Spinal Cord Infarction with Multiple Etiologic Factors

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Spinal cord infarction is uncommon and usually presents with sudden onset of paralysis and sensory disturbances. A variety of causes are described, but rarely with multiple factors involved. We report a case of a 63-year-old man with a history of diabetes mellitus, hypertension, and osteoarthritis who presented with acute onset of chest pain, numbness, and weakness associated with episodic hypotension. He had incomplete tetraplegia and was areflexic without spasticity. Pain and temperature sensations were impaired below the C7 dermatome and absent below the T4 dermatome bilaterally. Proprioception and vibration sensations were diminished on the right below the C6 dermatome. Magnetic resonance imaging showed spinal cord infarction affecting C6–T3 segments, and severe cervical and lumbar spine degenerative changes. This case illustrates an unusual presenting symptom of spinal infarction, the need to identify multiple risk factors for spinal cord infarction, and the importance of optimal preventive therapy in patients at risk.

KEY WORDS: spinal cord infarction; anterior spinal artery; preventive medicine.
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INTRODUCTION

Spinal cord infarction (SCI) is uncommon compared to cerebral stroke, but the outcome may be more disabling. Due to its rarity, reliable estimates of incidence are scarce; however, one study noted SCI accounted for 1.2% of all strokes.[1,2] SCI due to occlusion of the anterior spinal artery, the principal vascular supply to the spinal cord, characteristically presents with an abrupt onset of bilateral weakness, especially the lower limbs, sudden back pain that radiates caudad, flaccid paraplegia, areflexia, loss of pain and temperature sensations below the level of the lesion, sparing of proprioception and vibration sense, and autonomic dysfunction involving the bladder and bowel.[3] Other SCI syndromes involve disease affecting the aorta, intrinsic spinal arteries, or the radicular artery (see Table 1). Recent literature mentions atherosclerosis and hypertension occurring in approximately 50% of cases of SCI, and disease of the aorta as the most common location of pathology.[4]

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Etiologies listed in a review of 44 cases of SCI seen at two university hospitals over a 10-year period include aortic aneurysm repair, traumatic aortic rupture, arteriovenous malformation, cryptogenic (spontaneous), transient ischemic attack, and cardiac arrest as the most frequent diagnoses.[4] Aortic disease has replaced syphilitic arteritis as the most frequent cause of SCI since the introduction of penicillin.[4] Cervical spondylosis, stenosis of intervertebral foramina, hypotension, and arteritis secondary to diabetes mellitus are reported as infrequent causes of SCI in some series.[2,4–9]

We report a case of SCI with an unusual presentation and no definite cause but having multiple etiologic factors known to predispose to arterial infarction. These include chronic diabetes and hypertension, osteoarthritic degenerative changes of the spine that may cause direct occlusion or impingement of the spinal vessels as they transverse the intervertebral foramina, and an episode of recorded hypotension that may have worsened the extent of the SCI. Because the response to therapy and outcome are poor, the recognition of vascular risk factors and their prevention are emphasized in the discussion.

CASE REPORT

A 63-year-old man with a history of diabetes mellitus type 2, hypertension, and osteoarthritis was transferred to our tertiary-care facility after an acute onset of chest pain, numbness, and weakness while recovering from a mild ileus at a community hospital. While bending over, he experienced sudden substernal pain that did not radiate, lasted 10 min, and was relieved by sitting down. His blood pressure was initially recorded as 205/81 mmHg and he was administered a diuretic and beta-blocker. He was treated with empiric aspirin and clopidogrel at that time; however, an electrocardiogram and serial serum cardiac enzymes were nondiagnostic for myocardial injury. Over the next several hours, he developed urinary retention followed by sudden onset of numbness and weakness in the upper and lower extremities. Systolic blood pressure was recorded at 60 mmHg, and treatment of hypotension with intravenous fluids was initiated.

On admission to our hospital, systolic blood pressure was maintained above 120 mmHg. Neurological examination revealed normal speech, cognition, and cranial nerve function. Motor examination was normal for shoulder abduction (5/5), elbow flexion (5/5), and wrist extension (5/5). Wrist flexion was 3/5, grip strength 1/5, thumb abduction 1/5, and interossei 0/5: all bilaterally. Hip flexion was 0/5 right, 2/5 left; hip extension 0/5 right, 2/5 left; hip adduction and abduction 0/5 right, 3/5 left; knee extension 0/5 right, 3/5 left; knee flexion 0/5 right, 3/5 left; ankle dorsifexion 0/5

