Atypical Spinal Cord Infarction: A Prolonged and Stuttering Course for Six Days.

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Abstract

Purpose: Spinal infarction is a rare condition and usually presents with a sudden or acute course. A prolonged course is rare and may mimic the presentation of inflammatory myelitis. Here we present a case of atypical spinal cord infarction with a stuttering course for six days..

Case report: A 47-year-old male presented initially with symptoms of sudden onset, limb pain. Sudden chest pain radiating to the back, occurred three days later. Sudden urinary retention and quadriparesis were presented after another three days. The diagnosis of spinal cord infarction was made through diffusion restriction in spinal magnetic resonance imaging.

Conclusion: A prolonged course of spinal cord infarction is relatively uncommon but a stepwise and stuttering course may provide clues. Diffusion restriction in magnetic resonance imaging also may be helpful. The diagnosis of spinal cord infarction should always be kept in mind.

Keywords: Ischemic myelopathy; Spinal cord infarction; Spinal cord stroke; Vascular myelopathy.

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INTRODUCTION

Spinal cord infarction (SCI) is a rare condition that leads to acute myelopathy and high morbidity. It presents most often as an abrupt onset anterior spinal artery syndrome[1]. The stepwise or stuttering course of this disease has been reported but is relatively rare. When presented initially, SCI may mimic inflammatory myelitis (IM) or other lesions in the spinal cord and often creates a diagnostic dilemma. Recent studies have shown that 14% to 16% of patients referred for the evaluation of IM are ultimately diagnosed as having SCI[2,3]. Magnetic resonance imaging (MRI) is an excellent tool by which to confirm the presence, location, and extension of the infarction, but the diagnosis depends on the timing and quality of the images[4]. Here, we present a rare case of SCI. Its course mimics IM and was diagnosed finally as SCI.

CASE REPORT

A 47-year-old male taxi driver presented initially with symptoms of sudden-onset bilateral sharp pain in the upper limbs occurring six days before. Three days later,

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sudden chest pain that radiated to the back occurred. He visited the first hospital at that time, and both chest and cervical computed tomography (CT) revealed no evidence of aortic dissection. The spinal MRI showed C6-T1 spinal cord hyperintensity on T2- weighted imaging (Figure 1). The cerebrospinal fluid examination revealed total protein: 68.2 mg/dl (normal range: 20-40 mg/dl), glucose: 108 mg/dl (serum glucose: 180 mg/dl.), and WBC: 0/HPF. He was discharged against the doctor's advice. Three days later, he presented with symptoms of acute urinary retention accompanied by quadriparesis and visited our emergency department. His medical history was significant for diabetes mellitus. He was a current heavy smoker and

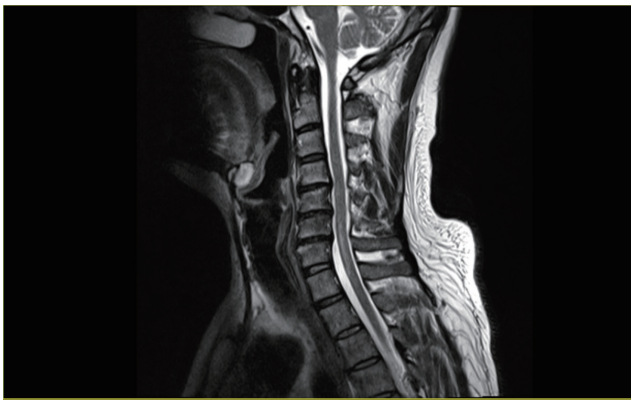

Figure 1: The spinal MRI done at the first hospital showed C6-T1 spinal cord hyperintensity on T2-weighted imaging.

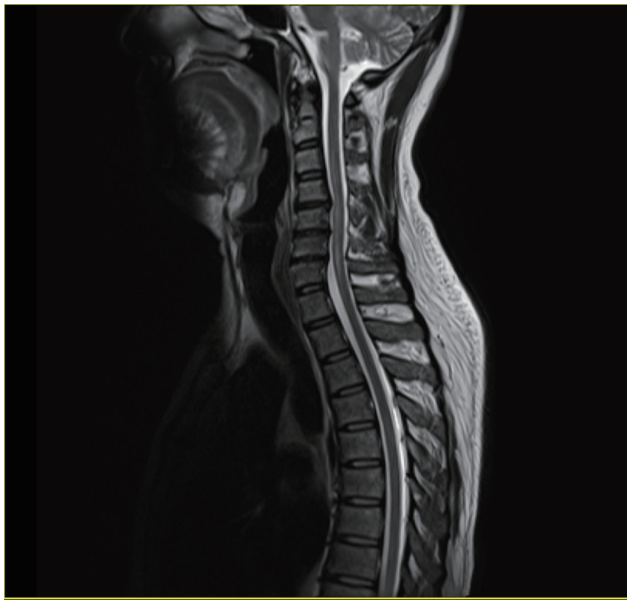

Figure 2A: Repeated cervical spinal MRI revealed anterior segment of C4-T1 spinal cord hyperintensity on sagittal T2 weighted imaging.

