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Thoracic Spondylolisthesis that Worsened Symptoms of the Thoracic Spinal Cord Potentially Caused by Patient Positioning During Surgery: A Case Report

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Abstract:

Thoracic spondylolisthesis is a rare disease, but symptoms become severe once a patient develops myelopathy. We experienced a case of thoracic spondylotic myelopathy caused by mild degree of spondylolisthesis in the upper thoracic spine associated with cervical and lumbar spine lesions. The symptoms worsened after prone positioned cervical decompression. Dynamic computed tomography imaging of the thoracic spine in forward and backward bending was useful in evaluating instability of the thoracic spine.

Because the caudal part of the region causing spondylolisthesis had fused with the anterior longitudinal ligament, regional instability potentially occurred based on the principle of lever arm even though the degree of spondylolisthesis was mild. Diagnosis of thoracic spondylolisthesis is difficult, but because symptoms are likely to become severe, it is necessary to carefully acknowledge pathological conditions.

Keywords: - Thoracic spondylolisthesis, mild degree, myelopathy.

Introduction

The thoracic spine shows high biomechanical stability because of the presence of ribs. The frequency of occurrence of thoracic spondylolisthesis is low and rare compared to that of lumbar or cervical spondylolisthesis, but once the symptoms appear, thoracic spondylolisthesis often presents with serious symptoms of myelopathy.[1]

In addition, because plain radiographic evaluation of instability in the thoracic spine is difficult, doctors often struggle to diagnose instability in thoracic spondylolisthesis.

We experienced a rare case of thoracic spondylotic myelopathy caused by thoracic spondylolisthesis which occurred due to sudden lower extremity paralysis. Mild degree of spondylolisthesis in the upper thoracic spine complicated by severe cervical spinal stenosis and lumbar spinal stenosis was identified, but there were no findings related to nerve compression in thoracic spine; therefore, we performed cervical laminoplasty and lumbar laminectomy. Exacerbation of lower extremity symptoms was found following the surgery, and then myelopathy associated with thoracic spondylolisthesis became evident.

Case Presentation

A 78-year-old male was immediately taken to hospital because, since the time of awakening, he had suffered severe paralysis of the right lower extremity, mild paralysis of the left lower extremity, and urinary retention. No trauma associated with his medical conditions was observed. He had experienced numbness in the lower extremities for some years and intermittent claudication for half of a year.

Neurological Findings

Upper extremity muscular strength was normal, but the scores for manual muscle testing (MMT) of the right lower extremity distal to the iliopsoas muscle and the left lower extremity were 1/5 and 4/5, respectively.

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Loss of sensation extended from the perineum and inguinal region to the distal region and sensory denervation of the right lower extremity were experienced by the patient.

Deep tendon reflex testing showed exaggerated upper extremity reflex and knee jerk reflex. Achilles tendon reflex was absent on both sides, and the Hoffman's sign was absent.

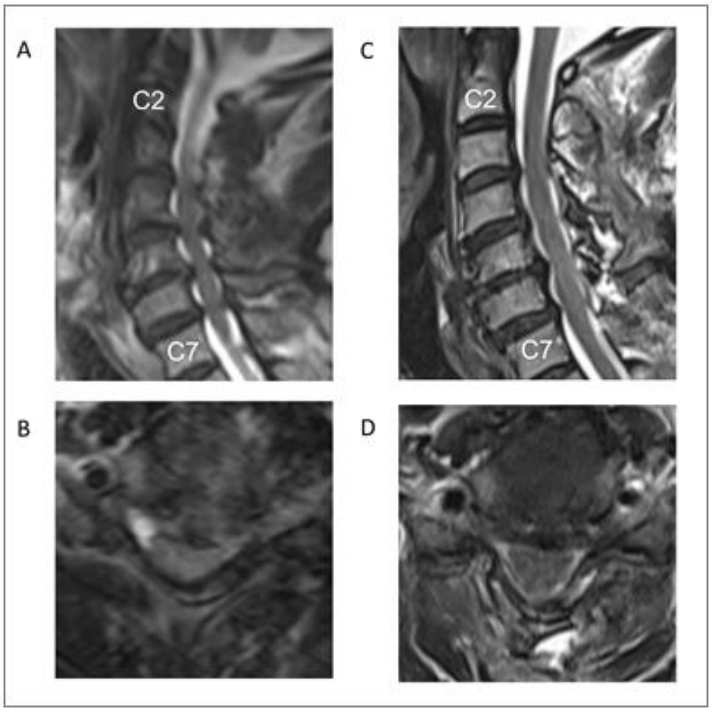

Figure 1: MRI findings of the cervical spine before and after nerve decompression. T2-weighted images.