Table 1. Spinal Vascular Syndromes and Location of Infarction

Vascular syndrome	Anatomical location of infarction
Paraplegia, areflexia,, sensory loss, dysautonomia, proprioception spared	Anterior spinal artery territory
Dysautonomia, bladder distension, erectile dysfunction, paralytic ileus	Intermediolateral cell column, sympathetic involvement
Brown–Sequard hemisection: ipsilateral paralysis and loss of proprioception, and contralateral loss of pain and temperature sensation	Sulcocommissural artery territory
Orthostatic hypotension	T4–9 segments, splanchnic nerve
Cardiac substernal pain	T4, [C4–T5], sympathetic pathways
Respiratory distress	C3–C5, diaphragm involvement
Watershed zone	T4, [T8–T10], [thoraco-lumbar]
Spinal TIA, episodic painless paraparesis or diplegia	Anterior spinal artery territory
Acute loss of proprioception sensation	Posterior spinal artery territory

right, 5/5 left; ankle plantarflexion 0/5 right, 5/5 left; and foot eversion and first-toe extension 0/5 right, 4/5 left. Passive range of motion in all extremities was full without spasticity.

Rectal examination revealed absent tone. Coordination in the upper limbs, evaluated by point-to-point, finger-to-nose and finger-to-thumb tests, was performed slowly, without tremor or ataxia. In the lower limbs, flaccidity precluded evaluation of coordination by heel-to-knee testing or foot tapping. Reflexes were absent bilaterally in the biceps, triceps, brachioradialis, patellar and Achilles tendons. Plantar responses were equivocal and the cremasteric reflex was absent. Rectal sensation was intact. Pain and temperature sensations were impaired below the C7 dermatome and absent below the T4 dermatome bilaterally. Proprioception and vibration senses were diminished in the right wrist and lower extremity, consistent with an ipsilateral dorsal column lesion involving C6 dermatome and below.

The initial differential diagnoses for the chest pain and bilateral extremity weakness included acute coronary syndrome and acute inflammatory demyelinating polyradiculoneuropathy. On admission to our hospital, the unfolding history and specific physical findings allowed a narrowing of the differential diagnosis to include spinal cord ischemia and possible cord compression. With further questioning, the patient reported paresthesias of the hands associated with movement of the back and neck over several months' duration. After prompt neurologic consultation, immediate magnetic resonance imaging (MRI) of the spine was performed that showed abnormal T2 signal in the anterior cord from C6 to T3 (Figs. 1 and 2), and severe degenerative changes of the cervical and lumbar spine, with narrowing of the right intervertebral foramina and cord compression at C5–6 (Fig. 3).

Computed tomography angiogram of the chest, abdomen, and pelvis showed no dissection of the aorta. Transesophageal echocardiography to evaluate for cardiac embolic source was not possible due to the limited mobility of the patient's neck from degenerative disease of the cervical spine. A transthoracic echocardiogram was performed instead that revealed normal left ventricular function and no evidence of embolic source.

No single cause for the spinal infarction was identified, but the patient's vascular risk factors and severe degenerative disease of the spine are proposed as the most likely etiology. Antiplatelet therapy with aspirin and clopidogrel was continued for prevention of recurrent stroke. The neurological deficits persisted unchanged, and the patient was transferred to the rehabilitation center at our institution.

