Acute spinal cord infarction syndrome unusually mimicking Transverse myelitis: A case report

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Abstract

Anterior Spinal Cord Infarction (ASCI) syndrome typically results from ischemia that affects the anterior spinal artery territory characterized by history of sudden onset flaccid paraplegia or quadriplegia depending on the level of the lesion. We describe a 57-years-old woman with sudden onset flaccid weakness of the lower limbs and numbness of the feet of two days duration, preceded by a day history of fever and several episodes of vomiting. Examination initially revealed bilateral hypotonia, hyporeflexia and dense paraplegia, with preservation of dorsal column sensations (fine touch/2-point discrimination, position and vibration sensation). Twenty-four hours thereafter a repeat examination showed hyper-reflexia and extensor plantar response with spastic paraplegia. Thoracic spine Magnetic Resonance Imaging (MRI) revealed thinning of the spinal cord extending from T1 to T9 levels with associated widening of the thecal sac suggestive of anterior spinal cord infarction with no other signal changes seen within the substance.

Introduction

Spinal cord infarction is a rare but devastating disorder of the spinal cord where patients typically present with sudden onset paraparesis or quadriparesis, depending on the level of the spinal cord involved.[1] It is caused by a wide array of pathologic conditions and the diagnosis is generally made clinically, with neuroimaging modalities to either confirm the diagnosis or exclude other conditions. Anterior spinal artery syndrome is the most common clinical presentation of a spinal cord infarction.[2] It typically presents as loss of motor function, pain and/ or temperature sensation, with relative sparing of proprioception and vibratory sense below the level of the lesion. In the acute stages, flaccidity and loss of deep tendon reflexes are characteristic findings, while spasticity and hyperreflexia develop over days and weeks. The flaccidity may be explained by the earlier involvement of the anterior horns bilaterally, while the spasticity results from subsequent involvement of the lateral corticospinal tracts bilaterally. Autonomic dysfunction may be present and can manifest as hypotension (either orthostatic or frank hypotension), sexual dysfunction, or bladder and/or bowel disturbance. In the evaluation of patients with spinal cord ischemia, it is important to recognize that hypotension may both be a cause as well as a manifestation. Rostral Anterior Spinal Artery (ASA) infarctions produce sensory loss in all modalities because of involvement of the medial lemnisci in the medulla. Respiratory symptoms may be seen if the lesion involves the rostral cervical cord. Patients usually present with bilateral ASA deficits, however unilateral ASA deficits have been frequently reported.[3] This occurs either because of occlusion of a unilateral sulcal artery, or because incomplete collateralization with the Posterior Spinal Arteries (PSA) maintains perfusion on one side of the cord.

Anatomically, the spinal cord is supplied by three major vessels arising from the vertebral arteries in the neck. There is an unpaired ASA and a pair of PSA. The former supplies the anterior two-thirds while the latter supplies the posterior one-third of the spinal cord. Both anastomose distally at the *Conus medullaris*. The ASA arises from the vertebral arteries at the level of the foramen magnum. It runs along the center of the anterior aspect of the spinal cord in the anterior median sulcus from the foramen magnum to the *Conus medullaris*, making it the longest artery in the body. Although it is typically continuous throughout its course, the diameter of the ASA varies considerably throughout its length. It is smallest in the thoracic segment and largest in the lumbosacral region. Along its course, the ASA is augmented by radicular arteries. These small arteries enter the spinal canal though the intervertebral foramen and supply blood to the emerging nerve roots. They originate from the vertebral arteries, intercostal arteries, or in rare instances, directly from the aorta. Typically just 6 to 10 of the 31 pairs of radicular arteries (one pair at each spinal level) contribute to the ASA. These arteries usually enter the spinal canal from the left neural foramen, but may be bilateral, particularly in the cervical spine. The thoracic spinal cord is particularly dependent on radicular contributions and may be the most vulnerable to infarction. The most prominent thoracic radicular artery is the artery of Adamkiewicz, also known as the artery of the lumbar enlargement. The artery of Adamkiewicz contributes to the ASA between the T9 to T12 level in 75 percent of individuals, but may be found above and below this level.[4] From the ASA, sulcal arteries run into the center of the cord and then branch to the right or the left to supply the deep structures of the spinal cord. The ASA also contributes to a peripheral arterial plexus, which gives off radial branches to supply the periphery of the cord. The ASA supplies blood to the anterior horns of the gray matter, spinothalamic tracts, and corticospinal tracts.

