

Spinal Cord Stroke: Report of 2 Cases

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Abstract

Spinal cord infarction (SCI) is much rarer than cerebral stroke, constituting 1% to 2% of all neurological vascular emergencies; however, it is a disabling disease and its early recognition is important as it may signify serious aortic conditions. Characterized by abrupt onset of neck or back pain and neurologic deterioration, arriving a definite diagnostic can be challenging for some physicians. Anterior spinal artery syndrome is the most frequent type of presentation. Even with early correct diagnosis and active therapy, some cases could evolve to critical situation and permanent disability. We introduce two cases of acute spinal cord stroke with clinical features and acquirement of simple imaging tools to support diagnosis.

Keywords: Spinal Cord Stroke; Infarct; Acute; Aortic Disease; Misdiagnosis

Introduction

Spinal cord stroke is considerably less common than cerebrovascular disease, merely 1% to 2% of every ischemic stroke and its early diagnostic is demanding [1]. Unlike cerebral infarction, little is known about the causes and risk factors associated with spinal cord infarction. Most SCI patients had serious, monophasic presentation, coming to nadir in < 24h [2]. Appropriate imaging studies are applied to rule out lesions which may require neurosurgical intervention. Treatment is usually symptomatic, and even though many SCI patients have low functional levels early in the disease course, more of these patients are discharged home than patients with cerebral infarction with comparable function [2]. In this article two cases of spinal cord stroke are described with changing evolution despite a complete work up.

Case Report

Case 1

A 72-year-old man presented to the emergency unit of a leading district clinic with sudden onset leg weakness posterior to back pain after a bus trip of 12 hours. He had a background of laryngeal cancer 11 years before, hypothyroidism, AF, anticoagulated. He described initially an acute pain in his lower back which subsequently developed into acute leg weakness. No changes in speech and facial or upper limb weakness were confirmed. He lived with his wife and was independently mobile. He had a blood pressure of 146/76 mm Hg, and his heart rate was at 75 bpm. Lower limb exploration demonstrated a flaccid paraparesis (2/5) with absent deep tendon reflexes. Pinprick and temperature were compromised below T11; touch, vibration and proprioception were patchy defective. A digital rectal assessment exposed usual perianal sensation along with gone anal tone. He had a tangible bladder and was consequently catheterized.

Baseline blood tests and urinalysis were unremarkable, others laboratory testing like serologic screening, vitamin B12, erythrocyte sedimentation rate, copper levels, serum protein electrophoresis, angiotensin-converting enzyme, and prothrombotic states were negative. ECG showed AF, and chest radiograph showed minimal left laminar pleural effusion. MRI of his thoracic-lumbar spine (Figure 1 and 2) was reported as normal and later, in a peer reviewed, as possible B12 vitamin deficit. Thorax, abdomen, pelvis CT angio tomography found small areas of parietal thrombosis in ascending thoracic aorta, abdominal aorta showed a small aneurysmal dilation of approximately 4.9 cm with preserved flow, and it was possible to identify a small aneurysmal dilation of approximately 30 mm of the left common iliac artery (Figure 3). After discussed with internal medicine and neurosurgery team, we agreed on spine infarct according to clinic presentation and images obtained.



Figure 1: Characteristic patterns of T2-hyperintense signal seen in SCI (A and B).

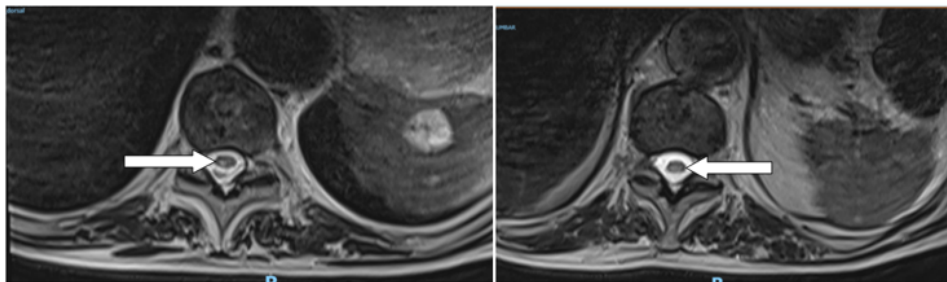


Figure 2: Axial MRI shows an axial view of the same infarct.

