## Case Report

# Secondary medulla oblongata involvement following middle cervical spinal cord injury associated with latent traumatic instability in a patient with ossification of the posterior longitudinal ligament

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**Study design:** A case report.

**Objectives:** To report a rare case of extension of edema and hemorrhage from initial C4–5 spinal injury to the medulla oblongata.

**Setting:** Center for Spinal Disorders and Injuries, Bibai Rosai Hospital, Japan.

**Methods:** A 68-year-old man with ossification of the posterior longitudinal ligament (OPLL) had sustained tetraplegia after tumbling over a stone. Initially, the patient was diagnosed with an acute C4–5 spinal cord injury without radiological abnormalities and was treated conservatively. At 7 h after the injury, the patient had an ascending neurological deficit, which required respiratory assistance. Magnetic resonance imaging revealed a marked swelling of the spinal cord above C4–5 extending to the medulla oblongata.

**Results:** Retrospective radiological assessment revealed that the spine was unstable at the injury level because of discontinuities in both anterior and posterior longitudinal ligaments. There was also signal intensity change within the retropharyngeal space at the C4–5 intervertebral disc. This injured segment was highly vulnerable to post-injury dynamic stenosis and easily sustained secondary neural damage.

**Conclusions:** This case report emphasizes a careful radiological assessment of latent structural instability in patients with OPLL in order to detect and prevent deteriorative change in the spinal cord.

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**Keywords:** cervical spinal cord injury; deteriorative change; medulla oblongata; ossification of the posterior longitudinal ligament

## Introduction

It has been documented that secondary neurologic deterioration after cervical spinal cord injury (CSCI) is one of the most severe management problems that might affect a patient's prognosis.[1,2]

Although the mechanism causing spinal deterioration is still unclear, a structural instability in the injured segment is one of the most important factors affecting these detrimental changes.[3] Surgical decompression and fixation of the cervical spine is the recommended treatment in a patient with structural damage, such as a spinal fracture-dislocation.[3] Conversely, it is debatable whether tetraplegic patients with no radiological evidence of structural spinal injury should be treated surgically or conservatively. However, some patients may actually harbor latent structural instability, which may cause secondary neurological deterioration. Therefore, it is important that latent instability should be promptly detected with a high index of suspicion during the acute management phase of CSCI.[4]

Here, we document an unusual case, where secondary detrimental change due to latent structural instability occurred in the medulla oblongata of a patient with ossification of the posterior longitudinal ligament (OPLL) following middle CSCI.

## Case report

A 68-year-old man tumbled over a stone and sustained complete tetraplegia. At 2 h later, the patient was found and admitted to a tertiary-care facility. The clinical parameters presented a normal range of the heart rate, blood pressure, and respiratory rate. Although the

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patient escaped from the brain, thoracic, and abdominal injuries, his neurological status was complete tetraplegia of C4. Plain X-ray, CT, and sagittal reformatted CT showed spinal canal stenosis due to OPLL (Figure 1). Although T1-weighted magnetic resonance imaging (MRI) failed to demonstrate any signal changes in the spinal cord, the T2-weighted image showed a low signal intensity area at the C4–5 level, which indicated intramedullary hemorrhage[5] (Figure 2a and b). Initially, the patient was diagnosed with an acute spinal cord injury at C4–5 without radiological abnormalities and treated conservatively. A Philadelphia collar was used but skull traction was not performed. Intravenous methylprednisolone was administered according to the recommended National Acute Spinal Cord Injury Study (NASCIS-2) protocol.[6]

