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Case Report

Venous hypertensive myelopathy associated with cervical spondylosis

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Abstract

BACKGROUND CONTEXT: Venous hypertensive myelopathy (VHM) results from spinal vascular malformations of arteriovenous shunting that increases spinal venous pressure, leading to congestive edema and neurologic dysfunction. There has been no report of VHM associated with cervical spondylotic myelopathy (CSM).
PURPOSE: The aim of this study was to report an extremely rare case of VHM likely due to CSM.
STUDY DESIGN: This study is a case report and review of the literature.
PATIENT SAMPLE: The patient was a 51-year-old man with CSM exhibiting relatively rapid neurologic deterioration with an abnormal expansion of a centromedullary hyperintense lesion on T2-weighted magnetic resonance imaging (MRI) in the absence of traumatic injury.
METHODS: Neurologic examination and radiologic imaging were taken by various means.
RESULTS: The patient developed a cervical radiculopathy, followed by gait disturbance and motor weakness. The MRI of the cervical spine demonstrated spinal canal stenosis due to disc bulging and flavum hypertrophy at the C5/C6 and C6/C7 levels as well as hyperintense area over the C5–C7 levels on T2-weighted images. Although decompression surgery was planned, an acute inflammatory process such as transverse myelitis or demyelinating disease other than cord compression was also considered, and the patient received intravenous steroids. His walking improved for several days. However, his symptoms then became significantly worse, and he had difficulty walking. Subsequent MRI demonstrated marked progression of the T2 hyperintense lesion over the C4–T1 vertebral levels. Flow voids were also noted on the dorsal surface of the upper cervical cord on T2-weighted MRI. His lab work, medical history, and the local enhancement on contrast-enhanced MRI indicated low probability of spinal inflammatory diseases. Therefore, the decision was made to perform anterior cervical discectomy and fusion surgery on two levels. Following surgery, his symptoms improved promptly.
CONCLUSIONS: Our case indicates that VHM could be caused by spondylotic cord compression in the absence of spinal vascular malformations. The diagnostic features for VHM are progressive deterioration of myelopathy, easing/worsening of symptoms associated with postural changes, and centromedullary hyperintensity over multiple segments and the flow voids on dorsal surface of the spinal cord on T2-weighted MRI.

Keywords: Ascending myelopathy; Diagnosis; Magnetic resonance image; Spinal inflammatory diseases; Spondylosis; Venous hypertensive myelopathy

Introduction

Cervical spondylotic myelopathy (CSM) is a common cause of compressive spinal cord dysfunction. Degenerative changes including disc bulging, osteophytes, and hypertrophy of posterior spinal ligament, reduction in canal diameter, and compression of the spinal cord, which can cause chronic neural injury and ischemia. The typical course of CSM is a relatively slow, progressive, and stepwise deterioration in

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neurologic function with stable plateau periods [1,2]. Rapid progress of neurologic deterioration of patients with CSM is fairly rare, and most cases are associated with minor trauma or segmental instability [3]. Even in such cases, signal changes are not often observed on MRI. Here, we report an extremely rare case of a patient with CSM exhibiting relatively rapid neurologic deterioration with an abnormal expansion of the high signal intensity area on T2-weighted MRI in the absence of traumatic injury.

