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Case Report

Sequential changes of ascending myelopathy after spinal cord injury on magnetic resonance imaging: a case report of neurologic deterioration from paraplegia to tetraplegia

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Abstract

BACKGROUND CONTEXT: Marked neurologic deterioration within a few days of traumatic spinal cord injury, known as subacute posttraumatic ascending myelopathy, is rare. Although several hypotheses regarding the pathogenesis of this condition have been proposed, the details remain elusive.

PURPOSE: To report a case of ascending myelopathy in which a series of magnetic resonance images (MRIs) taken through the course of the illness helped follow the course of the disease and discuss possible pathogenesis.

STUDY DESIGN: Case report and review of the literature.

PATIENT SAMPLE: A 75-year-old woman involved in a motor vehicle collision sustained a fracture dislocation of T7–T8 with complete paraplegia below T8.

METHODS: Neurologic examination and radiologic imaging taken by various means.

RESULTS: Posterior surgical stabilization was performed 18 hours after the injury. Both the surgical and postsurgical courses were uneventful. Four days after the injury, however, the patient reported feeling a tingling sensation in the right-hand fingers and gradually suffered from motor weakness of the upper extremities, deteriorating within a few hours to complete tetraplegia and ventilator dependence. Subsequent cervicothoracic MRI showed abrupt cord swelling with abnormal areas of signal intensity in the cervical and upper thoracic spinal cord during the interval between the onset of tingling and the development of motor paralysis in the arms. On the 20th postsurgical day, an area of hypointensity within the region of high intensity was observed on T2-weighted MRIs, indicating intramedullary spinal cord hemorrhage.

CONCLUSIONS: Our MRI findings suggest that systemically increased intraspinal pressure resulting from the impairment of spinal venous drainage is involved in the pathogenesis of ascending myelopathy. Although ascending myelopathy is often thought to be partly reversible, persisting increase of the intraspinal pressure may result in intramedullary hemorrhage and irreversible neurologic deficit.

Keywords: Spinal cord injury; Neurologic deterioration; Ascending myelopathy; Magnetic resonance image; Spinal cord swelling; Spinal venous drainage

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Introduction

Depending on the extent and the spinal cord segmental level of trauma, traumatic spinal cord injury (SCI) causes severe motor and sensory dysfunction, varying from partial to complete loss of the upper and lower limb functions. Because each spinal cord segmental level is associated with unique functional capacity and neurologic characteristics, subsequent loss of function of another spinal segmental level results in additional dysfunction. Despite early recognition of traumatic SCI, rapid transport, and spinal immobilization, secondary neurologic deterioration can occasionally occur. Harrop et al. [1] reported that 6% of complete cervical SCI patients exhibit neurologic deterioration during the first 30 days after injury.

The most common type of neurologic deterioration is a small rise in the level of the injury, but such deterioration tends to be slight, usually involving one or two segments at most. This deterioration usually occurs within the first several days after injury and is temporary. Thus, the final neurologic lesion is at the same level or lower than the lesion found immediately after injury [1]. Other less common forms of deterioration include ascending myelopathy, which involves delayed, subacute deterioration occurring more than four segments above the original level of injury [2]. In particular, neurologic deterioration ascended from the thoracolumbar region to the cervical level is distinctly rare. Because such spontaneous longitudinal spreading of the damage is so unlikely to occur, a double traumatic spinal lesion or a preexisting intraspinal tumor is speculated to be the cause of the deterioration [3]. Usually, magnetic resonance images (MRIs) taken initially on injury examine only the injured area. Thus, for example, a patient presenting with paraplegia would be subject only to a thoracic or thoracolumbar spinal cord MRI and not to a cervical/cervicothoracic MRI. A cervical MRI would be taken only when a patient presented tetraplegia. Therefore, MRIs of the cervicothoracic spinal cord taken after a patient presenting with paraplegia deteriorates to tetraplegia usually cannot be compared with MRIs taken before.

Here we describe an extremely rare case of ascending myelopathy, in which a traumatic SCI patient who sustained a T7–T8 fracture dislocation initially exhibited complete paraplegia, subsequently experienced with striking neurologic deterioration within a few days after the injury, resulting in complete tetraplegia. We compare cervicothoracic MRIs taken on the same day with those taken 1 and 1.5 years after injury. Based in the assessment of these MRIs, we consider the pathogenesis of ascending myelopathy that is not clearly understood.

Case presentation

A 75-year-old woman who was in a motor vehicle collision was taken to a local hospital, where on physical examination she had stable vital signs and was clear conscious. However, she exhibited complete paraplegia and sensory loss below the T8 level (ASIA Impairment Scale Grade A), and was therefore, transported to our spinal injury center. High-dose methylprednisolone was not administered. The patient also had hemopneumothorax, but her respiratory status was stable on room air.

A plain radiograph (Fig. 1, A) and computed tomography imaging (Fig. 1, B) showed a T7–T8 fracture dislocation with severe canal compromise. In addition, prominent ossification of the anterior and posterior longitudinal ligaments was observed at the thoracic level, indicating that the patient had ankylosing spinal hyperostosis. However, before the injury, she had not noted myelopathic symptoms, including numbness and/or sensory or motor disturbances of the lower extremities.

Magnetic resonance imaging scanning showed that the spinal cord was disrupted at T8 (Fig. 2, Left). Although slight anterior indentation brought about by ossification of the posterior longitudinal ligament was detected, there was no evidence of spinal cord lesions at the upper thoracic or cervical levels.

Eighteen hours after the injury, posterior surgical stabilization from T5 through T11 using a pedicle screw system was performed (Fig. 1, C and D). Surgical time was 124 minutes, and the blood loss was 134 mL. Both the surgical and postsurgical courses were uneventful. The patient was hemodynamically and neurologically stable, and no episodes of hypotension were observed.

Three days after the injury, the patient complained of increasing right chest and upper extremity pain. At that time, however, the MRI did not demonstrate any significant findings (Figure 2, Middle). The patient's motor function and sensation in the upper extremities were normal. The level of the initial spinal cord lesion could not be well visualized because of the presence of the surgical instrumentation.

Four days after the injury, the patient reported feeling a tingling sensation in the fourth and fifth fingers of her right hand, followed by weakness of motion in all the fingers of her right hand, which disappeared within hours. Four hours later, she complained of nuchal pain, and she reported that she could not move her right elbow and wrist. Five hours later, the patient began to suffer from motor weakness of the left upper extremity, and some hours later, the power of her left and right arms, including that of the deltoid muscle was 0/5. Trapezius muscle motion was almost normal. Later that day, an MRI of the brain showed absence of infarction (data not shown). A cervicothoracic MRI showed spinal cord swelling and areas of diffused, mildly hyperintense signal in the cervical and upper thoracic spinal cord in T2-weighted image (Fig. 2, Right). Thus, the cord swelling and the signal changes in wide segments (from the upper thoracic level through to the C2 level) emerged abruptly during the process of deterioration, coursing through several hours in the interval between the onset of tingling and the development of motor paralysis in the upper limbs. To reduce spinal cord edema, high-dose