

Case Report

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Paraplegia due to Spinal Cord Infarction After Lifting Heavy Objects

Spinal cord infarction is uncommon and usually presents with sudden onset of motor and sensory disturbances. We report a case of a 64-year-old woman without previous medical history, who presented with acute onset of paraplegia after lifting. However, radiologic examinations did not show any abnormal lesion in the spinal cord. And, cerebrospinal fluid studies also showed no remarkable findings. This case illustrates the cause of spontaneous paraplegia after lifting injury and we consider the presumptive cause of paraplegia as spinal cord infarction.

KEY WORDS : Paraplegia · Lifting · Spinal cord infarction.

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INTRODUCTION

Spontaneous non-traumatic paraplegia is one of the miserable events for patients. Their differential diagnosis should cover compressive myelopathies including hematomas, tumors, inflammatory myelitis and vascular myelopathies. Infectious myelitis may be one of the usual causes which is closely related to neurotropic viruses or mycoplasmas in conjunction with meningitis or encephalitis, which can lead to severe sensorimotor deficits. In case of non-infectious inflammatory myelitis, one must differentiate between multiple sclerosis, acute disseminated encephalomyelitis (ADEM), and idiopathic transverse myelitis. Psychogenic origin may also be one of the cause of paraplegia^{4,8}. However, after evaluation to rule out the compressive lesion or inflammatory myelitis, vascular myelopathies should then be considered and carefully studied.

Spinal cord infarction is a rare clinical entity characterized by a sudden onset of paralysis, bowel and bladder dysfunction, and loss of pain and temperature perception, with preservation of proprioception and vibration sense. Herein, we present a patient with paraplegia after lifting the heavy objects and evaluate this rare cause of spinal cord infarction.

CASE REPORT

A 64-year-old woman with no significant medical history presented to our emergency department, complaining of the severe midthoracic pain after lifting heavy objects. On physical examination, the vital signs were normal. On neurologic examination, paraplegia without deep tendon reflexes was noted and sensory examination showed paresthesias to light touch from approximately T10 distally throughout her trunk and lower extremities. She also had loss of pain and temperature sensation below T10. Proprioception and vibratory sensation were intact. She could not feel the voiding sense and rectal tone with no cremasteric reflex.

From the neurological examination, the anterior spinal artery syndrome was speculated. To verify the compressive myelopathy, emergency MRI scans of the thoracic, and lumbar spine were performed, which revealed signal hyperintensities on T2 weighted images at the T10-T12 spinal cord. But, their images were not remarkable and compressive lesion was not shown either. We tried to perform the diffusion MRI to make the confirmatory diagnosis, but the quality of diffusion-weighted images was poor because of artifacts (Fig. 1). Based on these findings, we performed cerebrospinal fluid study to differentiate from a demyelinating spinal lesion or myelitis, though cerebrospinal fluid study revealed non-specific findings.

We tried to persuade the patient to undertake the angiography, however she refused because of slowness in neural function of recovery. She refused other procedures as well, and complained

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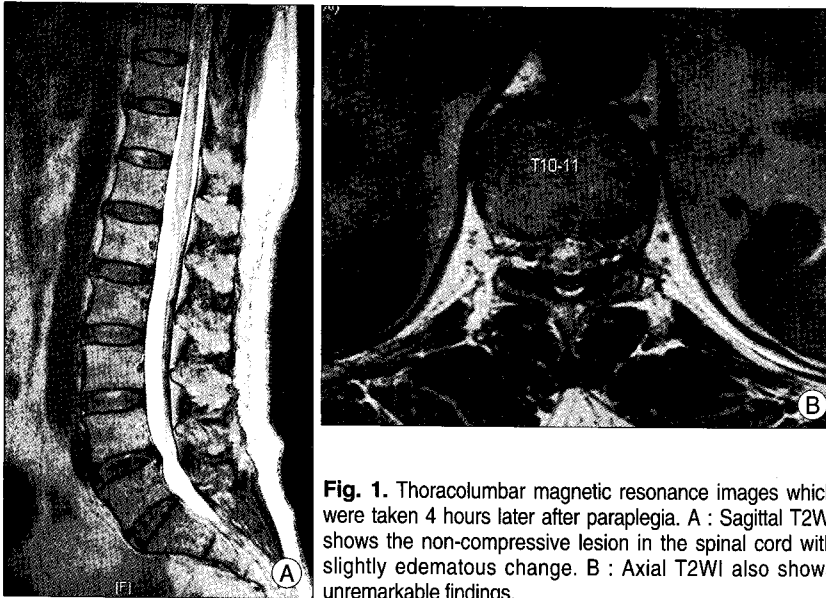


Fig. 1. Thoracolumbar magnetic resonance images which were taken 4 hours later after paraplegia. A : Sagittal T2WI shows the non-compressive lesion in the spinal cord with slightly edematous change. B : Axial T2WI also shows unremarkable findings.

that nothing was changed except diagnosis.