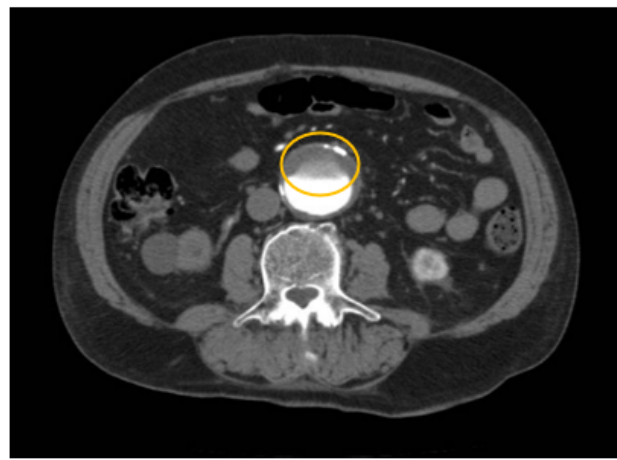


Figure 3: Angio CT revealed marked atherosclerotic plaques in the abdominal aorta with a mural thrombus.

Case 2

A 79-year-old male with a background of chronic obstructive lung disease, smoker, dyslipidemia, peripheral artery disease, presented to the emergency department with back pain during a shower that immediately leave him with impossibility to walk. On neurological examination, he showed severe paraparesis, sensory level and acute urinary retention requiring catheterization. Deep tendon reflexes were not present in lower limbs and no plantar responses were found. Abdominal reflexes were absent. Temperature, light touch, pinprick, and position sense were decreased in the trunk and lower limbs circumferentially. No modifications on spinal cord images were found after a gadolinium 1.5T spinal cord MRI carry out hours following symptom beginning. He denied any exposure to radiation, toxic, recent traumatic injuries, infections, vaccinations, scuba diving or Valsalva maneuver. ECG showed normal sinus rhythm. An aortic computed tomography angiography showed no sign of acute pathology. Brain MRI was unremarkable. CSF (cerebral spinal fluid) analysis returned normal. Other unvaried blood tests included cancer and systemic vasculitis. Because perseverance of clinical manifestations we decided to repeat MRI after 3 days and could confirmed the spinal ischemia (Figure 4). During the hospital stay, he suffered an acute myocardial infarction due to dissection of the thoracic/abdominal aorta. He underwent surgery to repair dissection, and days later discharged. He finally passed away one year later because renal failure and other complications.

Discussion

SCI is an infrequent disorder, just 1% to 2% of the entire ischemic strokes, raising to 4% to 33% amongst patients who experience thoracoabdominal aortic surgery [3-5]. A transient back or neck pain usually predominates the clinical presentation following of limb weakness. Abdominal aortic aneurysm with intramural thrombosis is not a common presentation also requires a sharp look from clinicians, to avoid an incomplete recognition. Arteries in the anterior territory are less numerous predisposing to a greater possibility of spinal cord infarcts in that region compared to the posterior one. Many studies have found that Adam Kiewicz artery start in 89% between T8 and L1, being the most dominant anterior radiculomedullary artery [5]. As soon as a spinal cord injury is suspected, the initial test that must be complete is a spinal MRI. Despite this, a normal image in the first few hours does not eliminate the diagnosis and etiology, as happened in our second case. We encourage use of simple and efficient tools to study patients, like CT angiography which also help detect penetrating aortic ulcers and intramural hematoma. In our two cases, the cause was suspected, one was confirmed with MRI and CT and the other despite extensive work up did not reach a certain diagnosis after clinical complications [6,7].

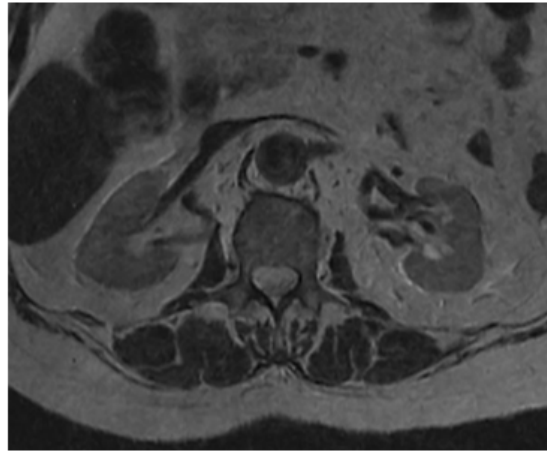


Figure 4: An axial view of the infarct with increased signal within gray matter.

Conclusion

Spinal cord infarction is an unusual entity. Aortic dissection/surgical procedures involving the aorta, are common etiopathogenic mechanisms. Only a few cases of unruptured aortic aneurysm generating anterior cord syndromes due to mural thrombi had been documented previously. In the presentation of these two cases, we can see the diagnostic complexity of spinal cord infarction, the high clinical suspicion that must be taken even without conclusive images, and the variable evolution despite reaching a correct diagnosis.

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