At 7 h after the initial injury, the patient experienced increased dyspnea and transient loss of consciousness. When the patient regained consciousness, his neurological status changed to complete respiratory tetraplegia with neurological dysfunction at the C2 level. The patient presented with a normal sensory condition of the face, a sense of taste, and swallowing function. Neurological examination revealed normal function of the cranial nerves.
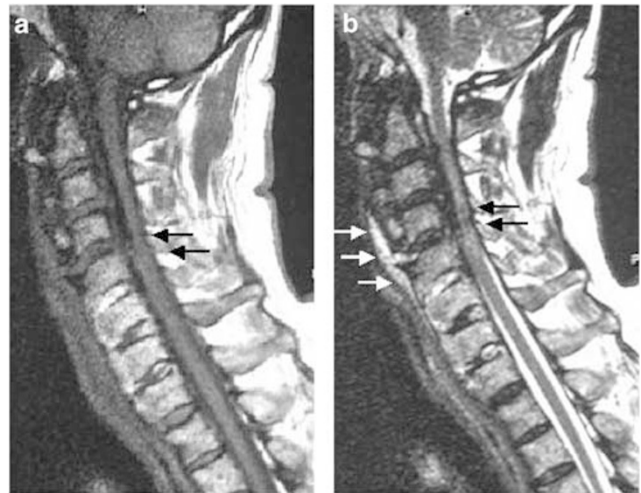
Despite normal cervical alignment, MRI at the onset of acute respiratory arrest showed a marked swelling of the spinal cord above C2–3 to the medulla oblongata. T1-weighted MRI showed high signal intensity in the
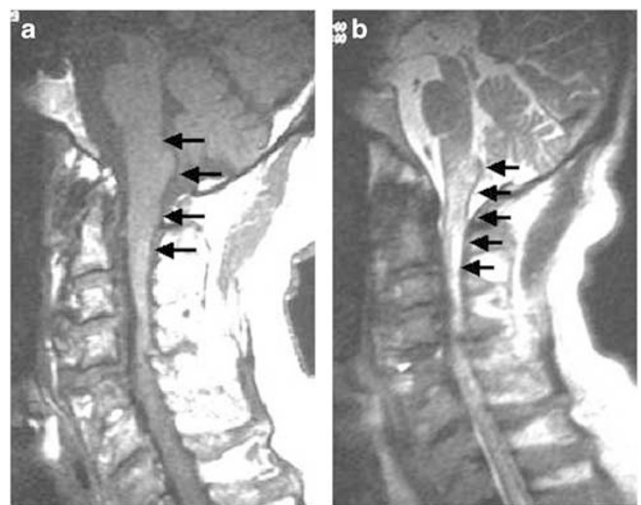
spinal cord from C4–2. T2-weighted MRI demonstrated diffuse heterogeneous high signal intensity within the spinal cord from C4 to the medulla oblongata. These findings indicated that spinal cord edema and intramedullary hemorrhage had extended cranially from the C4–5 level[5] (Figure 3a and b).

Retrospective radiological assessment revealed that the spine had been unstable at the injured level because of a discontinuity in both the anterior and posterior



**Figure 2** (**a** and **b**) MRI of the initial injury demonstrated the iso-signal intensity in the spinal cord on the T1-weighted image (**a**) and the low signal characteristics on the T2-weighted image at the C4–5 (**b**, black arrows). No structural injuries were initially identified, however, retrospective MRI assessment revealed a high signal intensity area on the anterior aspect of the C4–5 with the T2-weighted image (**b**, white arrows), which indicated structural instability at this site



**Figure 1** Reformatted CT revealing spinal canal stenosis accompanied with OPLL at the initial injury



**Figure 3** (**a** and **b**) MRI at the onset of acute respiratory arrest showed a marked swelling of the spinal cord above C2–3 as far as the medulla oblongata (arrows). T1-weighted image showed a high signal intensity in the cord from C4 to 2 (**a**). T2-weighted image revealed diffuse heterogeneous high signal intensity in the cord from C4 to the medulla oblongata (**b**)
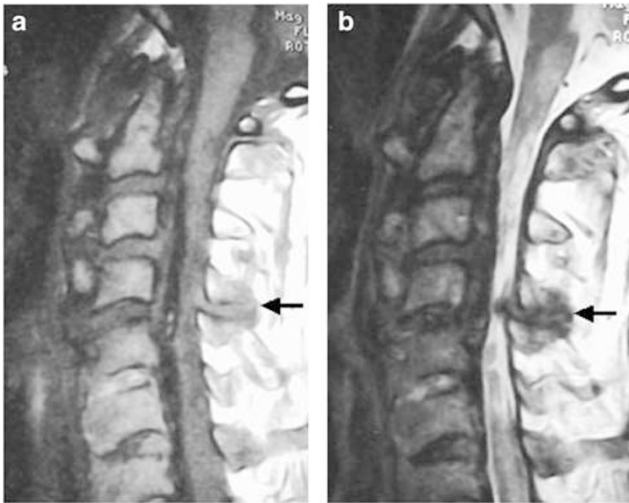
**Figure 4** (**a** and **b**) MRI performed 3 months after the injury showed extensive gliosis from the inferior medulla oblongata to all sections of cervical spinal cord. Moreover, it revealed hypertrophic change in the ligamentum flavum at C4–5 indicating structural instability at this site (arrow)

longitudinal ligaments and with a signal intensity change in the retropharyngeal space between the C4–5 intervertebral discs (Figures 1 and 2b).

