OPEN Case Report

Thoracic Myelopathy Caused by Thoracic Degenerative Spondylolisthesis and Lumbar Scoliosis

Hong Jin Kim, MD, PhD
Se-Il Suk, MD, PhD
Dong-Gune Chang, MD, PhD

From the Department of Orthopaedic Surgery, Inje University Sanggye Paik Hospital, College of Medicine, Inje University, Seoul, Korea (Dr. Kim, Dr. Suk, and Dr. Chang).

Correspondence to Dr. Chang: dgchangmd@gmail.com

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ABSTRACT

Introduction: Spine deformities, including degenerative spondylolisthesis and scoliosis, are caused by several factors such as intervertebral disk degeneration and facet joint arthrosis, leading to spinal instability. Degenerative spondylolisthesis of the thoracic spine is less common because of the stabilizing properties of the rib cage and facet joints in the anterior-posterior position.

Methods: A 70-year-old female patient reported of back pain and left lower extremity weakness. The patient's premorbid ambulation status was wheelchair-bound because of right-sided monoparesis caused by a history of cerebral palsy. Neurological examination revealed grade 1 left-sided hip flexion and knee extension.

Results: Radiological findings showed lumbar scoliosis, severe disk degeneration, degenerative spondylolisthesis with facet arthrosis, and yellow ligament hypertrophy at T10–T12. Increased high signal intensity in T2-weighted magnetic resonance imaging and cord compression was observed at T10–T11, indicating thoracic myelopathy. We performed total laminectomy and posterior instrumented fusion at T10–T12. At a 3-month follow-up, the patient's motor grade improved to grade 4.

Conclusion: Thoracic myelopathy in our case was caused by segmental instability, including multilevel lower thoracic degenerative spondylolisthesis, the presence of ossification of the ligament flavum, and lumbar scoliosis. This rare case, involving multiple complex spinal conditions, provides valuable insights into the development of thoracic myelopathy.

Spine deformities, including degenerative spondylolisthesis and scoliosis, are caused by several factors such as intervertebral disk degeneration and facet joint arthrosis, leading to spinal instability.[1-3] Degenerative spondylolisthesis of the thoracic spine is less common than that in the lumbar regions because of the stabilizing properties of the rib cage and

facet joints in the anterior-posterior position.[1,4-8] Furthermore, scoliosis is associated with global disability caused by cerebral palsy (CP), which encompasses a range of heterogeneous conditions, including abnormal muscle tone, movement, and posture.[9-11] These conditions, which lead to spinal instability, can cause thoracic myelopathy, potentially resulting in neurological deficits.[4] Thoracic myelopathy can be diagnosed through evidence of cord compression and high signal intensity on T2-weighted MRI as well as electrophysiological studies.[1] Given that the thoracic spinal cord is highly susceptible to compression and ischemic damage, surgical decompression of the compression source is crucial to prevent unavoidable complications.[3,4] In this report, we present a rare case of thoracic myelopathy caused by spinal instability due to degenerative spondylolisthesis in the lower thoracic region, accompanied by lumbar scoliosis as a sequela of CP.

