Cervical Flexion Myelopathy Eleven Years after a Cervical Spinal Cord Injury

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Abstract:
We herein describe a 37-year-old man who developed cervical flexion myelopathy 11 years after suffering a cervical spinal cord injury. Cervical magnetic resonance imaging 11 years after the accident demonstrated atrophy and hyperintense lesions at the C6 and C7 levels in the cervical cord with an abnormal alignment of the vertebrae. In the neck flexion position, an anterior shift of the cervical cord was evident. Our patient's condition suggests that an abnormal alignment of the cervical spine and spinal cord injury due to a traumatic accident could be risk factors in the subsequent development of cervical flexion myelopathy.

Key words: cervical flexion myelopathy, Hirayama disease, cervical spinal cord injury, MRI

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Introduction

Cervical flexion myelopathy (CFM) is characterized by spinal cord injury caused by neck flexion (1). Several mechanisms cause spinal cord damages in patients with CFM, such as the compression of the spinal cord between vertebrae and the anterior-shifted posterior wall of the dura mater (tight dural canal in flexion), compression to the vertebral posterior surface without a tight dural canal in flexion, and overstretching of the spinal cord (2). Although CFM is usually associated with cervical spondylosis (3), not all patients with CFM demonstrate an abnormal alignment of the cervical spine. Hirayama disease is a subtype of CFM showing a tight dural canal in flexion as a prime mechanism with the absence of obvious cervical spondylosis (4). The developmental disproportion between vertebrae and the spinal cord has been thought to cause a tight dural canal in flexion that leads to ischemic damage in the cervical anterior horns during recurrent compression (5). Patients with Hirayama disease show homogeneous clinical symptoms, including unilateral or bilateral asymmetric muscle atrophy in the ulnar side of forearm and the absence of sensory disturbance. However, the sensory disturbance and long tract signs could be observed in patients with severely affected Hirayama disease (6).

In this case report, we describe a patient with CFM whose progressive neurological symptoms emerged 11 years after an episode of cervical spinal cord injury. An abnormal alignment of the cervical spine and spinal cord injury due to a traumatic accident could thus cause subsequent CFM.

Case Report

A 37-year-old man was admitted to the hospital due to muscle weakness and atrophy in his bilateral upper limbs. At the age of 24, he suffered a cervical spinal cord injury. He had an accident by which quadriplegia and dysesthesia in his distal four limbs developed. Cervical magnetic resonance imaging (MRI) demonstrated an abnormal alignment of the vertebral bodies and hyperintense lesions on T2-weighted images in the right-side cervical spinal cord at the C6 and C7 vertebral levels (Fig. 1A). After 6 months of conservative treatment with bed rest and rehabilitation, his weakness and dysesthesia improved. No apparent disabilities were observed regarding his activities of daily living.

Eleven years after the cervical spinal cord injury, he noticed progressive muscle atrophy and weakness in his bilateral distal upper limbs. Cervical MRI demonstrated atrophy and hyperintense lesions on T2-weighted images at the C6 vertebral level, which was consistent with the previously injured areas (Fig. 1A, B). In the neck flexion position, ante-
rior displacement of the spinal cord was observed (Fig. 1C). Surgical treatment was recommended; however, the patient chose observational treatment instead. Two years later, he was admitted to our hospital due to symptom progression. He showed left-dominant muscle atrophy and weakness in his distal upper limbs and upper motor weakness in his lower limbs. Muscle atrophy in the forearms was more prominent on the ulnar side with no obvious fasciculations. His left leg was spastic. Hyperreflexia with extensor plantar reflex was apparent. He presented with hypothermesthesia and hypalgesia in his proximal lower limbs. Vibration and position sensation in his left leg was decreased. His gait was spastic.

The findings of hematological and cerebrospinal fluid studies were unremarkable. The results of nerve conduction studies and sensory evoked potentials in his four limbs were normal. Electromyogram studies suggested chronic denervated states, including high amplitude units and reduced interference patterns, in the atrophied muscles (left extensor carpi ulnaris muscle and left interosseous dorsalis muscle). Reduced interference patterns were also observed in the left vastus medialis muscle and the left tibialis anterior muscle. Cervical MRI on T2-weighted images demonstrated atrophy and hyperintense lesions in the gray and white matter and the bilateral lateral funiculus at the C6 vertebral level. In the flexion position, an anterior shift of the spinal cord and spinal cord compression were evident. The imaging study results were consistent with those taken 2 years previously (Fig. 1B, C). Computed tomographic myelography also demonstrated an anterior displacement with flattening of the cervical spinal cord in the neck flexion position (Fig. 2). Cervical flexion myelopathy long after the traumatic cervical cord injury was thought to be the mechanism. We performed conservative management using a cervical collar.

Discussion

Eleven years after suffering a cervical spinal cord injury, the patient showed progressive muscle atrophy and weakness in his four limbs in addition to a sensory disturbance in the lower limbs. The MRI and myelography findings showed an anterior displacement of the atrophied cervical spinal cord and compression of the cervical spinal cord in the neck flexion position (Fig. 1B, C, 2). Although these imaging features were similar to those of Hirayama disease (1), CFM is a more appropriate diagnosis for our patient due to the fact that cervical MRI and myelography findings demonstrated the lack of any obvious anterior shift of the posterior wall of the dural matter in addition to the presence of the sensory disturbance in his lower limbs and cervical spondylotic changes (3, 4).

No patients who developed CFM many years after a cervical traumatic accident have been reported. Furthermore, the relationship between traumatic accidents and the development of Hirayama disease has not yet been elucidated in
Developmental disequilibrium between the vertebrae and the spinal cord could be associated with the development of Hirayama disease (5). In addition to developmental disproportion between vertebra and the spinal cord, an acquired abnormal alignment of the cervical spine and the spinal canal induces cervical spinal cord damage in neck flexion similar to that seen in CFM (7). In our patient, the abnormal cervical vertebral alignment which was likely due to an old cervical traumatic cord injury, could have created abnormal conditions leading to the development of CFM, although we have no information of the cervical alignment before the accident. Moreover, we presumed that the previous spinal cord injury may have increased the vulnerability of the cervical spinal cord to chronic compression. The previously injured portion and subsequently developed lesions of the cervical cord were located closely together (Fig. 1A, B).

In conclusion, cervical traumatic accidents and cervical spondylotic changes, such as the alignment change of the cervical vertebrae and spinal cord lesions, could therefore be risk factors for the development of CFM.

The authors state that they have no Conflict of Interest (COI).

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