Illustrative Teaching Case

Section Editors: Daniel Strbian, MD, PhD, and Sophia Sundararajan, MD, PhD

Evaluation of a Patient With Spinal Cord Infarction After a Hypotensive Episode

Nuttawan Vongveeranonchai, MD; Manaf Zawahreh, MD; Daniel Strbian, MD, PhD; Sophia Sundararajan, MD, PhD

Case Description

A 72-year-old man with past medical history significant for peripheral vascular disease, bilateral carotid stenosis, hypertension, dyslipidemia, and heavy tobacco use developed sudden severe chest pain and received sublingual nitroglycerin with pain resolution. The patient was able to walk to the ambulance, but on arrival in the emergency room had a systolic blood pressure of 60 mm Hg, which was thought to be secondary to nitroglycerin. He was given intravenous fluids. A few hours later, he complained of being unable to move his legs and was found to be paraplegic with urinary retention and a T4 sensory level to pain and temperature with preserved light touch, vibration, and joint position sense. Additional intravenous fluids resulted in minimal improvement. Emergent MRI of the spine was nondiagnostic, but a follow-up MRI the next day showed abnormal signal intensity within the anterior gray matter on T2-weighted images from the T2 through T5 level, with corresponding signal on diffusion weighted imaging. A computed tomography of the chest showed no aortic dissection. Cardiac telemetry and echocardiogram were unremarkable. A fasting low density lipoprotein was 120. He was treated with intravenous fluids, aspirin 81 daily, an increased statin dose, and eventually discharged to an acute rehabilitation facility. Six month later, his lower extremity strength had improved. He was able to walk 45 feet with a wheeled walker and required intermittent urinary catheterization.

Discussion

Acute spinal cord infarction (SCI) is uncommon, accounting for 1.2% of stroke admissions and 5% to 8% of acute myelopathies.1 The vast majority of spinal infarcts involve the anterior spinal artery (ASA) and have distinct clinical features because of sparing of the posterior columns. These patients have preserved posterior column function despite loss of pain and temperature with bilateral lower extremity weakness. Recognizing spinal cord ischemia early is critical so that the processes causing ischemia can be addressed to limit SCI.

Vascular Anatomy of the Spinal Cord

To understand the mechanisms of spinal cord ischemia, one must first understand the vascular supply of the cord, which leaves the thoracic cord particularly vulnerable to ischemia (Figure). There is a single ASA and 2 or 4 posterior spinal arteries (PSAs). The ASA is derived from branches of the vertebral arteries arising at the level of the foramen magnum. Segmental vessels at the level of each spinal nerve root divide into anterior and posterior radicular arteries. At variable levels these course medially and anastomose with either the ASA or the PSAs. During development the majority of anterior radicular arteries regress leaving only 4 to 8 arteries to feed into the ASA. Fewer anastomoses make this territory more susceptible to ischemia. In the cervical cord the anterior radicular arteries are branches of the vertebral and ascending cervical arteries. In the rostral thoracic cord they are branches of the deep and ascending cervical arteries. In the middle and lower thoracic cord the remaining anterior radicular arteries are less robust and more variable. The lower thoracic and lumbar cord is supplied by the artery of Adamkiewicz, which originates between T9 and L2. The vascular supply of the lumbar sacral cord originates from the hypogastric and sacral arteries.2 T1 to T4 is particularly vulnerable to infarction during global hypoperfusion because of the large distance between radicular arteries.3 The ASA gives rise to sulcal arteries, which supply the anterior two thirds of the cord, including the gray matter, anterior columns, and lateral funiculi. Thus, spinal cord ischemia during hypotension produces the classic picture of motor findings and dissociated sensory loss. The superficial white matter of the cord receives blood supply from circumferential vessels. As a result, there is a watershed border zone between the territories of the sulcal and circumferential arteries, and SCI may not always adhere to the classic presentation.

The PSAs are also derived from the segmental arteries, but fewer posterior radicular arteries regress, leaving 10 to 20 feeder vessels. Because of the more reliable vascular supply of the posterior cord, infarction is seen less frequently.
Penetrating branches from PSAs supply the dorsal columns and dorsal horns.