In conclusion, we assumed the cause was spinal cord infarction and treatment with corticosteroids was initiated. The patient's condition practically remained unchanged over 2 months. After two months of physical therapy, left big toe movement was slightly improved. After 1 month, she could flex left leg without assistance, though her right leg power was remain unchanged. Unfortunately, neurologic deficit has been unchanged 1 year after the presumptive diagnosis was made.

DISCUSSION

In cases of accidental paraplegia, every possible cause should be excluded. These include multiple sclerosis, transverse myelitis, medial disc herniation with cord compression, severe cervical canal stenosis, spinal contusion, spinal hematoma, spinal cord tumors, vascular malformations such as dural arteriovenous fistula with consecutive venous congestion and spinal cord infarction^{2,9}. However, in this case, we could not find out the spinal cord compression in radiologic studies. Unremarkable results in cerebrospinal fluid study could also rule out the demyelinating spinal lesion or myelitis. In that matter, we assumed that this case would be spinal cord infarction, though we could not make the confirmatory diagnosis from diffusion MRI.

Spinal cord infarction is much less frequent than cerebral infarction, accounting for only 1% of all strokes⁸. Furthermore, there are only a few reports about spinal cord infarction, showing various causes such as spontaneous and traumatic vertebral artery dissection⁴, hypotension, atherosclerosis of the vertebral arteries with severe stenosis⁵, infrarenal abdom-

inal aneurysm repair^{1,3,6,7}. Therefore, the pathogenesis and natural history of spontaneous or nonsurgical spinal cord infarctions remain largely unknown.

Novy et al.⁵ analyzed the clinical manifestation of 27 patients with acute spinal cord infarction between 1990 and 2003. They mentioned that they could not figure out the identifiable cause in most patients (20 patients, 87%). In rest of cases, they assumed that prolonged episode of arterial hypotension just before the infarction and disc prolapse or herniation may be the suspected causes. In our case, we also could not figure out the cause of spinal cord infarction. Reviewing her spine MRI, there was no identifiable

disc prolapse or herniation.

Only clue in our study was that she had lifted heavy objects and immediately after that promptly paraplegia had developed. Novy et al.⁵ asserted that movement of the spine can lead to acute vascular compression from their experiences of spinal cord infarction. Pryse-Phillips⁷ also asserted the similar point of view, where he reported the case of a young woman with a left vertebral artery dissection during fitness classes. Presumptive cause of dissection may have been due to sustained rotation of the neck during exercise. He also insisted that in the view of the potential unwanted effects due to this exercise, it was doubtful whether it should be recommended. We presumed that lifting movement of heavy objects would be closely related with vascular compression leading to spinal cord infarction.

Wenger et al.¹⁰ also assumed the possible cause of cord edema with nonspecific radiologic findings after mechanical compression. They presumed that the spinal cord might be injured directly from mechanical compression in patients with damaged facets of the cervical or posterior column. In such cases, the imaging can be nonspecific, showing only spinal cord swelling¹⁰. In present case, we can exclude this possibility of cause, because we could not detect the structural bone and ligament injury.

In her MRI, which was taken 4 hours later after paraplegia, we already mentioned that we could not find any remarkable things. Novy et al.⁵ also asserted the similar unremarkable finding in MRI. They reported that comparable to the early MR imaging of cerebral infarction, T2-weighted images were negative in their patients who underwent MR imaging in the first 3 hours after the onset of clinical symptoms. They also reported that follow-up imaging of the fifth and eight

day showed the confirmative spinal stroke, revealing hyperintensities on T2-weighted images and associated swelling of the spinal cord. They also reported a "pencil-like" hyperintensity in sagittal T2-weighted images was the typical MR imaging feature⁵⁾. It was unfortunate that our case that we did not take such a follow-up MRI. Instead, we tried to perform the diffusion MRI to have the confirmatory diagnosis, though the quality of diffusion-weighted images too poor to detect the clue. In reviewing the literature, there are only a few case reports about diffusion-weighted MR imaging of the spinal cord, showing changes of MR imaging in the case of acute spinal cord infarction.

Regretful thing in this study was the patient's refusal of resignation of spinal angiography, for which we tried to persuade but failed. Novy et al.⁵⁾ reported in his cases that there were half of cases that showed positive finding in their spinal cord angiography.

According to the literature reviews, treatment was only supportive and majority of patients had a substantial recovery over a period of weeks. However, in this case, she could not walk for herself until now.

CONCLUSION

Currently, there have been only few reports about spinal cord infarction of any origin. Treatment remains supportive

only with rehabilitative therapy. Here, we present a rare case of spinal cord infarction and discuss the presumptive cause after lifting the heavy objects

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