Case Report

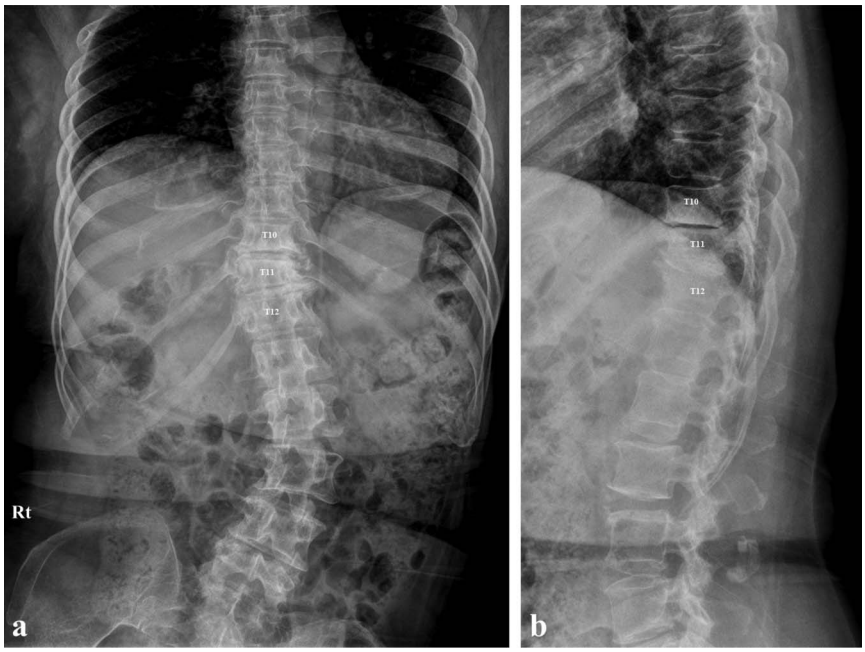
A 70-year-old female patient reported of back pain and left lower extremity weakness. The patient's premorbid ambulation status was wheelchair-bound because of right-sided monoparesis caused by a history of CP. Neurological examination revealed grade 1 left-sided hip flexion and knee extension as recent deterioration manifestations and grade 1 right-sided hip flexion and knee extension as a sequela of CP. The plain radiograph showed lumbar scoliosis with a rotational change of L1–L3, sclerotic change of vertebral end plates at T10–T12, and loss of lordosis and kyphosis (Figure 1). CT imaging of the thoracic spine presented vacuum phenomena and height decrease of the intervertebral disks at T10–T12, ossification of the ligament flavum (OLF) at T10–T11 presented with a black arrow (Figure 2, A), degenerative spondylolisthesis at T10–T12 and facet cyst in the lower end plate of T11 vertebra's posterior portion presented with a red arrow (Figure 2, B), facet arthrosis, and marginal osteophytes (Figure 2, C–F). MRI demonstrated severe cord compression at T10–T11, increased high signal intensity in T2-weighted imaging, and ligament hypertrophy at T10–T12, indicating thoracic myelopathy (Figure 3, A–D). No specific findings for degenerative changes were observed on the lumbar lesions (Figure 3, E). We performed total laminectomy and posterior instrumented fusion at T10–T12 with the expectation of alleviating the patient's symptoms and signs (Figure 4, A and B). The open surgical procedure involved decompression through a posterior approach using total laminectomy, pedicle screw instrumentation by the free-hand technique, and posterior fusion using resected autogenous bone and demineralized bone matrix. At the 3-month follow-up, the patient's symptoms had improved, with the motor grade showing a score of 4 for left-sided hip flexion and knee extension. The symptoms remained well improved at both the 6-month and 1-year follow-ups. At the 2-year follow-up, radiological findings showed relieved end plate sclerosis at T10 to T12 (Figure 4, C and D).

Discussion

Thoracic myelopathy can occur secondary to degenerative disorders, such as ossification of the posterior longitudinal ligament and OLF.[5,6] However, degenerative changes in thoracic spine can lead to devastating conditions such as chronic myelopathy. Therefore, it is crucial to understand how degenerative changes in the thoracic spine can lead to segmental instability and result in thoracic myelopathy with neurological deficits. Our case illustrates these manifestations, emphasizing the significance of lumbar scoliosis and degenerative spondylolisthesis. Scoliosis in the lumbar region, observed as a sequela of CP, gradually increased the load on the lower thoracic region, leading to segmental instability. This instability, involving two-level thoracic degenerative spondylolisthesis, and the existence of OLF caused compression on the narrow spinal cord, resulting in thoracic myelopathy that necessitated surgical intervention.

Hou et al[6] described the clinical features of thoracic spinal stenosis–associated myelopathy. In 427 cases, the disease was mainly caused by OLF (72%) or thoracic disk herniation with posterior osteophytes (43%) at T9–L1.[6] In 2006, Shimada et al[4] initially reported five cases of single-level degenerative spondylolisthesis accompanied by disruption of the facet joint. Hsieh et al[7] also reported five case series of single-level degenerative spondylolisthesis with concomitant lumbar spondylosis. Recently, Wang et al. presented a case of thoracic degenerative spondylolisthesis–associated myelopathy at T11–T12.[1] The commonality with our case was that degenerative spondylolisthesis was mainly observed in the lower thoracic region, especially at T10–T12, and had a predisposition for segmental instability, such as facet joint disruption and/or arthrosis.[1,4,7] Furthermore, the existence of OLF exacerbated the rapid onset of thoracic myelopathy.