Spinal blood flow just like the brain is also influenced by perfusion pressure, which is the difference between mean arterial pressure and intraspinal canal pressure. It is maintained at a constant level over a range of mean arterial pressures through intrinsic autoregulatory mechanisms.[5]

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Key words: Spinal cord; infarction; anterior spinal artery; paraplegia.

Conflict of interest: The authors have no conflict of interest to declare.

Availability of data and materials: All data underlying the findings are fully available.

Ethics approval and consent to participate: No ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals. Informed consent was obtained from the patient included in this study.

Consent for publication: The patient gave her written consent to use her personal data for the publication of this case report and any accompanying images.

Received for publication: 21 September 2020.
Revision received: 20 March 2021.
Accepted for publication: 21 May 2021.

Pyramid Journal of Medicine 2021; 4:110
doi:10.4081/pjm.2021.110

However, there are lower and upper limits of systemic blood pressure beyond which autoregulation fails. Severe systemic hypotension or increased intraspinal canal pressure may decrease the perfusion pressure and thereby putting the cord at risk. Because the spinal cord exists in a fixed space, intraspinal canal pressure is also sensitive to changes in the contents of the spinal canal and may rise significantly in disease states.

Case Report

A 57-years-old woman who presented with sudden onset ascending weakness of the lower limbs of two days' duration. The weakness initially started with the left leg and over some few hours involved the right leg. This was preceded by a day's history of fever and several episodes of vomiting with numbness of the feet but there was no bladder or bowel disturbance. She had no prior history of low back pain, swelling and trauma to the neck or back. She was known to have hypertension for more than 5 years and has good compliance with her medications (Amlodipine and Lisinopril). She had earlier presented to a peripheral clinic where she was found to have low blood pressure (hypotension) for which she was resuscitated with intravenous fluids (normal saline) and discharged home on oral antibiotics and anti-malarials. She was later brought in to our hospital because of the sudden onset ascending leg weakness and urinary retention a day after. Examination revealed an apprehensive middle-aged patient with apparently normal general physical findings, mild elevation of the supine BP (130/100 mmHg) and a loud aortic component of the 2nd heart sound on precordial examination. Motor system examination showed normal muscle bulk, bilateral lower limb hypotonia and hyporeflexia with muscle power of 0/5 using the Medical Research Council (MRC) scale. Sensory modalities for touch, cold temperature perception (using tip therm ®) vibration and position sense were normal. However, coordination in the legs could not be elicited because of the weakness. An initial working diagnosis of Guillain-Barre syndrome (post-enteritic) was made, with differential diagnoses of transverse myelitis, compressive myelopathy (likely from disc prolapse or Pott's disease) and anterior spinal cord infarction.

However, 24 hours after admission bilateral spastic tone with hyperreflexia and extensor plantar response was noticed in both lower limbs. Additionally, sensations of touch, and cold temperature perception were also lost with preservation of vibration, position sense and persistence of sphincteric disturbance. Urgent bedside investigation results done revealed: Sodium 140 mmol/L, Potassium 3.8 mmol/L, Bicarbonate 22 mmol/L, Chloride 104 mmol/L, Urea 5.3 mmol/L and Creatinine 51 umol/L. Full blood count with ESR, fasting lipid profile and serum uric acid assays were essentially within normal reference limits. A multi-planar (parasagittal, axial and coronal views), multi-sequence thoracic spine MRI (using 1.5 tesla scanner) revealed thinning of the spinal cord extending from T1 to T9 levels (Figures 1 and 2) with associated widening of the thecal sac suggestive of anterior spinal cord infarction with no signal changes seen within the substance (Figure 3). The patient was managed for anterior spinal artery thrombosis with anticoagulants (low molecular weight heparin - subcutaneous enoxaparin), her routine hypotensive agents and passive physiotherapy. She was subsequently discharged home after 13 days on admission with remarkable improvement in her general well-being, although the muscle power was still 0/5 in the lower limbs and only the dorsal column sensory modalities were preserved. She was advised to come for follow-up visits in the adult Neurology clinic and to continue with the physiotherapy.

