

Diagnosing spinal cord ischemia

Are we doing enough catheter angiography?

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Spinal cord ischemia is an important and potentially treatable cause of acute myelopathy. Similar to brain ischemia, the accurate diagnosis of spinal cord ischemia is necessary to begin timely treatment and avoid debilitating and/or permanent morbidity. Diagnosis in the absence of obvious inciting traumatic, procedural or vascular source (e.g., aortic aneurysm), however, may be elusive for many patients. The recently proposed diagnostic criteria specify the presence of a T2 hyperintensity and/or diffusion-restricted intramedullary spinal cord lesion and supporting factors of associated vertebral body infarction and/or arterial narrowing or occlusion.¹ Spinal cord ischemia is also framed as a diagnosis of exclusion, to be considered after eliminating alternative etiologies such as extrinsic cord compression and inflammatory etiologies. This exclusion alludes to the fact that many of the clinical and imaging abnormalities of spinal cord ischemia are, in isolation, nonspecific and may be insufficient for confident diagnosis.

This issue of *Neurology: Clinical Practice* has 2 articles of interest to the field of spinal cord ischemia. English et al.² presented a single-institution series of spontaneous spinal cord infarction from the Mayo Clinic in Rochester, MN. Among 133 cases of spinal cord infarction found over a 20-year period, they describe 4 patients who had spinal cord TIA. Spinal cord TIA was defined as acute myelopathy that lasted <24 hours in patients who made full recoveries; the deficits were abrupt and brief usually resolving within minutes. Two patients had cervical and 2 had thoracic cord involvement. Symptoms included acute upper extremity numbness, paresthesia, and paraparesis with or without sensory loss. Spinal imaging was not described in these 4 patients, although 1 was reported to have a brain infarct at diagnosis and another a brain infarct 2 years later. The authors conclude that presentation with symptoms typical for spinal cord TIA should be concerning for a vascular etiology and prompt appropriate vascular workup and consideration of risk factor modification. However, in practical terms, given the rarity of spinal cord TIA, they also propose that other differential diagnoses should be “strongly considered”—including spinal dural arteriovenous fistula, degenerative compression, and demyelinating diseases.

Gailloud et al.³ reported a single case of acute spinal cord infarction from intersegmental artery dissection proximal to the artery of Adamkiewicz. MRI showed T2 hyperintense intramedullary changes from T11 to the conus tip with subtle enhancement. Although these imaging results were by themselves not specific, the authors describe them as consistent with subacute ischemia in the clinical setting of pain, weakness, bladder incontinence, and decreased sensation. Catheter angiography demonstrated dissection and subsequent narrowing of the right L1 intersegmental artery, which improved and then resolved over months with anticoagulation. The authors conclude that catheter angiography can in some cases be necessary to determine the pathomechanism and guide the treatment of spinal cord infarction.

These 2 articles add to the body of knowledge about the distinctly uncommon entity of spinal cord ischemia and infarction. Spinal cord TIA, although fleeting and self-limited, may be a harbinger for spinal cord or brain infarction and should prompt risk factor modification.² Mechanisms of spinal cord infarction are myriad and may often require catheter angiography

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for diagnosis—including the previously undocumented mechanism of intersegmental artery dissection proximal to the dominant radiculomedullary artery.³ The great anterior radiculomedullary artery or artery of Adamkiewicz is usually found as a single vessel arising between T8 and L1 on the left side.⁴ Patients with spinal cord ischemia are often younger and have fewer cardiovascular risk factors than those who suffer from brain ischemia.⁵ Spinal cord ischemia is marked by an apoplectic onset of severe back pain and is associated with motor, sensory, and autonomic dysfunction. The clinical presentation varies, however, depending on the cause, vascular territory, size of lesion, and collateral circulation. Diagnosis will often rely on integration of available clinical and imaging findings.

There is unfortunately no standardization of MRI sequences or acquisition parameters to evaluate the spinal cord. Diffusion restriction is the hallmark of ischemia/infarction, yet may not be performed as part of routine spine MRI scans performed for pain. Intramedullary T2 hyperintense changes may occur with ischemia and with other nontraumatic etiologies such as transverse myelitis, demyelinating, inflammatory, neoplastic, hemorrhagic, and/or metabolic diseases including nutritional deficiencies. Intramedullary enhancement is variable—often absent in the acute phase and more likely during the subacute phase—and may also potentially muddle the diagnosis of spinal cord ischemia. Despite advances in magnetic resonance angiography and CT angiography and their ability to detect the artery of Adamkiewicz,^{6,7} their resolution is often insufficient to diagnose subtle arterial pathologies and they are not incorporated into most diagnostic workups for spinal cord ischemia.

If a vascular lesion is suspected, and MRI does not provide an alternative diagnosis or any localizing information, a thorough conventional spinal angiogram should be considered to evaluate the small caliber, complex angioarchitecture of the spinal cord. A higher and earlier diagnostic certainty could widen the treatment spectrum to thrombolysis, endovascular treatment (angioplasty and stenting), and/or intravenous anticoagulation. It might also prevent subjecting patients to therapies (blood pressure augmentation with volume,

vasopressors, lumbar drainage, and steroids) that are ineffective or potentially deleterious for the specific pathology underlying spinal cord ischemia.

In conclusion, given the rarity of spinal cord ischemia, the high sensitivity but limited specificity of MRI abnormalities, and the short window for potential intervention to avoid permanent deficits, spinal cord ischemia remains a difficult problem. It is often a diagnosis of exclusion and requires high-clinical suspicion for appropriate workup. The diagnostic yield and treatment options tailored to the specific pathology can be increased with the use of conventional spinal angiogram.

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