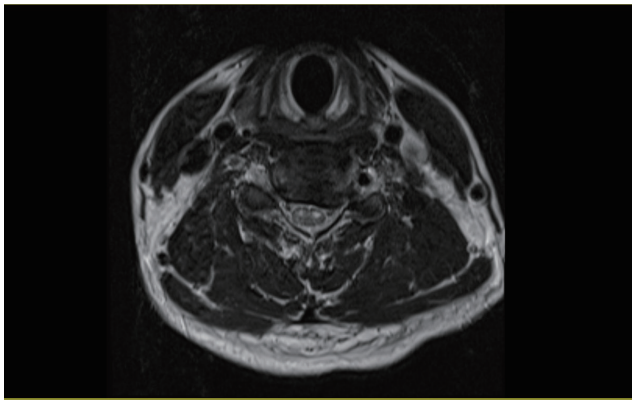

Figure 2B: "Owl's eye sign" on T2 weighted axial MRI at C5-C6.

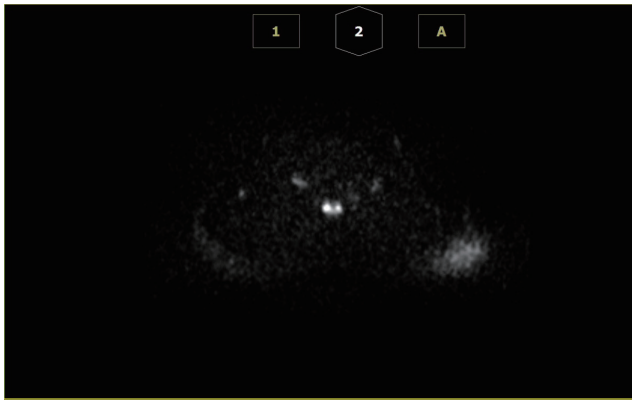

Figure 2C: DWI revealed hyperintensity at the same level.

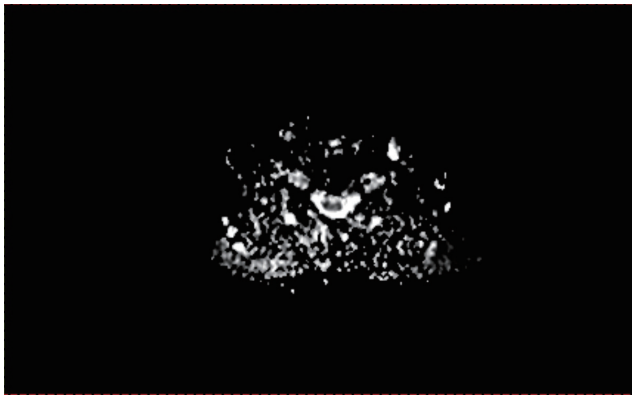

Figure 2D: Corresponding hypointensity in ADC.