1A: Sagittal image of the cervical spine at disease onset.
1B: Axial image of the cervical spine (at the C5-C6 levels) at disease onset. Compressive deformation of the spine was observed.
1C: Sagittal image of the cervical spine after cervical laminoplasty.
1D: Axial image of the cervical spine (at the C5-C6 levels) after cervical laminoplasty. MRI shows successful decompression of the spinal cord.

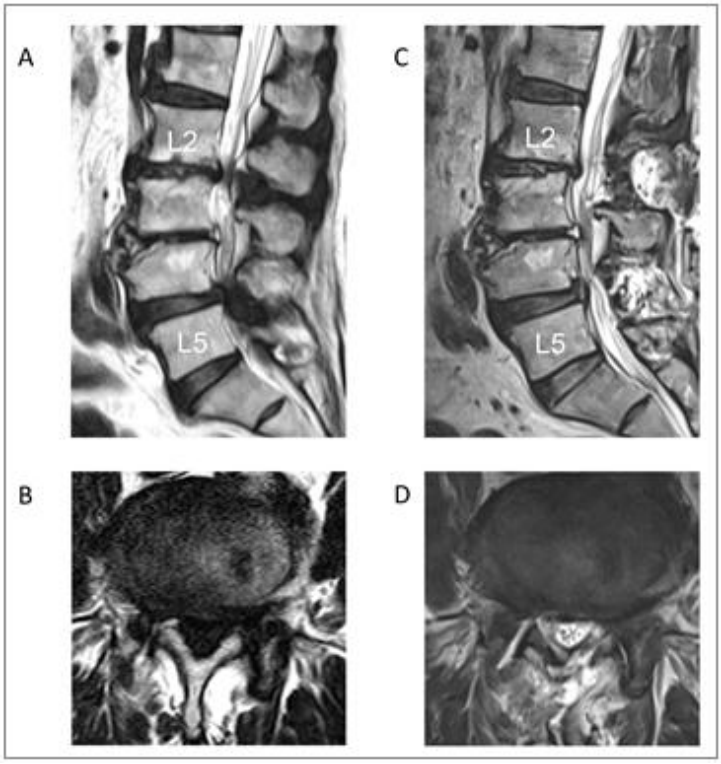

Figure 2: MRI findings of the lumbar spine before and after nerve decompression. T2-weighted images.

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2A: Sagittal image of the lumbar spine at disease onset. Severe stenosis was found at the L2-L3 and L4-L5 levels.
2B: Axial image of the lumbar spine (at the L4-L5 levels) at disease onset. The dural sac was not identified due to severe stenosis.
2C: Sagittal image of the lumbar spine after nerve decompression. MRI shows adequate decompression of the dural sac.
2D: Axial image of the lumbar spine (at the L4-L5 levels) 7 days after nerve decompression. MRI provides good depiction of the dural sac.