**Mechanisms of Spinal Cord Ischemia**

Several mechanisms can cause impaired perfusion of the spinal cord. As illustrated by our patient, the upper thoracic cord is a watershed zone between anterior radicular arteries. In one case series, systemic hypotension was identified as the cause of nonsurgical SCI in 11% of the patients. Hypotension during surgery is of particular concern, especially in patients undergoing aortic surgery. Open thoracoabdominal aortic aneurysm repairs are associated with a 5% to 21% risk of perioperative SCI because of aortic cross-clamping. As a precaution, a lumbar drain can be used to maintain cerebrospinal fluid pressure <10 cm H2O during and 48 hours after the procedure, to increase spinal perfusion pressure and protect against increased cerebrospinal fluid pressure that accompanies reperfusion after aortic cross-clamping. Individual radicular arteries can be compromised by arterial dissection in the setting of aortic dissection or can be occluded because of embolization, atherosclerotic plaque rupture, sickle cell crisis, or vasculitic processes including syphilis or giant cell arteritis. Mechanical stress can cause compression or traumatic injury of a radicular artery. Hypercoagulable states, polycythemia, and vasoactive drug use can also contribute to spinal cord ischemia. Spinal vascular malformation may cause neurological deficits because of hemorrhage or occasionally can cause venous infarction clinically indistinguishable from arterial infarction.

**Diagnosis**

**Clinical Features**

Symptomatology is dependent on the spinal level involved. Sixty percent of patients report back or neck pain at the onset of symptoms, usually localized at the level of the lesion. Symptoms generally evolve for several minutes and reach maximal deficit at 5 hours. Because the lateral corticospinal and spinothalamic tracts are supplied by the ASA, infarction classically is characterized by an areflexic flaccid paraplegia/tetraplegia with a dissociated sensory deficit (impaired pain and temperature with preserved proprioception). Over time patients develop hyper-reflexia and spasticity. The autonomic system is also disrupted and loss of bowel and bladder continence is reported in a majority of patients.

Special attention should be paid to patients with lesions between C3 to C5 and T4 to T9. C3 to C5 supplies the phrenic nerve, whereas T4 to T9 supplies the greater splanchnic nerve and, therefore, vasomotor tone. If either level is involved, patients are at risk for respiratory failure or orthostatic hypotension.

The PSA syndrome is exceedingly rare but includes the loss of proprioceptive and vibratory sense below the level of the lesion. Weakness can sometimes be found as the corticospinal tracts lie at the borderzone between the ASA and PSA territories. Cases of central cord infarction manifested by bilateral spinothalamic deficits and transverse infarction manifested by bilateral motor deficits with sensory symptoms attributable to both the spinothalamic and the dorsal horns have been reported in patients with prolonged hypotension. Cervical radiculopathy associated with SCI from vertebral artery dissection has also been described.

**Imaging**

MRI with diffusion weighted imaging can be valuable in the diagnosis of SCI but is not sensitive, and absence of diffusion signal, especially early in the course, does not rule out the diagnosis. Serial MRIs may be helpful. Other MRI abnormalities, which may be seen, include hyperintensity on T2-weighted images, cord swelling, and enhancement with gadolinium on T1-weighted images. Changes within the vertebral bodies may be seen because they share blood supply with the spinal cord. Despite initial insensitivity, MRI must be performed emergently to rule out cord compression requiring emergent intervention. If MRI is contraindicated or unavailable, then a computed tomography and myelography.
can exclude cord compression but are unlikely to provide information about SCI.

**Differential Diagnosis**
The differential diagnosis of acute spinal cord dysfunction includes several nonischemic causes. Spinal cord compression caused by trauma or neoplasm should be emergently ruled out. Although a history of trauma should be sought, falls may be secondary to weakness and may not represent the primary cause of spinal cord dysfunction. Demyelination because of transverse myelitis or multiple sclerosis may present with similar complaints and appearance on neuroimaging, with the exception of diffusion signal.

**Treatment**
Treatment is focused on addressing and correcting the underlying mechanism of injury. For example, in the case of hypotension, fluid resuscitation and pressor support are used to sustain mean arterial pressure at ≥90 mm Hg. In suspected embolization, anticoagulation or antithrombotics can be used. Anticoagulation has been used in the case of dissection but there are no randomized controlled studies of radicular artery dissection. There are no reports in the literature of the use of thrombolysis for SCI.

**Prognosis**
SCI is rare and there are limited data to guide prognostication. A large case series analyzed the outcome of 115 patients. At maximal deficit 81% required a wheelchair and 86% required catheterization. At last follow-up (mean, 3 years), 23% had died. Fifty-eight percent of survivors were able to walk. Of the 74 patients using a wheelchair at the time of discharge, 41% were walking at last follow-up. Of 83 patients requiring catheterization at discharge, 33% were able to void at last follow-up. Lack of preserved motor function at maximal deficit is the strongest predictor of being wheelchair bound and requiring catheterization at the final follow-up.

**Conclusions**
It is important to recognize SCI acutely and address the potential cause. Careful attention to the history and clinical presentation, including vital signs, can provide important clues to the diagnosis. Treatment needs to be initiated without delay. Imaging may confirm the diagnosis, but a negative study does not rule it out.
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