Case presentation

A 51-year-old man, with no significant medical history including infections, presented with a tingling sensation in his left thumb and index finger with spontaneous onset. Within a week, his symptoms progressed, and he began to have difficulty with fine motor movements and weakness of his left hand. At 3 weeks after onset, he developed weakness of his left upper and lower extremity and gait disturbance with increased tone and stiffness. He got bilateral arm and leg numbness when fully supine position after several minutes. He slept on his right side because sleeping on his left side also caused numbness in the left arm and leg. He was examined by a neurosurgeon, and imaging studies were ordered. Plain radiographs demonstrated moderate degenerative spondylotic changes without evidence of instability. Magnetic resonance imaging demonstrated spinal canal stenosis at the C5/C6 and C6/C7 intervertebral levels as well as a relatively large area of high intensity area on T2-weighted images (Fig. 1). There were no signal changes in the spinal cord on T1-weighted MRI. Although the decompression surgery was planned, an acute inflammatory process, such as a transverse myelitis or demyelinating disease other than cord compression, was also considered given MRI findings. His reflexes were increased in both upper extremities, and Hoffman sign as well as Babinski were positive on the left side. He received intravenous steroids, and his walking improved for 1–2 days. However, his symptoms then worsened, and he had significant difficulty with walking. He then consulted with a neurologist, and repeat MRI of the cervical spine demonstrated marked progression of T2-weighted high signal area over the C4–T1 vertebral levels (Fig. 2). T2 high-intensity area was also observed in the right side of spinal cord in the axial section at the C5 vertebral level (Fig. 2B). However, the patient did not exhibit symptoms on his right side. Contrast-enhanced magnetic resonance (MR) images demonstrated the cord enhancement at C5/C6 level (Fig. 3, Top left), and only the left hemicord was enhanced in the axial plane, although there was no significant laterality in canal narrowing at this level (Fig. 3, Bottom left). To rule out the myelitis, he was tested for Lyme and neuromyelitis optica, and additional imaging of the whole spine and brain was performed. However, no abnormalities were noted in his subsequent work up, and he was then transported to our institution. Differential diagnoses included compressive myelopathy, transverse myelitis, demyelinating disease, and, less likely, tumor. After being

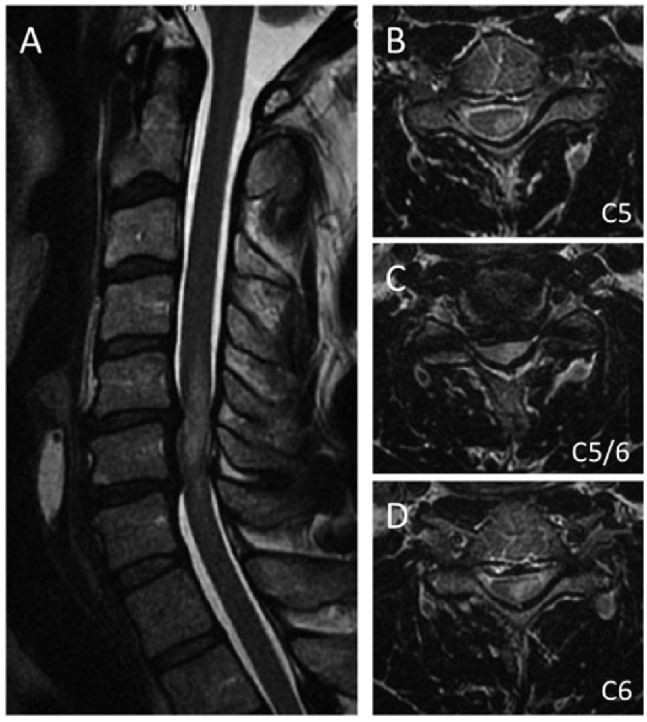

Fig. 1. Cervical magnetic resonance (MR) images before rapid deterioration. (A) Sagittal images demonstrate both anterior and posterior indentations at C5/C6 and C6/C7 levels as well as the T2-weighted high-signal intensity area. (B–D) Axial images show circumferential effacement of subarachnoid space at C5/C6 and T2-weighted high-signal intensity area at the left side of cord.

evaluated again by the neurology, neurosurgery, and orthopedic spine services, it was determined that the presence of his cervical disc herniations at C5/C6 and C6/C7 had some harmful impact on the cord edema, resulting in progressive neurologic deterioration. In addition, flow voids were noted on the dorsal surface of the upper cervical cord on T2-weighted MRI of whole spine (Fig. 3, Right), which is very specific for spinal venous hypertensive myelopathy (VHM) [4]. He agreed to undergo the operative decompression despite a higher risk of further neurologic deterioration in patients with pre-existing cord injury. A two-level anterior cervical discectomy and fusion (ACDF) with anterior plate was performed. Both the surgical and postsurgical courses were uneventful. After operation, his gait disturbance was significantly improved although his partial loss of dexterity in the left hand remained.

Discussions

We report an extremely rare case of ascending myelopathy presenting with rapid neurologic deterioration as well as abnormal expansion of high signal intensity area on T2-weighted MRI. In this case, coexisting transverse myelitis or multiple sclerosis other than compressive myelopathy was suggested by the enlargement of the T2 high-intensity area. However, the localized enhancement on contrast-enhanced