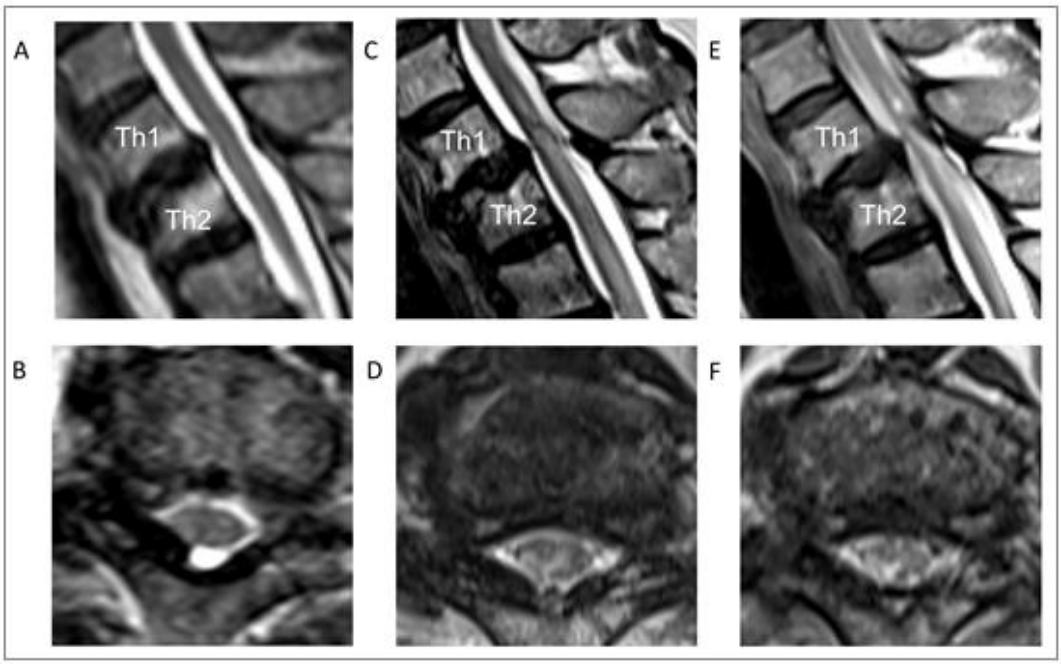

Figure 3: Time dependent change of MRI of the thoracic spinal cord. T2-weighted images.

3A: Sagittal image of the thoracic spine (at the Th1-Th2 levels) at disease onset. Mild spinal stenosis was observed, but the apparent hyperintensity of the spinal cord and spinal cord edema were not found. Visualization of cerebrospinal fluid flow on MRI was successfully obtained.
3B: Axial image of the thoracic spine (at the Th1-Th2 levels) at disease onset. Neither deformation of the spinal cord nor intramedullary spinal cord changes in signal intensity were identified.
3C: Sagittal image obtained a week after the onset of the disease. Edematous changes and hyperintensity of the spinal cord caudally below the level of Th1 appear.
3D: Axial image of the thoracic spine (at the Th1-Th2 levels) obtained a week after the onset of the disease. No deformation of the spinal cord was observed, but intramedullary spinal cord changes in signal intensity were identified.
3E: Sagittal image obtained a month after the onset of the disease. Localized intramedullary high-signal intensity was seen around the level of Th1 and Th2.
3F: Axial image of the thoracic spine (at the Th1-Th2 levels) obtained a month after the onset of the disease. Intramedullary high-signal intensity increased around the right half the spinal cord.

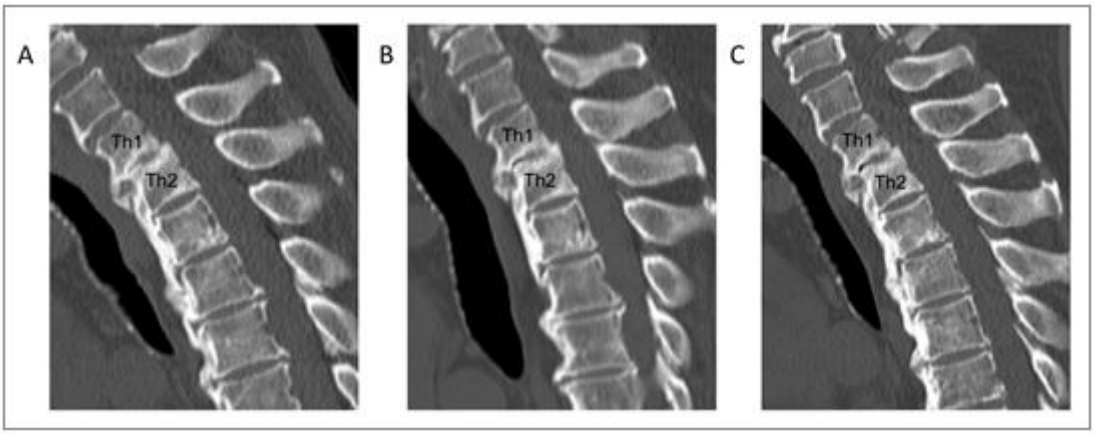

Figure 4: Dynamic CT imaging of the thoracic spine.

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4A: Cervical forward bending. The interspinous space between Th1 and Th2 was widened compared to that in a midline position.
4B: Thoracic spine CT scan in a midline position at disease onset. The anterior longitudinal ligament caudally below the level of Th2 had been fused.
4C: Cervical backward bending. The interspinous space between Th1 and Th2 was narrowed compared to that in a midline position, and vacuum phenomenon occurred within the intervertebral disc lying between Th1 and Th2.

