



Reversal of Paraplegia After Anterior Decompression in a Patient Underwent Posterior Decompression and Fusion for Thoracic Ossification of Posterior Longitudinal Ligament

Posterior Longitudinal Ligamentin Torasik Ossifikasyonu için Posterior Dekompresyon ve Füzyon Yapılan Bir Hastada Anterior Dekompresyon Sonrasında Paraplejinin Geri Dönmesi

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ABSTRACT

This report describes a rare clinical entity, thoracic myelopathy due to ossification of the posterior longitudinal ligament (OPLL), and its management.

A 40-year-old woman presented with thoracic myelopathy due to OPLL, extending from T2–T9. We performed a posterior laminectomy and instrumented fusion. However, postoperative paraplegia occurred within 36 h post-surgery. Emergent anterior decompression and interbody fusion was performed via the trans-thoracic approach. Neurological deterioration was reversed following this anterior procedure. Posterior decompression and instrumented fusion for thoracic OPLL is less technically demanding and presents a lower risk of neurological complications. However, some controversies remain regarding the prevalence and management of postoperative neurological deterioration associated with this technique. Our patient showed recovery with subsequent anterior decompression when paraplegia occurred after posterior decompression and fusion to treat thoracic OPLL. Additional anterior decompression should be considered when posterior decompression and fusion lead to neurological deterioration.

KEYWORDS: Fusion, Laminectomy, OPLL, Postoperative paraplegia, Thoracic

ÖZ

Bu rapor, posterior longitudinal ligament ossifikasyonu (OPLL) nedeniyle torasik myelopati şeklinde nadir bir klinik durumu ve tedavisini tanımlamaktadır.

40 yaşında bir kadın T2–T9 arasında uzanan OPLL'nin sebep olduğu torasik myelopatiyle geldi. Posterior laminektomi ve enstrümantasyonlu füzyon gerçekleştirdik. Ancak ameliyattan 36 saat sonra parapleji gelişti. Transtorasik yaklaşımla acil anterior dekompresyon ve cisimler arası (interbody) füzyon yapıldı. Bu anterior işlem sonrasında nörolojik bozulma geri döndü. OPLL için posterior dekompresyon ve enstrümantasyonlu füzyon teknik olarak daha kolaydır ve nörolojik komplikasyonlar açısından daha az risklidir. Ancak bu teknikle ilişkili postoperatif nörolojik bozulmanın prevalansı ve takibi ile ilgili bazı tartışmalar devam etmektedir. Hastamız torasik OPLL tedavisi için posterior dekompresyon ve füzyondan sonra parapleji gelişince sonraki anterior dekompresyonla iyileşti. Posterior dekompresyon ve füzyon nörolojik bozulmaya neden olduğunda ek anterior dekompresyon yapılması düşünülmelidir.

ANAHTAR SÖZCÜKLER: Torasik, OPLL, Laminektomi, Füzyon, Postoperatif parapleji

INTRODUCTION

Ossification of the posterior longitudinal ligament (OPLL) is a well-known cause of cervical myelopathy (14). It also occurs, although less commonly, in the thoracic spine, causing severe myelopathy (15). When thoracic myelopathy caused by OPLL does not respond to conservative management, surgical intervention is considered. To date, a variety of surgical methods have been introduced to manage thoracic OPLL: posterior decompressive laminectomy or laminoplasty

alone (12); posterior decompression and instrumented fusion (15); anterior decompression (4); posterior and anterior decompression (13); and circumferential decompression through a single posterior approach (11). However, the surgical outcome of thoracic OPLL has compared unfavorably with that of cervical OPLL. Yamazaki et al. introduced the surgical procedure of posterior decompression with instrumented fusion for the management of thoracic OPLL (15). They concluded that a considerable degree of neurological recovery

was obtained by posterior decompression with instrumented fusion, and the rate of postoperative complications was extremely low with that procedure. Although posterior decompression and fusion is less technically demanding and has a lower risk of neurological complications, this technique provides indirect decompression and the clinical recovery rate tends to be lower than anterior decompression and fusion (8). Unfortunately, we still encounter postoperative paraplegia after posterior decompression and fusion for thoracic OPLL. Here, we report a case of thoracic myelopathy due to thoracic OPLL, in which paraplegia occurred after posterior decompression and instrumented fusion and was reversed by subsequent anterior decompression and fusion.

CASE REPORT

A 40-year-old woman with a 2-month history of progressive gait disturbance was admitted to our institute. She could stand and walk with assistance for 10–20 meters but complained of bilateral lower extremity motor weakness. A manual muscle test (MMT) found her iliopsoas to be at 4/5, quadriceps at 4/5, tibialis anterior at 4/5, extensor hallucis longus at 5-/5-, and flexor hallucis longus at 5/5. Knee and ankle jerks were increased and ankle clonus was present. The preoperative Japanese Orthopaedic Association (JOA) score was 5 (3).