Discussion

Ischemia typically affects the anterior spinal artery and patients present with a history of sudden onset flaccid paralysis, which may be either paraplegia or quadriplegia depending on the level of ischemia. In addition, patients may also present with autonomic dysfunction, loss of pain and temperature sensation.[6] The site most commonly affected is the lower thoracolumbar segments with sensory level frequently located at T10.[7] The diagnostic modality of choice is MRI, which may show pencil-like hyper-intense signal on sagittal cuts of T_2 weighted images and FLAIR.[8] The aim of treatment is to improve blood flow via collaterals. Treatment is usually supportive with IV thrombolytics and rapid favorable outcome in 2 out of 4 patients with spinal cord infarction.[9]

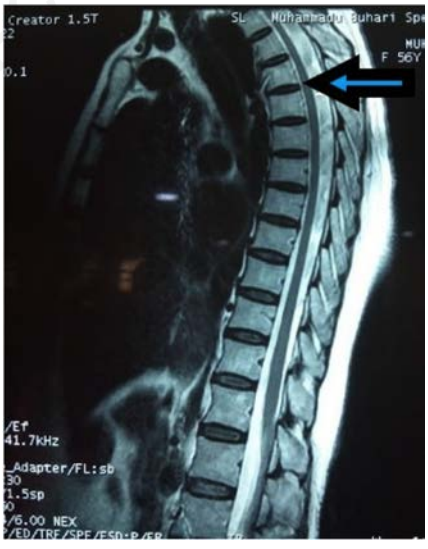

Figure 1. The multi-planar (parasagittal) multi-sequence thoracic spine T2-weighted MRI (using 1.5 tesla scanner) revealing thinning of the spinal cord extending from T1 to T9 levels.

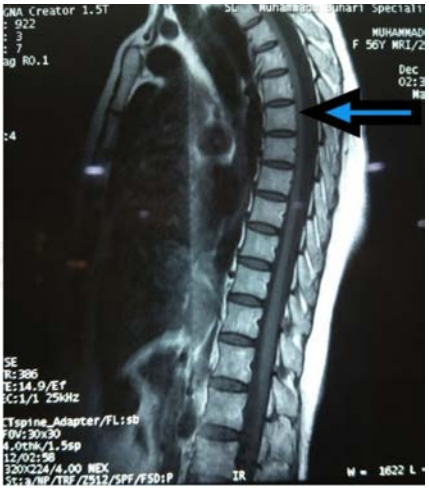

Figure 2. The parasagittal thoracic spine T1-weighted MRI (using 1.5 tesla scanner) revealing thinning of the spinal cord extending from T1 to T9 levels.

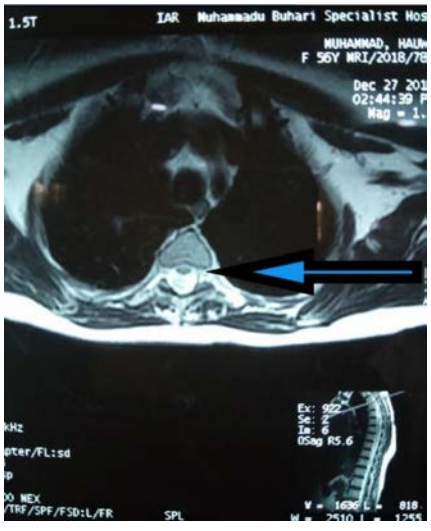

Figure 3. The axial sequence thoracic spine T2-weighted MRI (using 1.5 tesla scanner) revealing associated widening of the thecal sac suggestive of anterior spinal cord infarction with no signal changes seen within the substance.

Prognosis depends on the severity of the neurologic deficit at nadir. Other factors that worsen prognosis include female sex, older age and more extensive ischemic changes on MRI.[10]

At about 5 months of follow-up, she was noticed to have started walking with some support from caregivers and later with a walking frame, and by 12 months of her clinic visit she could walk without any support. During her last clinic visit Enoxaparin was stopped and low dose Aspirin (antiplatelet agent) was added to her routine antihypertensive medications.

Conclusions

Our case report demonstrated a rare case of ASCI unusually mimicking transverse myelitis, which may present with early diagnostic and management challenges especially in our low-resource settings.

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