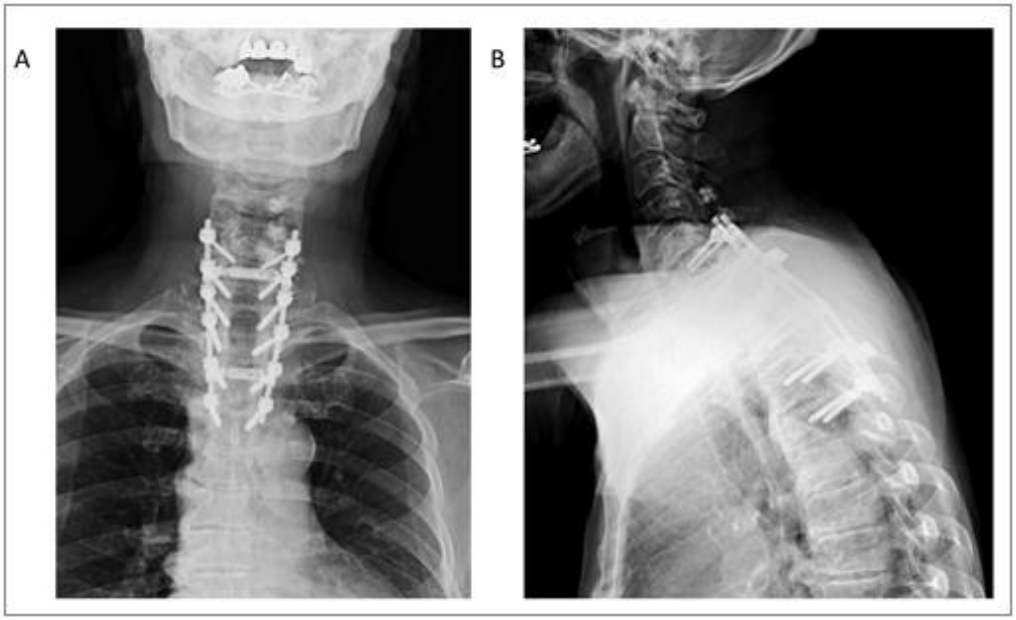

Figure 5: X-ray image of thoracic spine surgery following C6 to Th4 posterior fixation.

5A: Frontal view
5B: Lateral view

Imaging findings (at onset of the disease)

Whole spine computed tomography (CT) and magnetic resonance imaging (MRI) were performed, and images of the entire spine were acquired for evaluation.

Severe cervical spinal stenosis (Figure 1A and 1B) and severe lumbar spinal stenosis at the L2-L3 and L4-L5 levels (Figure 2A and 2B) were found, and prominent compressive deformation of the dural sac was observed.

The anterior longitudinal ligament below the level of Th2 had the tendency to fuse, and mild degree of spondylolisthesis (forward slippage of Th1 on Th2, Figure 4B) and mild thoracic spinal stenosis were observed. There were no findings of compressive deformation of thoracic spinal cord on MRI (Figure 3A and 3B).

Follow-up after cervical and lumbar spine surgery

Because cervical spondylotic myelopathy associated with cervical spinal stenosis was considered to be the origin of the paralyzed proximal lower extremity muscles and exaggerated tendon reflexes (except for Achilles tendon), cervical laminoplasty was performed.

Spasticity of the lower extremities became worse immediately after the surgery. Paralysis of right lower extremity remained unchanged. MMT score of left lower extremity strength became 3/5, indicating a worsening of the paralysis. Lumbar decompression at the L2-L3 and L4-L5 levels was subsequently performed, but this procedure did not help improve the symptoms.

After the surgery, MRI scans confirmed that nerves in the cervical spine and lumbar spine were adequately decompressed (Figure 1C and 1D, Figure 2C and 2D). A week after the onset of the disease, spinal cord edema and T2-weighted intramedullary high-signal intensity around the level of Th1 and Th2 were observed (Figure 3C and 3D).

Later, spinal cord edema was alleviated, and intramedullary high-signal intensity at the Th1-Th2 levels increased over time (Figure 3E and 3F).