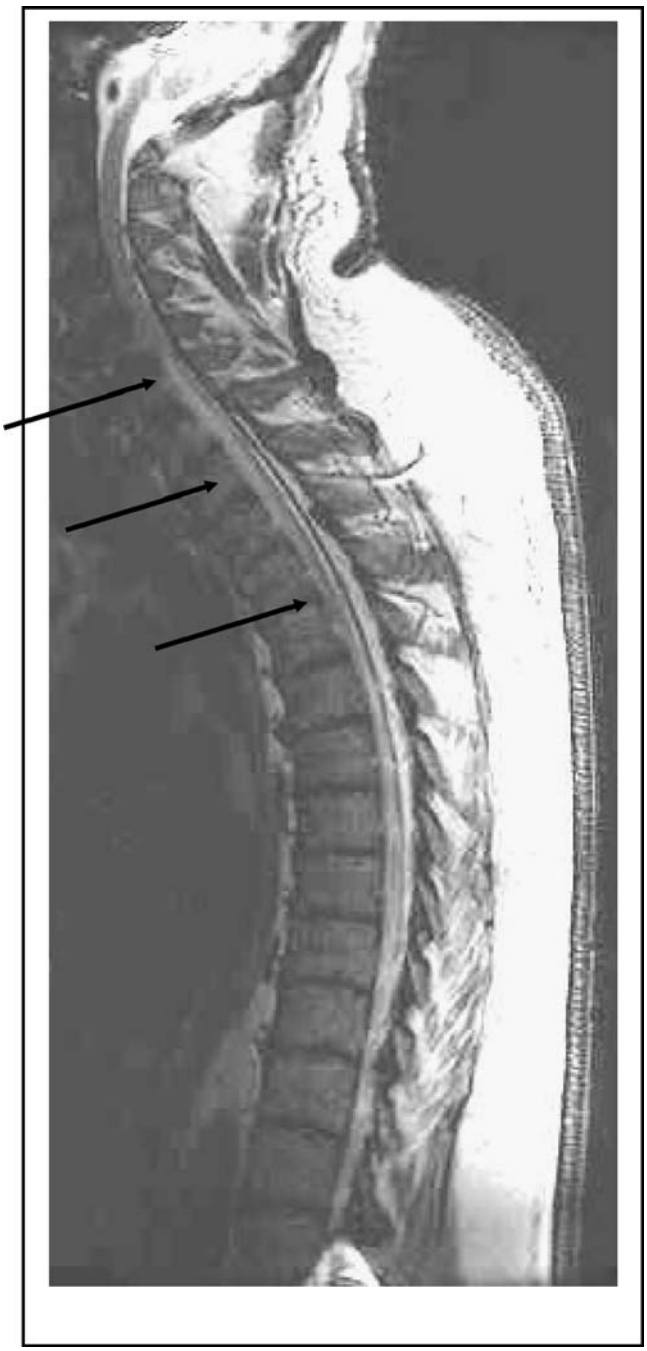

Figure 1. Sagittal T2-weighted sequence MRI with hyperintense lesion of the anterior spinal cord from C6-T3, consistent with infarction (*arrows*).

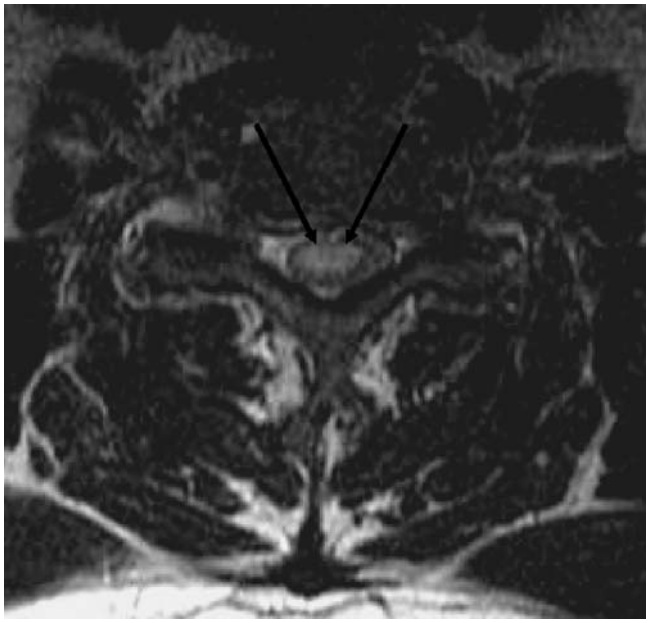

Figure 2. Axial T2 weighted sequence MRI showing cross-section of lesion at the level of C6–7 (*arrows*).

DISCUSSION

The pathogenesis of SCI requires an understanding of the vascular supply of the cord. The anterior spinal artery supplies the anterior two-thirds of the spinal cord, including the anterior horns of gray matter, the corticospinal and lateral spinothalamic tracts. The posterior spinal arteries supply the posterior columns, subserving proprioception and vibration sense, and the posterior horns. The posterior spinal arteries descend separately in the posterolateral sulcus, and receive collaterals from the subclavian, intercostals, and lumbar arteries by way of the dorsal roots.[3,10] Posterior spinal artery infarction is rare.[11]

The anterior spinal artery originates from branches of the intracranial vertebral arteries in the upper cervical region. In the lower cervical and thoracolumbar course, it receives tributaries from the radicular branches of the cervical, intercostal, and lumbar arteries. The thoracic region has the largest spacing between radicular arteries in comparison to other spinal segments.[10] A watershed zone of increased ischemic vulnerability is located at the interface of the anterior and posterior spinal artery territories, described by many authorities to be at the level of T4.[4–6,10] The extent of this patient's lesion suggests a particularly large area of ischemic vulnerability in the distribution of the anterior spinal artery.

The major anterior radicular artery (Adamkiewicz) arises on the left side, anywhere from T5 to L2.[4] Interestingly, the upper level of origin of this artery approximates that of the infarction in our patient. Infarction affecting the major anterior radicular artery is considered clinically indistinguishable from the anterior spinal artery syndrome.[4]

Neurovascular syndromes are defined by the level of the infarction, the most common being the anterior spinal artery syndrome, presenting with weakness, back pain, areflexia, spinothalamic sensory loss, and autonomic dysfunction.[3] Lesions that involve C3–5 segments can compromise respiration, and those affecting the splanchnic nerve of T4–9 segments may result in orthostatic hypotension, sexual dysfunction, and impaired thermoregulation. The Brown–Sequard syndrome, spinal cord hemisection, results from occlusion of the sulcocommissural artery and manifests as ipsilateral paralysis, ipsilateral loss of proprioception, and contralateral loss of pain and temperature sensation. This patient differs from a classic anterior spinal artery pattern due to partial involvement of the dorsal columns that may be explained by occlusion of the radicular posterior spinal arteries on the right side. The presentation of symptoms in this case was also unusual because of the substernal pain, simulating cardiac ischemia.

