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HYPOTENSION-INDUCED TRANSVERSE SPINAL CORD ISCHEMIA

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ABSTRACT

Background: Spinal cord infarction is rare, with spontaneous, non-traumatic cases being extremely uncommon. Severe atherosclerosis may predispose to spinal ischemia during episodes of systemic hypotension, particularly in mid-thoracic watershed zones.

Case report: We present a case of a 56-year-old woman with schizophrenia, a history of heavy smoking, and chronic use of nonsteroidal anti-inflammatory drugs. She presented with ischemic lower limbs and hypotension, following a self-inflicted neck wound. Computed tomography angiography revealed extensive chronic atherosclerotic disease of the distal aorta and iliac arteries with collateral circulation but no acute occlusion. After surgical control of bleeding and stabilization, she was found to have flaccid paraplegia and sensory loss below the TH10 level. Magnetic resonance imaging confirmed acute spinal cord infarction with well-demarcated ischemia, that was treated conservatively. Despite prompt hemodynamic stabilization, her neurological deficits did not resolve.

Conclusion: This case highlights spontaneous spinal cord infarction as a rare but serious complication of transient hypotension in patients with severe atherosclerosis. Prompt recognition is essential, though therapeutic options remain limited.

KEYWORDS

Spinal cord ischemia, hypotension, atherosclerosis, artery of Adamkiewicz

LEARNING POINTS

- Atherosclerosis is the leading cause of non-traumatic spinal cord ischemia.
- Diagnostic value of magnetic resonance imaging in early diagnosis.
- Treatment is mainly supportive with mean arterial pressure elevation.

INTRODUCTION

Spinal cord ischemia is a rare clinical condition, accounting for less than 1% of all strokes, and is most commonly associated with thoracoabdominal aortic surgery or aortic dissection, where segmental spinal cord blood flow is disrupted. In contrast, spontaneous, non-traumatic spinal

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cord infarction is exceedingly uncommon, particularly in the form of complete transverse lesion, due to the extensive collateral blood supply[1]. However, recent studies have identified severe atherosclerosis as an important underlying cause in spontaneous cases, where even short episodes of systemic hypotension may critically reduce perfusion – especially in vulnerable watershed zones of the spinal cord such as the mid-thoracic region, typically supplied by the artery of Adamkiewicz[2].

We describe a rare case of transverse spinal cord ischemia precipitated by hypotension in a patient with previously undiagnosed extensive atherosclerotic disease. The case illustrates the diagnostic complexity and limited treatment options associated with this condition.

CASE DESCRIPTION

A 56-year-old female with a medical history of severe schizophrenia presented to the emergency department with a self-inflicted bleeding neck wound. The patient was a heavy smoker with chronic obstructive pulmonary disease and regularly used nonsteroidal anti-inflammatory drugs (NSAIDs) and paracetamol for chronic hip pain. Her chronic medications included acetylsalicylic acid, bisoprolol, levothyroxine and multiple psychiatric drugs. Despite her hip-related limitations, she had been independently mobile prior to the injury. After inflicting the injury at home, she was admitted to the emergency department resuscitation area with hypotension (blood pressure, BP 89/54 mmHg) and active bleeding from the neck wound. She remained alert and reported bilateral leg numbness; her lower limbs appeared ischemic and showed no motor response to stimuli. She was intubated for diagnostic evaluation, and surgical control of bleeding from the right internal jugular vein was successfully performed.

Following fluid resuscitation and elevation of BP, lower limb perfusion improved and she remained normotensive. Initial tests showed a lactate level of 14 mmol/l, haemoglobin of 120 g/l, creatinine of 140 µmol/l, and low serum myoglobin. Computed tomography angiography imaging revealed advanced chronic atherosclerotic disease, including thrombi causing up to 50% narrowing of the distal thoracoabdominal aorta, chronic occlusion of the left common iliac artery (AIC) and subtotal occlusion of the right AIC, with collateral distal circulation bilaterally *(Fig. 1)*. In view of the patient's clinical improvement and the lack of acute vascular lesions on imaging, a conservative treatment approach was advised. She was transferred to the intensive care unit, still sedated, hemodynamically stable, with normalized lactate levels and no further bleeding from the neck wound.