Because thoracic myelopathy associated with thoracic spondylolisthesis at the Th1-Th2 levels was considered as an etiological factor, dynamic CT of the thoracic spine in forward and backward bending was performed (Figure 4). The interspinous space between Th1 and Th2 was widened or narrowed when the patient bent his neck forward or backward. Forward slippage of Th1 became worse when he bent his neck forward (Figure 4A), and vacuum phenomenon occurred within the intervertebral disc lying between Th1 and Th2 when he bent his neck backward

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(Figure 4C). For these reasons, we considered that there was spinal instability between Th1 and Th2, and therefore, thoracic decompression and spinal fusion surgery were performed (Figure 5).

Findings on thoracic spine surgery

C6 to Th4 posterior fixation was performed (Figure 5), followed by partial laminectomy at the Th1-Th2 levels. In terms of patient positioning during surgery, neck was stabilized in a midline position, and special care was taken for thoracic spondylolisthesis and nerve compression which can be potentially exacerbated by muscle relaxation during general anesthesia.

Follow-up after thoracic spine surgery

After the surgery, the right lower extremity (MMT: 2/5) and the left lower extremity (MMT: 3/5) became less severe. The patient was discharged from the hospital after walking exercises.

Discussion

The frequency of occurrence of thoracic myelopathy is low compared to that of cervical myelopathy[2,3], but once thoracic myelopathy develops, symptoms progress rapidly and become severe. Thoracic myelopathy is mostly caused by herniated intervertebral disc and spinal stenosis, and in East Asia, particularly ossification of the posterior longitudinal ligament or the ligamentum flavum is one of the most common causes of thoracic myelopathy in addition to the above diseases[2,3,4,5]; however, there are only a few reports on thoracic myelopathy triggered by thoracic spondylolisthesis in lower thoracic spine[2,6,7,8], and this is the first case report of thoracic myelopathy associated with upper thoracic spondylolisthesis. Because the thoracic spine is mechanically stable due to the presence of ribs, the frequency of occurrence of thoracic spondylolisthesis is low compared to that of lumbar spondylolisthesis or cervical spondylolisthesis.

On the other hand, the symptoms become severe when they appear in the thoracic spinal cord. This vulnerability of the thoracic spinal cord is considered due to a natural outward curvature (kyphosis) which is vulnerable to the forces pressing against the front of the body or the fragility of spinal cord hemodynamics compared to other regions.[1]

Evaluation of instability in the spine is usually performed with dynamic X-ray. Although it is possible to evaluate instability of the cervical or the lumbar spine using this procedure in detail, plain radiographic evaluation of instability in the thoracic spine is difficult due to the presence of ribs, the scapula, and the lung. In our case, dynamic CT was useful in evaluating the presence or absence of instability of thoracic spondylolisthesis.

We knew that our patient had a mild degree of thoracic spondylolisthesis at disease onset, but because compression and deformation of the spinal cord were not identified by MRI at disease onset and because thoracic spondylolisthesis was complicated by cervical and lumbar spine lesions, we did not recognize that thoracic spondylolisthesis was the cause of pathology.

In our case, the patient was heavily paralyzed in the lower extremities without trauma associated with his medical conditions, which indicates the possibility that his sleeping position caused compression of the thoracic spinal cord. Moreover, the position during surgery may further adversely affect thoracic spondylolisthesis because symptoms have been exacerbated after cervical spine surgery performed in prone position.

During posterior approach for cervical spine surgery, it is unlikely that the patient has poor postures such as forward or backward bending because the head is fixed using a Mayfield three-point skull clamp in midline, but simultaneously it may be hard to realize that the entire head has shifted forward or backward. In our case, because the anterior longitudinal ligament below the level of Th2 had the tendency to fuse, instability at the Th1-Th2 levels occurred based on the principle of lever arm. Regarding bone spur formation in the front of the vertebral body, a bone spur in the front of Th1 had formed on the bone spur in the front of Th2; therefore, tolerance against head position shifting forward seems to be low. Instability of the thoracic spine could have become even worse due to muscle relaxation during general anesthesia when compared to the evaluation of instability using dynamic CT.

Conclusion

We experienced a severe case of thoracic spondylotic myelopathy complicated by mild degree of thoracic spondylolisthesis.

Once symptoms of myelopathy appear, the symptoms progress rapidly and become severe; therefore, carefully acknowledging pathological conditions is required for thoracic myelopathy.

COI disclosure

The authors declare that there is no conflict of interest regarding the publication of this paper.

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