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Single Case – General Neurology

A Rare Case of Cardioembolic Spinal Stroke in a Young Female: Case Report

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Keywords
Spinal cord infarction · Cardioembolic stroke · Case report

Abstract
Spinal cord infarction (SCI) is a rare neurovascular disorder often presented with acute spinal cord syndrome. The diagnosis is generally made clinically, with appropriate neuroimaging to confirm the diagnosis and exclude other causes. We present an unusual case of a 48-year-old woman with no relevant past medical history, admitted with acute paraparesis and a spinal cord infarct on magnetic resonance imaging. A thorough investigation revealed asymptomatic unknown heart failure secondary to hypertrophic cardiomyopathy, suggestive of a cardioembolic etiology. The patient was treated with anticoagulation and improved significantly with physical rehabilitation.

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Introduction

Spinal cord infarction (SCI) accounts for 0.3–2% of all vascular neurological pathology [1, 2]. It is caused by acute spinal cord blood supply disruption, resulting in ischemia, infarction, and spinal cord dysfunction. The clinical presentation is defined by the vascular territory involved, the most common of which is an anterior spinal artery syndrome [2]. Lower thoracolumbar spinal strokes are more common than upper cervical ones. These patients usually present with less severe neurological deficits compared to those with upper infarcts; however, they tend to improve more gradually [1].

The most common cause of spinal stroke is iatrogenic. Among 91,212 patients who had repair of an aortic aneurysm or dissection described by Gialdini et al. [3], SCI occurred in approximately 1 in 130 patients undergoing aortic dissection or ruptured aortic aneurysm

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repair and in 1 in 600 patients undergoing unruptured aortic aneurysm repair. However, in cases of spontaneous spinal strokes, identifying the mechanism can be challenging. One study investigating 27 patients found no identifiable cause in 74% of patients, and the most common risk factors were disk prolapse or herniation [4]. Cardioembolic spinal stroke is extremely rare. The largest cohort study evaluating 133 patients with a spontaneous SCI found that 68% were idiopathic with atherosclerotic risk factors, followed by 14% secondary to fibrocartilaginous embolism, 5% aortic dissection, 4% hypercoagulability, 3% vertebral artery dissection, 2% systemic hypotension, 2% vasculitis, and only 2% cardioembolic [5]. Another study of 55 consecutive patients with spinal cord ischemia in a 19.8-year period found no cardioembolic mechanism at all. Mechanisms of infarcts were arteriosclerosis of the aorta and vertebral arteries in 23.6% of patients, aortic surgery or interventional aneurysm repair in 11%, aortic and vertebral artery dissection in 11%, and etiology remained unclear in 23.6% [6].

Computed tomography and digital subtraction angiography are not diagnostic for SCI diagnosis. However, in older patients, the visualization of atheromatosis or vascular lesions in the aorta or lumbar arteries can support the diagnosis of vascular mechanisms. Magnetic resonance imaging (MRI) is the preferred imaging modality for diagnosing ischemia and excluding other causes. Demonstrations of abnormally increased T2 signal and restricted diffusion in the spinal cord are diagnostic for acute ischemia. Contrast media can also aid in the diagnosis at the acute stage [7, 8].

Case Report

A 48-year-old patient with a history of depressive disorder and no known vascular risk factors presented with acute weakness and pain of the lower limbs associated with lower back pain. She had no motor symptoms of the upper limbs or any sensory disturbances. There was also no history of trauma, fever, or substance abuse. The motor deficit evolved and reached its peak within 24 h.

On neurological examination, she was fully conscious and alert, with no neck stiffness and no cranial nerves or upper limb deficits. The lower limbs had flaccid tone with distal 1–2/5 more than proximal 3–4/5 weakness. Deep tendon reflexes were brisk on the left patellar and hypoactive on both Achilles. Plantar responses were flexor. Sensory examination showed decreased pinprick sensation on both feet with normal temperature and position sense and no sensory level. The rectal tone was decreased, and she had urinary incontinence while being evaluated in the emergency room. Soon after that, a catheter was inserted due to urinary retention. A spine MRI scan, performed about 20 h after symptom onset, revealed an abnormal T2 signal in the central spinal cord at T12 and L1 vertebrae levels. The lesion was bilateral, symmetric, and involved predominantly gray matter in an “owl eyes” pattern. Diffusion restriction within the spinal cord was demonstrated at the level of the lesion (shown in Fig. 1). The findings were compatible with acute ischemia in the distribution of the artery of Adamkiewicz. Brain and cervical spine MRI did not show additional lesions, supporting infectious or inflammatory etiology. Cerebrospinal fluid was completely normal with no pleocytosis or elevated protein level. Computed tomography angiography excluded any vascular lesions of the brain and neck arteries or aorta. A transthoracic echocardiogram showed moderate left ventricular dysfunction with an ejection fraction of 38% and left ventricle regional wall motion abnormality. Cardiac CT excluded a thrombus or evidence of coronary disease. Cardiac MRI demonstrated non-obstructive hypertrophic cardiomyopathy. Laboratory results showed no signs of infectious or inflammatory disease, and no thrombophilia was found: CRP and ESR were not elevated; syphilis screening, HBV, HCV, HIV, EBV,