Sedation was discontinued six hours after the emergency department visit; the patient regained consciousness and was extubated. Neurological examination revealed flaccid paralysis of the lower limbs with absent reflexes and flexion plantar response. Sensory testing revealed symmetric hypoesthesia, paresthesias, and absent sensation to temperature, pinprick, vibration, and proprioception in the lower limbs. A clinical spinal cord injury level was identified at Th10. Thoracic spine magnetic resonance imaging (MRI) demonstrated diffuse T2/STIR hyperintensity and restricted diffusion in the central spinal cord extending from Th9 distally, consistent with acute spinal cord ischemia *(Fig. 2)*. Despite maintaining an elevated mean arterial pressure over the subsequent hours, no clinical improvement was observed. The patient was transferred to the ear, nose, and throat department and after 2 days to the psychiatric ward for further evaluation.

DISCUSSION

The spinal cord is generally protected from ischemia by a rich collateral vascular network, composed of the anterior and paired posterior spinal arteries, further augmented by segmental radicular arteries – most notably the artery of Adamkiewicz, which typically originates between T8 and L2[1]. However, certain regions, particularly the mid-thoracic segment (T4–T8), are considered hemodynamic "watershed" zones. These regions are particularly vulnerable during episodes of systemic hypoperfusion, especially when the anterior spinal artery is anatomically discontinuous or spinal cord perfusion is compromised by underlying atherosclerotic disease[2,3].

A recent pooled analysis reported a high prevalence of vascular risk factors in cases of spontaneous spinal cord infarction, with hypertension (40%), smoking (30%), dyslipidaemia (29%), and diabetes (16%) being the most common[4]. These findings support the notion that atherosclerosis is a primary underlying mechanism in spontaneous spinal cord infarction. Multiple vascular risk factors were identified in our patient with previously undiagnosed atherosclerotic disease, including heavy smoking, chronic NSAID use and

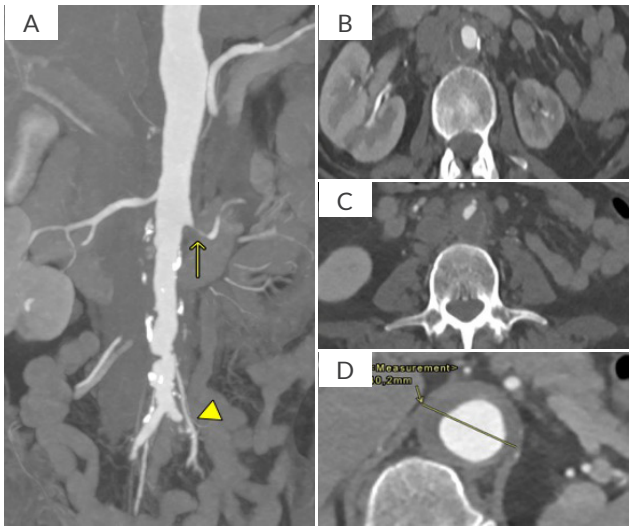

Figure 1. Computed tomography angiography of the A-D) abdominal aorta demonstrates severe eccentric noncalcified "soft" and mildly calcified atheromatous plaque; B,C) The lumen of the infrarenal aorta is unevenly narrowed. We can also appreciate D) mild dilatation of descending thoracoabdominal aorta, severe stenosis of the left renal artery with atrophy of the left kidney (arrow ←) and chronic occlusion of left common iliac artery (arrow ◄).