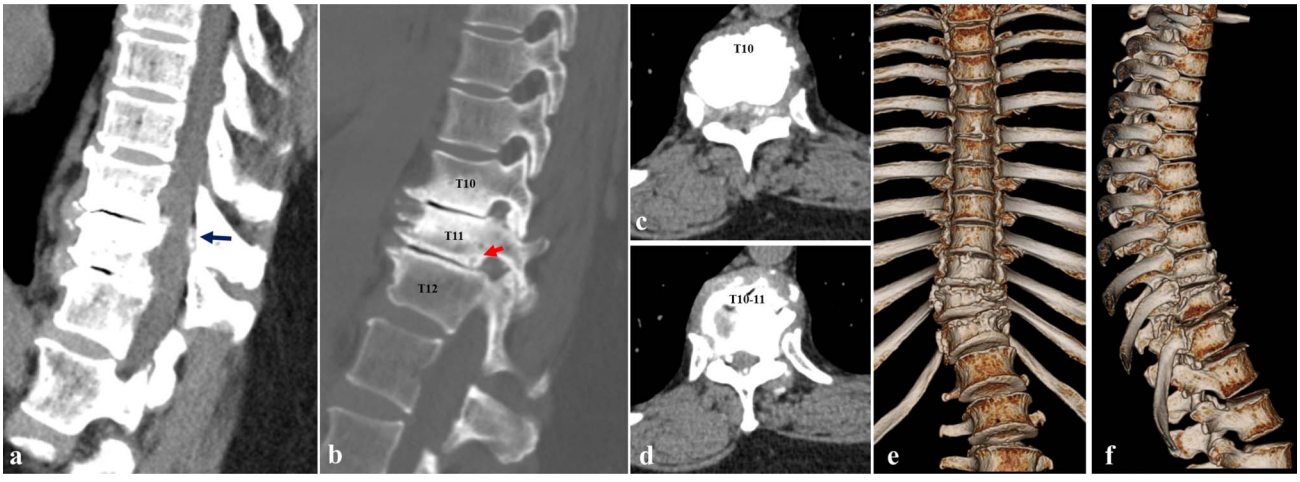
Figure 1



Anteroposterior radiographic view showing lumbar scoliosis with a rotational change of L1–L3 (Nash-Moe scale grade 1) and sclerotic change of vertebral end plates at T10–T12 (**A**). Lateral radiographic view showing degenerative changes of intervertebral disk at T10–T12 and loss of lordosis and kyphosis (**B**).

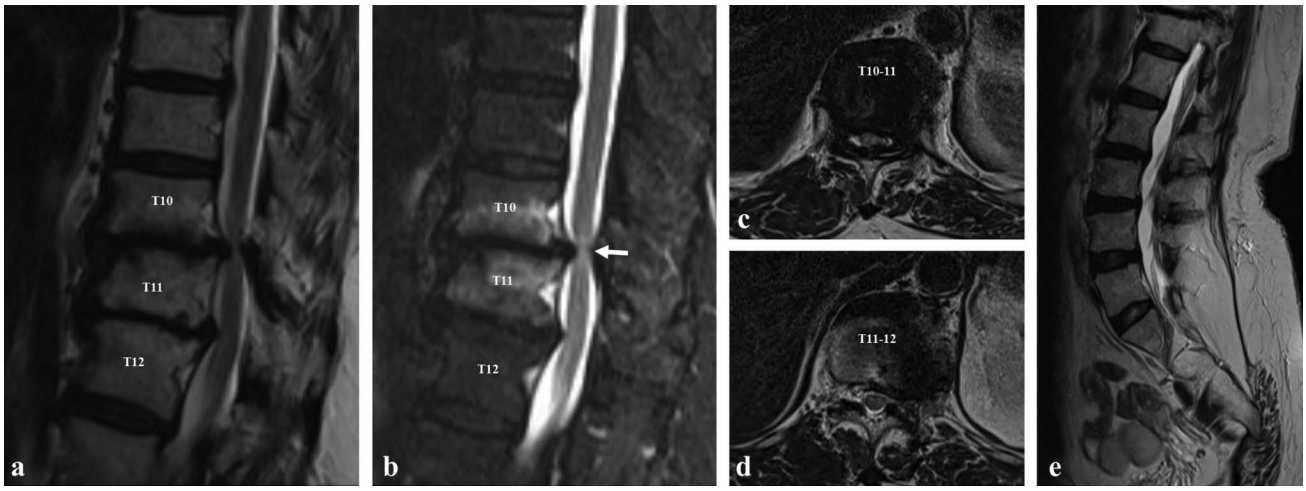
There are few cases accompanied by segmental instability of degenerative spondylolisthesis–associated thoracic myelopathy. Our case distinctively showed degenerative spondylolisthesis at T10–T12 with lumbar scoliosis. However, we did not capture the dynamic views to prove unstable spondylolisthesis; it is a limitation on this study, so dynamic view may be required such as this case. Scoliosis of CP is not only commonly associated with C-shaped but also prevalent, affecting 41% of individuals with CP.[12] The severity of scoliosis is strongly correlated with gross motor function, with impacted patients frequently exhibiting limb-length discrepancies and postural asymmetry.[11,12] These factors contribute to increased loads on spinal alignment in this case. Moreover, thoracic hypokyphosis associated with the scoliotic curve imposes an additional load, which may have contributed to the

Figure 2



Lateral view of spinal CT showing vacuum phenomena and height decrease of the intervertebral disk at T10–T12 and calcific change of the yellow ligament at T10–T11 (**A**). Degenerative spondylolisthesis with segmental instability at T10–T12 (**B**). Axial view of spinal CT demonstrating facet arthrosis and marginal osteophytes (**C** and **D**). 3-dimensional reconstruction CT views (**E** and **F**).