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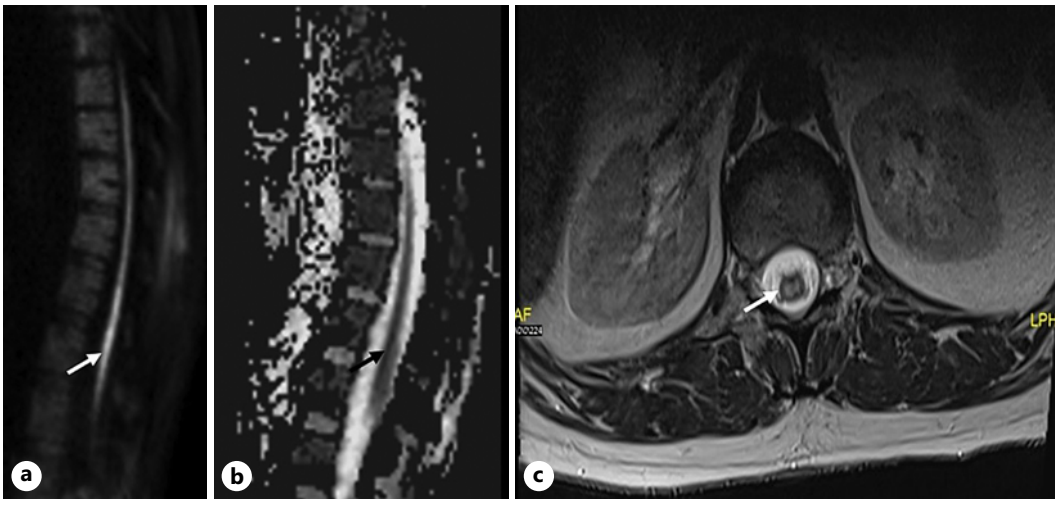

Fig. 1. Spine MRI demonstrates an acute infarct at T12 and L1 vertebrae levels: sagittal image shows a lesion with restricted diffusion demonstrated by a high signal on DWI (**a**), confirmed by low ADC values (**b**); axial image demonstrates bilaterally symmetric pathologically increased T2WI signal (**c**), which involves predominantly gray matter. This pattern is also known as an "owl-eyes" sign or a "snake-eyes" sign. MRI, magnetic resonance imaging; DWI, diffusion-weighted imaging; ADC, apparent diffusion coefficient; T2WI, T2-weighted imaging.

and CMV were negative; ANA, ANCA, antiphospholipid antibodies, lupus anticoagulant, C3, and C4 were negative. Genetic testing for Fabry disease was also negative.

The patient was initially treated with antiplatelets. However, following a finding of cardiac condition and the absence of atherosclerotic disease or vascular risk factors, a multidisciplinary team concluded a cardioembolic etiology was the presumptive diagnosis at that point, and empirical anticoagulation treatment was started until further investigation. Full-dose low-molecular-weight heparin and appropriate treatment for heart failure were initiated. An implantable loop recorder was inserted in search of cardiac arrhythmias. At follow-up a month later, a short asymptomatic atrial flutter was demonstrated, and the patient continued treatment with Apixaban. The patient's functional status improved, and she regained independence with intense multidisciplinary physical rehabilitation. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000531779).

Discussion

SCI is a rare and often devastating neurovascular disorder. We present a case of a young female patient with acute paraparesis due to a cardioembolic spinal stroke. Spontaneous SCI accounts for only 0.9–1% of total stroke incidents in the USA [9], and among these patients, a cardioembolic mechanism is relatively rare, described in up to 2% of patients [5].

Maximal neurological deficit is reached within 12 h in 50% of patients, and within 72 h in most patients, as same as occurred in our patient [2]. Neurological examination on admission was atypical as deep tendon reflexes were brisk on the left patellar and hypoactive on both achilles, and the sensory deficit was more suggestive of peripheral neuropathy with no sensory level. Absence of sensory level, which may be associated with a better outcome, was described in 25 out of 115 patients with SCI in the study by Robertson et al. [10]. Motor deficits without sensory abnormalities or sphincter dysfunction can be secondary to

incomplete anterior spinal artery syndrome [2]. Hypoactive reflexes on presentation may be secondary to a spinal shock state, a phenomenon associated with various acute spinal cord injuries including a vascular one [11]. Back pain at the level of the lesion was a prominent feature in our case. It has been present in 72% of the 36 patients described by Kumral et al. [12].

Thoracic spine MRI findings were compatible with the diagnosis of acute infarction in the distribution of the artery of Adamkiewicz, with a classic owl-eyes pattern. Infarction of the vertebral body, occasionally seen in spinal cord ischemia, was not present in our case [13, 14]. Advanced cardiac evaluation, including a cardiac MRI and an implantable loop recorder, demonstrated asymptomatic heart failure secondary to hypertrophic cardiomyopathy and atrial flutter, suggestive of a cardiac embolus.

The patient was treated with anticoagulation and regained independence with physical rehabilitation. Evidence for thrombolysis and anticoagulation in SCI is limited [5, 15–18]. Although few studies report thrombolysis for spinal stroke patients without harm, there is no evidence for efficacy. Our patient was not eligible for thrombolysis, and despite the lack of quality evidence for empirical anticoagulation, treatment was initiated even before finalizing the investigation. In a meta-analysis of 125 case reports and a series of patients with non-iatrogenic spinal cord ischemia, improved outcome, such as in the case presented, were found to be more common in idiopathic causes or aortic vascular pathologies compared with trauma or systemic/chronic causes [15].

This case describes a rare cause of an uncommon neurovascular disorder. It highlights the need for a thorough and urgent investigation to rule out SCI in patients presenting with acute paraparesis, even in the absence of typical signs on neurological examination.

Statement of Ethics

Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images. Ethical approval is not required for this study in accordance with local guidelines.

Conflict of Interest Statement

None of the authors have any financial disclosures or conflicts of interests.

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Author Contributions

Rom Mendel conceptualized and designed the work including data acquisition and drafting of the manuscript. Irina Tsirkin took part in the drafting of the manuscript. Salo Haratz and Eugene Soikher took part in manuscript revision. All authors reviewed and edited the manuscript, approved the final version, and took full responsibility for the data, the analyses, and the interpretation.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material. Further inquiries can be directed to the corresponding author.

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