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Spinal Cord Ischemia Secondary to Atrial Fibrillation: A Case Report

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Abstract **Case Report**

Spinal cord ischemia is exceptionally uncommon, accounting for 0.3–1% of all strokes. Here, we reported the case of a 64-year-old patient with spinal cord ischemia secondary to atrial fibrillation. Spinal magnetic resonance imaging showed increased intramedullary signal intensity at the T9 level in T2-weighted. An electrocardiogram showed atrial fibrillation. The patient was given corticosteroids and antiplatelet agent therapy. After 8 weeks of rehabilitation, the patient was able to walk with the support of a medical walker.

Keywords: Spinal cord ischemia, Atrial fibrillation, Paraplegia.

INTRODUCTION

Spinal cord ischemia is exceptionally uncommon, accounting for 0.3–1% of all strokes. The relationship between spinal cord ischemia and vascular surgical interventions, like aortic aneurism surgery, is well-established in adults; however spontaneous spinal cord ischemia is extremely rare [1-3]. Here we describe a case of spinal cord ischemia secondary to atrial fibrillation in a 64-year man.

CASE PRESENTATION

A previously healthy 64-year-old man was admitted to the emergency department of our hospital, for a sudden onset of lower limbs weakness and urinary retention. Other symptoms were denied, such as fever, infectious symptoms, or numbness. No significant medical or family histories were found.

On admission, his vital signs were normal and he was afebrile. The neurologic examination showed a flaccid paraparesis with absent deep tendon reflexes.

Baseline blood tests and urinalysis were unremarkable. An electrocardiogram showed atrial fibrillation. Spinal magnetic resonance imaging (MRI) was performed and revealed increased intramedullary signal intensity at the T9 level in T2-weighted images (Figure 1).

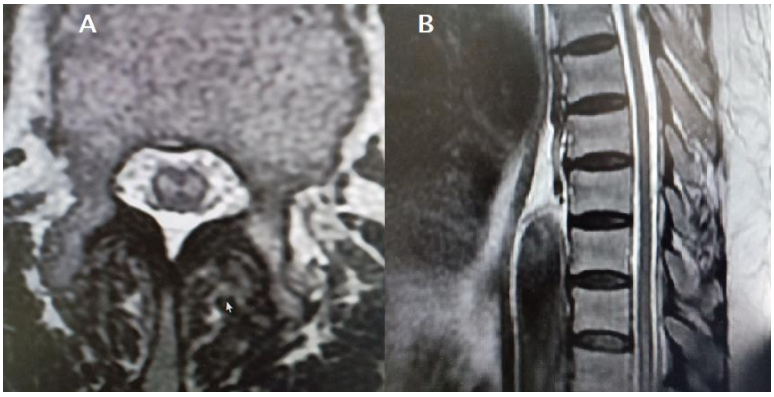

Fig-1: Spinal MRI showed increased intramedullary signal intensity at the T9 level in T2-weighted (A) axial, and (B) sagittal images

We diagnosed the patient with spinal cord ischemia secondary to atrial fibrillation. The patient was given corticosteroids and antiplatelet agent therapy (cardioaspirine and low-molecular-weight heparin) and supportive therapy while urinary retention was treated by placement of a urinary catheter into the bladder. Functional exercise was added during the following days. After 8 weeks of rehabilitation, the patient was able to walk with the support of a medical walker.

DISCUSSION

Spinal cord ischemia is a rare entity whose diagnosis is challenging due to the great variability of clinical presentations and its broad differential diagnosis [4, 5]. There are numerous etiologies implicated in spinal cord infarctions including, in adults, aortic aneurysms, venous thromboembolism, coagulopathies, and all surgical procedures performed on the aorta. Additional, although less specific, factors could be atherosclerosis, arterial hypertension, diabetes, and other cardiac diseases [6].

The clinical picture primarily depends on where the occlusion is, which may accompany back pain radiating caudally and loss of sphincter control with hesitancy or inability to void or defecate. The acute stage may last for several days and is marked by initial flaccid paralysis, depression, or absence of reflexes with a sensory level distal to the lesion where pain and temperature are not perceived. Over time there is increased muscle tone and reflexes become hyperactive and Babinski reflexes positive. Proprioception and vibration are usually intact as they are supplied by the dorsal columns which lie medially whereas the spinothalamic tract lies more laterally. In contrast, sensation to touch is a subjective finding during the examination as light touch and well-localised touch is carried by spinothalamic and dorsal columns [7].

MRI is the most useful l tool for diagnosis of spinal cord infarction. Most infarctions appear as pencil-like hyperintensities on T2-weighted sequences. When lesions exclusively affect the grey matter, they present an owl-eyes pattern on axial T2-weighted sequences. Some cases may be associated with hemorrhagic transformation, with hyperintense lesions on T1-weighted sequences; other cases may present infarction of the adjacent vertebral body. Gadolinium uptake may also be observed. However, a considerable percentage of patients with spinal cord infarction show no MRI alterations, particularly when the study is performed in the early stages of the event. Diffusion-weighted sequences are more sensitive in these cases [4, 8].

Treatment focuses on risk factors and rehabilitation. As the most causative agent is likely to be atherosclerosis, treatment with antiplatelet therapy is fundamental following spinal cord infarction. If the source is judged to be embolic, anticoagulation drugs can be started. Some studies have looked at corticosteroid therapy administered within a short time following spinal cord infarction due to protective effects on cell function and reducing oxidative stress following ischaemic injury [5, 7].

Spinal cord ischemia presents elevated morbidity and mortality rates. Poor prognostic factors include severe impairment at presentation (complete paralysis, bladder dysfunction, or proprioceptive deficits), female gender, advanced age, and lack of improvement in the first 24 h [4, 5].

CONCLUSION

Spinal cord ischemia is a rare cause of paraplegia with a wide array of aetiological factors. MRI is an essential tool for diagnosis. Long-term functional prognosis is poor and depends on the patient's baseline characteristics and the form of presentation. Yet, patients who regain some function during hospitalization can continue to improve for a long time with adequate rehabilitation.

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