Figure 3

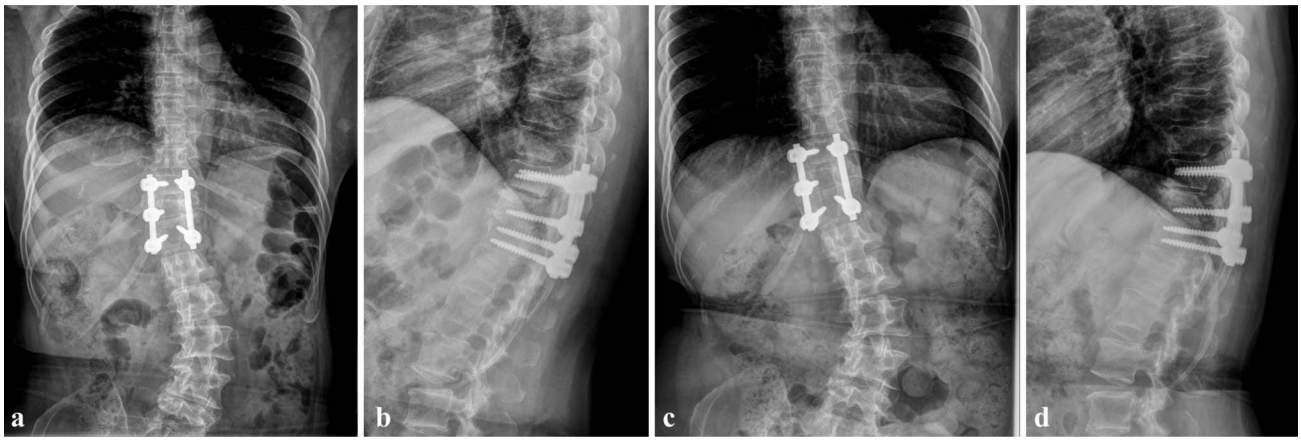


Images showing severe cord compression at T10–T11, increased high signal intensity on T2-weighted imaging, and ligament hypertrophy at T10–T12, indicating thoracic myelopathy (**A**–**D**). No specific findings for degenerative changes were observed on the lumbar lesions (**E**).

development of thoracic myelopathy.[13] For surgical decision making, deformity correction would have been ideal given the scoliotic and hypokyphotic curves. However, based on the patient's preference, we focused on symptom relief. Close observation after surgical treatment was necessary because of the preexisting spinal deformity. At the 2-year follow-up, we observed radiologic improvements in end plate sclerosis in the T10-T12 region. In cases of preexisting scoliosis, regular long-term follow-up is necessary to monitor deformity progression.

The cases in the literature were mainly caused by facet joint laxity and intervertebral disk degeneration.[1,4,7,8] The micromotion from segmental instability compressed the relatively narrow thoracic spinal cord, producing thoracic myelopathy.[6] Interestingly, our case showed lumbar scoliosis as a sequela of CP, which increased the vulnerability to lower thoracic intervertebral disk degeneration. The excessive load on the thoracolumbar junction from lumbar scoliosis aggravates degenerative changes in lower thoracic region, leading to segmental instability and degenerative spondylolisthesis at T10–T12.[9,10] In addition, the presence of OLF is considered one of the main factors contributing to the development of thoracic

Figure 4



Postoperative follow-up radiographs showing total laminectomy and posterior instrumented fusion at T10–T12 performed under the diagnosis of thoracic myelopathy (**A** and **B**). The radiographs at the 2-year follow-up were relatively stable, and end plate sclerotic changes at T10, T11, and T12 were improved (**C** and **D**).

myelopathy. Therefore, our case suggests that it is important to prevent devastating conditions such as thoracic myelopathy by assessing the lower thoracic region when lumbar scoliosis is present. As in our case, the surgical treatment of thoracic degenerative spondylolisthesis is decompression and short-segment instrumented fusion.[1,4,6-8] Meanwhile, 2-year follow-up radiological findings presented an improvement in end plate sclerosis but progression of the scoliotic curve. It is proposed that mechanical force and loading on the patient have resulted in degenerative changes in thoracolumbar region, rather than the further progression of a scoliotic curve. Meanwhile, we did not include patient-reported outcome measures, such as disability scores, pain scores, and modified Japanese Orthopaedic Association scores, which would have provided a more comprehensive understanding of the diagnostic process and postoperative recovery. This represents a limitation of our case report.

Conclusion

Thoracic myelopathy in our case was caused by segmental instability, including multilevel lower thoracic degenerative spondylolisthesis, the presence of OLF, and lumbar scoliosis. This rare case, involving multiple complex spinal conditions, provides valuable insights into the development of thoracic myelopathy. Therefore, special attention is required when the consideration of lower thoracic cord compression in patients presenting with diffuse lower extremity weakness in the setting of lumbar deformities.

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