The patient was referred to our institute 4 weeks after injury. He was managed conservatively because surgical intervention at such a late stage, after neural damage, would be expected to provide little chance of any neurological improvement. Furthermore, the patient and his family did not elect for surgery to be performed.

MRI obtained 3 months after injury showed that gliosis extended from the medulla oblongata to middle part of the cervical spinal cord, which exhibited a uniform signal intensity on the T1-weighted image but a high signal intensity on the T2-weighted image (Figure 4a and b). Moreover, hypertrophy of the ligamentum flavum was observed at C4–5 segment. This finding proved that there was an initial, undetected instability within C4–5 segment, which led to secondary deterioration of the injured spinal cord.

## Discussion

The incidence of neurological deterioration from spinal injury was reported to be between 1.84, and 5.8%.[1,2] Many reports have documented that hemorrhage, microvascular infarction, spasm, spinal cord edema, and free radical production are relevant factors affecting this serious condition.[7,8]

Lu et al[9] reported the predictable risk factors for delayed apnea following middle to lower CSCI in the presence of diffuse and extensive cord lesions, respiratory distress, and bradycardia. In this case, however, the initial spinal cord lesion was localized to the C4–5 segment and cardiopulmonary functions were normal.

The most important point regarding initial management of this case was that the patient was managed as though he had a stable injury that required no surgical decompression and stabilization. However, in this case, there was mechanical instability in the interrupted portion of OPLL (C4/5 segment). When mechanical instability exists between the OPLL, spinal cord mechanical stress is usually focused at the same site. Consequently, secondary spinal cord injury often aggravates the neurological condition. Owing to the fact that CSCI without osteoligamentous damage is quite common in the elderly population with degenerative spinal stenosis including OPLL, CSCI with spinal stenosis tends to be managed as a stable injury without detailed radiological evaluation. Therefore, it is important to identify any latent mechanical instability using MRI and to perform adequate management using surgical decompression with or without fusion in the acute phase of injury.[4]

Both ankylosing spondylitis (AS) and diffuse idiopathic skeletal hyperostosis are well-known predictive neurological deterioration risk factors.[10,11] Bohlman[12] reported that 50% of identified AS patients had a delay in the diagnosis of cervical spine structural injury and all of them subsequently developed secondary neurological deterioration. Reid et al[13] documented a high incidence of neurological deterioration due to this delay in diagnosis. In this case, all cervical spinal segments except C4–5 were fused by ossification of both anterior and posterior longitudinal ligaments. Therefore, any force applied to the cervical spine would be concentrated on the weak C4–5 segment in a similar situation of a fractured ankylosing spine. Therefore, the C4–5 segment of the spinal cord in our case was vulnerable to postinjury dynamic stenosis and easily sustained secondary neural damage.

The clinical manifestations of patients with medulla oblongata injuries are unclear because such patients rarely survive. This is the first clinical case report describing the deterioration in the ascending medulla oblongata following middle CSCI. After postmortem neuropathological examination, Ito et al[14] revealed longitudinal spreading of cord lesions upward into the medulla oblongata in one of eight patients who died after traumatic CSCI. They also documented that there was a completely necrotic lesion without any cell reaction in several cord segments as well as the initially damaged lesion. They suggested that the spreading of the lesion might be induced by increased intramedullary pressure that resulted from an intra- and extramedullary circulatory disturbance. In the current case, the critical instability of the C4–5 segment associated with severe spinal stenosis caused by OPLL could have led to an acutely increased intramedullary pressure, which permitted the extension of the spinal cord lesion.

The patient escaped from cranial nerve damage in spite of medulla oblongata involvement. This may be explained because the involved lesion was located within only the inferior portion of the medulla oblongata that contains no the cranial nerve nuclei but only the spinal

trigeminal nuclei. In addition, there were discrepancies between the patient's neurological status and the MRI findings. Therefore, the apnea was not necessarily the result of the secondary involvement of the medulla oblongata, but might simply result from the involvement of the C4 segment in which the phrenic nerves are located.

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