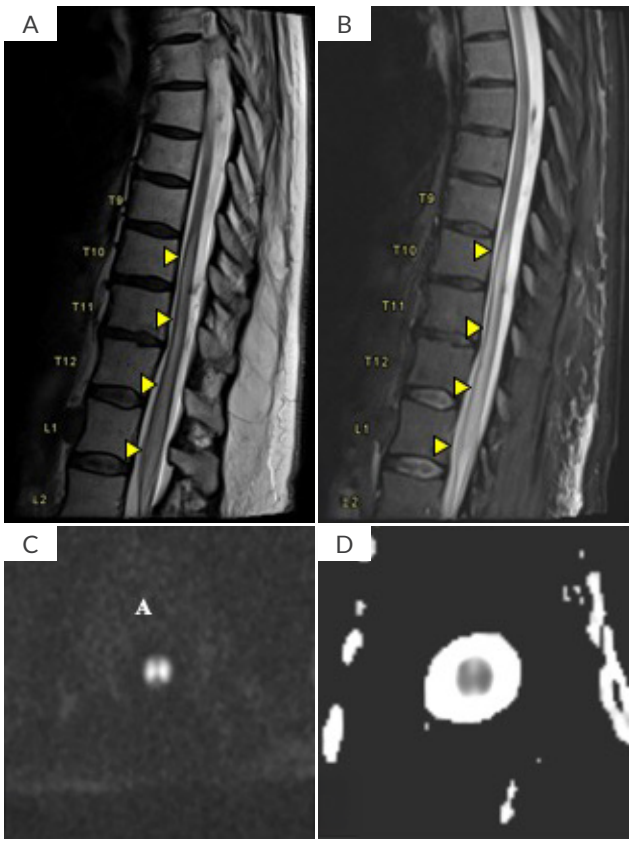

Figure 2. Magnetic resonance imaging of A,B) the spinal cord demonstrates long segment oedema of the distal third of the spinal cord, best seen on sagittal T2-weighted turbo spin echo sequence and on sagittal turbo inversion recovery magnitude sequence (arrow ◄) with "H-shaped" high signal intensity on diffusion weighted images and low values on an apparent diffusion coefficient map ("diffusion restriction") which is consistent with acute ischemic infarction of gray matter of the spinal cord.

on further evaluation symptoms suggestive of peripheral arterial disease. Computed tomography angiography confirmed advanced chronic atherosclerotic disease with overt collateral circulation.

Hypoperfusion during an acute hypotensive episode secondary to blood loss led to ischemia and subsequent spinal cord infarction. Clinically, the presentation was consistent with a lesion at the T10 level, characterized by sudden-onset flaccid paraplegia and sensory loss. MRI findings supported the diagnosis, revealing diffuse T2-weighted/short-tau inversion recovery hyperintensity and restricted diffusion in the central thoracic spinal cord—hallmarks of acute ischemic injury[3,5]. Diffusion-weighted imaging is particularly useful for early identification of spinal cord infarction, often demonstrating restricted diffusion within hours of onset, correlating with cytotoxic oedema[3]. Due to the rarity of spinal cord infarction and the limited sensitivity of imaging during the hyperacute phase, diagnosis is often delayed or initially missed in the emergency setting[3,5].

Despite early diagnosis following sedation withdrawal in our case, treatment options for spinal cord ischemia remain limited and largely supportive, with mean arterial pressure elevation one of the main goals. High-dose corticosteroids are typically reserved for inflammatory or traumatic aetiologies and have not shown benefit in spinal cord infarction[6]. Antiplatelet therapy is most commonly initiated after confirmation of the diagnosis, whereas the efficacy and safety of anticoagulation or thrombolysis remain uncertain due to a lack of supporting data[3,4]. In this case, the patient was managed conservatively with low-dose acetylsalicylic acid, as the lesion was well-demarcated and the risk of haemorrhagic transformation was deemed to outweigh the potential benefits of more aggressive intervention. Additionally, the therapeutic window for thrombolysis had already elapsed.

Despite rapid restoration of hemodynamic stability, neurological deficits persisted – likely due to the extent and severity of the initial ischemic insult with underlying severe atherosclerotic disease. Unlike partial anterior spinal artery syndromes, which may spare some function and offer recovery potential, transverse infarctions often result in irreversible damage (e.g. paralysis) with more severe initial presentation associated with poorer prognosis[7]. Novy et al. noted better outcomes in patients with incomplete lesions or segmental ischemia, whereas those with complete infarcts often had persistent deficits[5].

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