In two previous case reports of a similar presentation, the authors hypothesize that lesions in the lower cervical region involve pain fibers from the cardiac plexus, resulting in pain.[12,13] Our report emphasizes the importance of rapid identification of neurologic signs associated with chest pain, so that the differential diagnosis is not limited to cardiac ischemia.

Spinal transient ischemic attacks or intermittent spinal claudication syndrome was first described by Dejerine in 1911, and manifests as painless paraparesis or drop attacks without loss of consciousness.[4] Ischemia due to impingement of the vascular supply to the cord due to degenerative changes in the cervical spine is a possible explanation for the intermittent transient sensory symptoms of upper extremities that the patient exhibited prior to presentation. The association of these symptoms with manipulation of the neck also suggests either intermittent compression of the cord or nerve roots, also due to the spondylosis seen on MRI.

The patient who presents with acute paraparesis is a medical emergency that requires prompt evaluation and diagnosis, primarily to exclude the need for immediate surgical correction of a possible sudden cord compression. Once the differential diagnosis is narrowed to ischemic etiology by emergent imaging, vascular risk factors such as diabetes

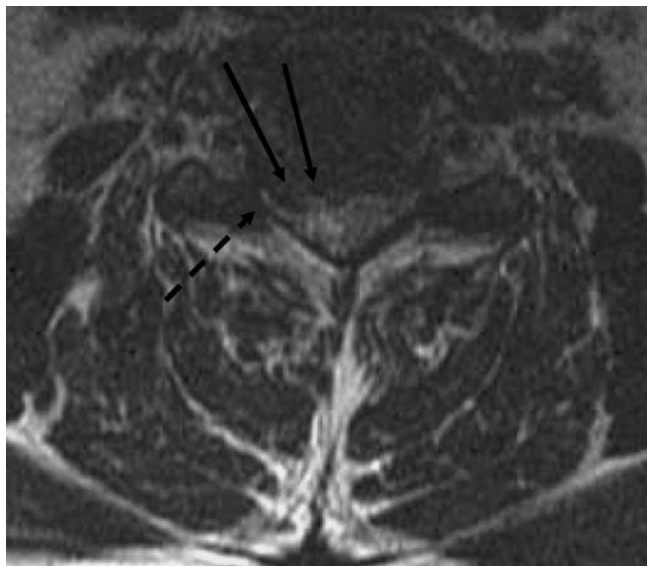

Figure 3. Axial T2-weighted sequence MRI showing narrowing of the right intervertebral foramina (*dashed arrow*) and cord compression at the level of C5–6 (*arrows*)

mellitus, hypertension, hyperlipidemia, and the various causes of arteritis are addressed. Similar to cerebral stroke management, acute management of SCI includes close monitoring and avoidance of hypotension. After identification of a risk factor, measures to reduce the significance of the insult with appropriate medical management are undertaken.

Because of the relative rarity of SCI, no studies at this time specifically address measures for anticoagulation in spinal stroke. However, extrapolation from current recommendations for prophylaxis of recurrent cerebral stroke and myocardial infarction suggest inhibition of platelet aggregation as potentially beneficial, care being taken to screen patients for increased risk of bleeding.[14] Ongoing care includes referral for inpatient rehabilitation and long-term management and prevention of medical complications of immobility.

SCI may present with multiple etiologic factors including diabetes mellitus, atherosclerosis, hypertension, episodic hypotension, and degenerative disease of the spine. Sudden weakness and sensory symptoms typically characterize the onset of SCI, but acute chest pain can be an unusual presenting symptom of spinal infarction, simulating cardiac ischemia. The prognosis of SCI is generally poor, emphasizing the importance of optimal preventive therapies. Patients with multiple risk factors may require antihypertensive agents, strict blood glucose control, appropriate antiplatelet therapy, and, if indicated, treatment for infectious or immune causes of arteritis. Medical and surgical procedures known to increase the risks of SCI, such as arteriography or lumbar epidural anesthesia, should be replaced by less invasive techniques when possible.[4,15] The prompt recognition of the symptoms of spinal ischemia, and early observance and aggressive reduction of risk factors would help in the prevention of SCI and permanent sequelae.

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