful features
Trauma	Most common cause of hematomyelia
Cavernous malformation	Common cause of hematomyelia; young male patient; heterogeneous lobular mass, popcorn appearance with dark rim of T2 hypointensity; pain frequently precedes myelopathy deficits
Arteriovenous malformation	Common cause of hematomyelia; young patient; intramedullary flow voids
Bleeding diatheses and medications	Prior history of bleeds; family history; contributing medications or supplements
Neoplasm	Focal masslike component not typical of intramedullary spinal cord cavernous malformation; history of primary neoplasm; contrast enhancement
Miscellaneous **(spinal cord aneurysm, intrasyringal hemorrhage, vasculitis, radiation, infection [eg, postinfluenza, varicella-zoster virus, herpes simplex virus type 2, acute hemorrhagic leukoencephalitis], checkpoint inhibitor)**	Rare; typical radiographic and clinical features for respective disorders
Idiopathic	Common diagnosis of exclusion in hematomyelia; hypertension as typical risk factor

Degenerative disk disease has been questioned as a contributing factor, and sometimes no mechanism is found.

High-resolution CT of the spine is often the initial imaging modality; however, MRI has better sensitivity and provides detailed information regarding the extent of compression for surgical planning. Acute spinal epidural hematoma is hyperdense on CT, whereas MRI patterns vary depending on timing. T1 signal will be isointense acutely and become more hyperintense in the subacute phase. Conversely, hemorrhage will appear hyperintense on T2-weighted sequences in the first 12 hours but become hypointense in the subsequent acute and subacute phase (FIGURE 2-10). Chronic spinal epidural hematoma will be both T1 and T2 hypointense. Heterogeneous patterns are often seen with anticoagulation-related hemorrhage. In contrast to epidural abscesses, gadolinium enhancement is not expected in spinal epidural hematoma.

Similar to management of other hemorrhages, reversal of any contributing coagulopathy should be performed immediately, and any contributing medications should be discontinued. Treatment of symptomatic spinal epidural hematoma is emergent surgery, with improved outcomes when performed within 12 hours of symptom onset.[119] If patients are asymptomatic or have minimal deficits, conservative management with close observation is an option, but it should be understood that patients may later rapidly deteriorate. Poor outcomes have been associated with worse clinical severity at presentation, motor and sensory impairment, and longitudinally extensive hematomas.[120]

KEY POINTS

- Spinal epidural hematoma is most commonly encountered in trauma or after surgery, epidural catheterization, or lumbar puncture.
- Treatment of symptomatic spinal epidural hematoma is emergent surgery, with improved outcomes when performed within 12 hours of symptom onset.

Subdural Hematoma

Spinal subdural hematoma is much less common than spinal epidural hematoma but can present similarly with acute back pain, myelopathy, or radicular symptoms. Nontraumatic causes are generally iatrogenic from procedures and use of anticoagulation/antiplatelet agents.[83] Chronic spinal subdural hematomas are rare, reported with antiplatelet treatment and anticoagulation,[121,122] but also may be related to prior trauma or small bleeds. In the setting of recent spine surgery, radiographic evidence of spinal subdural hematoma is not uncommon.[123] Management considerations for symptomatic spinal subdural hematoma are similar to spinal epidural hematoma.

Subarachnoid Hemorrhage

Spontaneous spinal SAH is rare, accounting for 1% of all SAHs, and is the initial presenting symptom in 10% of spinal AVMs.[124] Symptoms typically include the sudden onset of severe neck or back pain, and patients also frequently have headache, meningismus, and hyponatremia. Depending on the extent of hemorrhage and etiology, a variable severity in myelopathy deficits can be seen. Redistribution of blood products throughout the CNS and typical complications of SAH may occur (eg, vasospasm). Management is generally conservative, addressing the underlying mechanism, and treating complications similar to cerebral aneurysmal SAH. Etiologies and risk factors include hypertension (common), spinal AVM, aneurysm, vascular neoplasm, intramedullary spinal cord cavernous malformation, spinal dural arteriovenous fistula, vasculitis,[125] blood dyscrasia, collagen vascular disease, anticoagulation, coarctation of the aorta, and acute aortic dissection.[126,127] Of note, spinal aneurysms rarely rupture.[8] Also, the accumulation of subarachnoid blood products in the thecal sac can occur over time from prior lumbar punctures or spinal anesthesia with

anticoagulation and compress the spinal cord and roots, termed subarachnoid space hematoma.

CONCLUSION

Vascular myelopathies represent uncommon but frequently underrecognized neurologic disorders. Clinicians need to carry a high index of clinical suspicion for a possible vascular etiology in patients presenting with myelopathy or the correct diagnosis will often be missed or delayed because of the overlapping clinical and imaging features that may mimic other causes of myelopathy. Providing patients with the proper medical management while avoiding potentially harmful and unnecessary treatments may help improve outcomes.

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