consumed one pack of cigarettes per day. He had no history of recent infections or vaccination. Neurological examination revealed hypoesthesia below and including the C5 dermatome but with preserved vibration/proprioception sensation; hyperreflexia in the lower extremities, with bilateral positive Babinski sign; negative Hoffman sign and motor strength of grade 3/5 of deltoid muscles and all muscles inferior to the deltoids. Sphincter examination revealed hypotonicity. Based on the clinical course, the impression was IM. However, due to the presence of preserved proprioception sensation, we could not exclude anterior spinal cord syndrome. We repeated cervical MRI with diffused weighted imaging (DWI) and apparent diffusion coefficient (ADC). It revealed an anterior segment of C4-T1 spinal cord hyperintensity on sagittal T2 weighted imaging (Figure 2A). The "Owl's eye sign" was demonstrated on T2 weighted axial MRI at C5-C6 (Figure 2B). DWI revealed hyperintensity with corresponding hypointensity in ADC at the same level (Figures 2C and 2D). The image findings suggested the diagnosis of SCI. The laboratory investigation, including complete blood cell count, human T-lymphotropic virus 1+2 antibody, anti-nuclear antibody, anti-double strand DNA antibody, anti-phospholipid antibody, and tumor markers, was unremarkable. The CRP was <0.08 mg/dl (normal range: <0.3mg/dl). The carotid dopscan and trans-cranial Doppler revealed normal hemodynamics of bilateral extracrainal and intracranial vertebral arteries. External sphincter electromyogram revealed detrusor-sphincter dyssynergia. Aspirin was prescribed. After five days of treatment, the patient's quadriparesis improved enough to demonstrate motor strength of grade 4/5. However, he still had difficulty walking and gradually improved gradually over the course of one month. He still needed an indwelling Foley catheter after two months of follow-up. During the whole course of admission and follow-up, the patient's respiratory pattern was smooth and oxygen support was not needed.

DISCUSSION

SCI represents about only 1% of all types of stroke[4]. Presentation in the cervical region is even unusual[5]. It usually presents with a hyperacute course[2]. Our patient presented with a stepwise and stuttering course within six days with initially upper limb pain, chest pain with radiation to back, and then progressed to quadriparesis and acute urinary retention. The initial impression was IM, according to the clinical course. The diagnosis was made after repeating C-spinal MRI, including DWI and ADC. The temporal profile of symptoms (initial onset to nadir dysfunction) is the crucial feature that distinguishes SCI from IM at first presentation[2]. However, many prior cases of SCI have demonstrated a prolonged time to nadir over many hours, even days[2,6]. The observed condition makes the diagnosis even harder to differentiate between IM, infectious myelitis, and SCI. The prolonged and stuttering course with time to nadir > 4 hours, which is often considered unusual, are common and the diagnosis of SCI should always be kept in mind[7]. The Proposed SCI Diagnostic Criteria promoted by Zalewski et al.[7] suggests taking the following conditions as factors for diagnosing SCI: 1. If onset to nadir and severe deficits are present for 12 hours or less, 2. If the stuttering course is more than 12 hours, and severe deficits develop rapidly for 12 hours or less. Our case presented with the course of severe deficits developing rapidly within 12 hours. However, a prolonged course with time to nadir approaching six days has only rarely been described.

A history of sudden back pain or pain worsening on exercise point toward a vascular etiology[2]. In our case, the acute chest pain, which radiated to the back initially, mimicked aortic dissection. However, chest CT with contrast revealed no evidence of aortic dissection. The back pain may also point to the etiology of SCI.

Spinal MRI was essential for this case due to its prolonged time to nadir, which makes it hard to differentiate between IM and SCI. MRI is often normal in the acute phase of SCI (24%), and thus repeating imaging days later is recommended[7]. MRI patterns are described in patients with anterior spinal artery infarcts as having an "owl's eye" appearance on T2 weighted axial and pencil-like hyperintensities on T2-weighted sagittal images[8]. These classic findings may help to identify suspected SCI but are neither specific nor required for the diagnosis[7]. DWI of the spine, which is not routinely performed in clinical practice, is recommended to confirm the diagnosis of SCI and will reveal hyperintensity with the corresponding hypointensity in ADC, as in this case. However, the sensitivity of DWI was limited, and

the absence of diffusion restriction does not exclude SCI[6]. The Proposed SCI Diagnostic Criteria promoted by Zalewski et al.[7] considered DWI to be a "specific" MRI finding for the diagnosis of "definitive" SCI. Some articles also have suggested vertebral body infarction as an indicator of SCI that may help distinguish between myelitis and SCI[9].

The cause of nontraumatic acute SCI is variable. Aortic disease is the most common cause. Other etiologies include atherosclerosis, adjacent spinal degenerative disease, fibrocartilaginous embolism, decompression sickness, vertebral dissection, cardiac embolism, coagulopathies, vasculitides, surfing-related myelopathy, and systemic hypotension[8,10]. Atrial fibrillation is also associated with increased risk of subsequent SCI[11]. A thorough evaluation of potential underlying cardioembolic sources in patients with otherwise unexplained SCI may be needed. In our present case, no obvious etiology was found.

CONCLUSION

Spinal infarction is a rare condition and usually presents with a sudden or acute course. The prolonged course is rare and may mimic the presentation of IM. The diagnosis of SCI should always be kept in mind.

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