Computed tomography (CT) scan showed OPLL at T2-9 (Figure 1A, B), and magnetic resonance imaging (MRI) showed severe anterior compression of the spinal cord due to the ossification mass and a spinal cord signal change at T5-6 and T6-7 (Figure 1C).

We performed a T5-7 posterior laminectomy and T4-8 posterior instrumented fusion with pedicle screw fixation (Figure 2A). No specific incident or spinal cord irritation occurred during the operation. After surgery, the patient showed neither improvement nor worsening of the motor weakness, which remained at 4/5; however, she reported feeling better about bearing weight on her lower extremities. At 36 h after the operation, her neurological signs rapidly worsened, and she complained of paraplegia of her lower extremities. Her MMT was 0/0 in both lower extremities, and sensory tone was decreased to sensory point 1 in the American Spinal Injury Association (ASIA) impairment scale from sensory point 2 at the preoperative and immediate postoperative state.

We performed emergency re-operation as the neurological changes appeared to result from postoperative hematoma formation. However, there were no findings of epidural hematoma or tissue that might be compressing the spinal cord posteriorly. Hence, we decided to perform an anterior decompression. First, we removed the inserted pedicle screws at T6 and T7 and inserted additional pedicle screws at T9, connecting with the previously placed T4 and T5 screws. Next, anterior decompression was done by partial corpectomy of T6 and T7 using the Left transthoracic approach. Finally, interbody fusion of T6-8 was done using a resected rib graft (Figure 2B).

After the 2nd operation, the patient could raise her left leg and flex her left ankle. Right-sided motor weakness was not improved, but sensation was improved vs. the preoperative

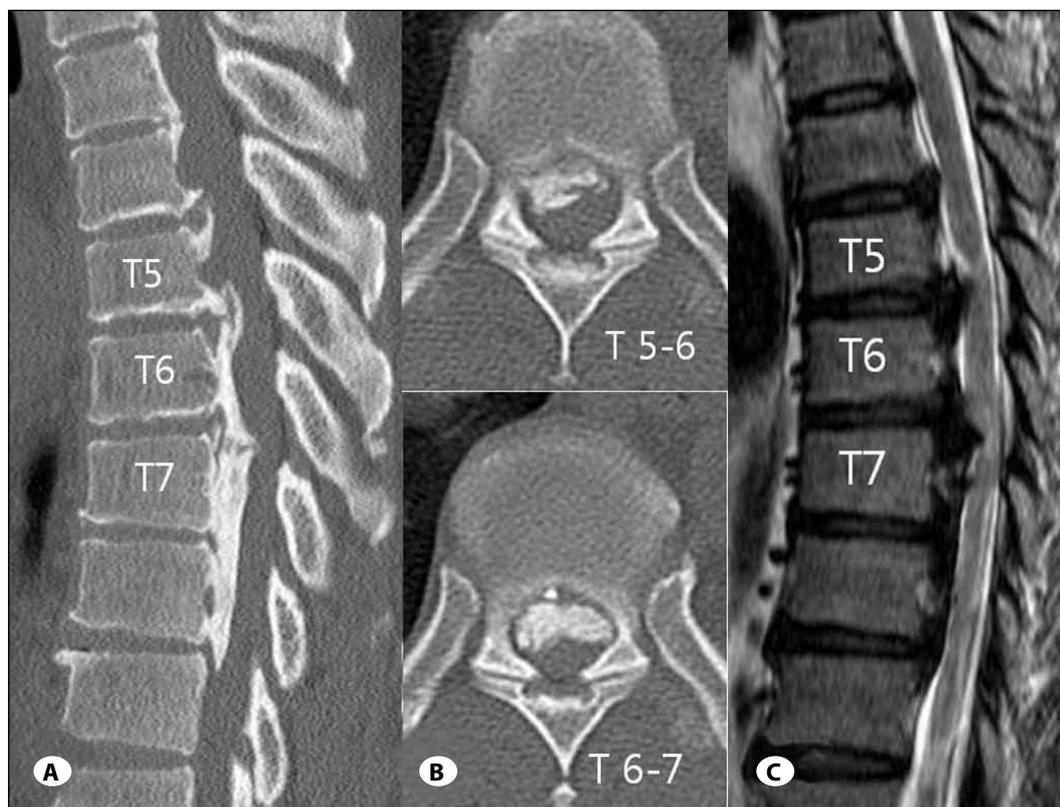


Figure 1:
A) Preoperative midsagittal and **B)** axial CT scans showing compression of the spinal cord anteriorly by a beak-type continuous ossification of the posterior longitudinal ligament (OPLL). **C)** An MRI scan showed severe compression of the spinal cord anteriorly due to the ossification mass and spinal cord signal change at T5-6 and T6-7.

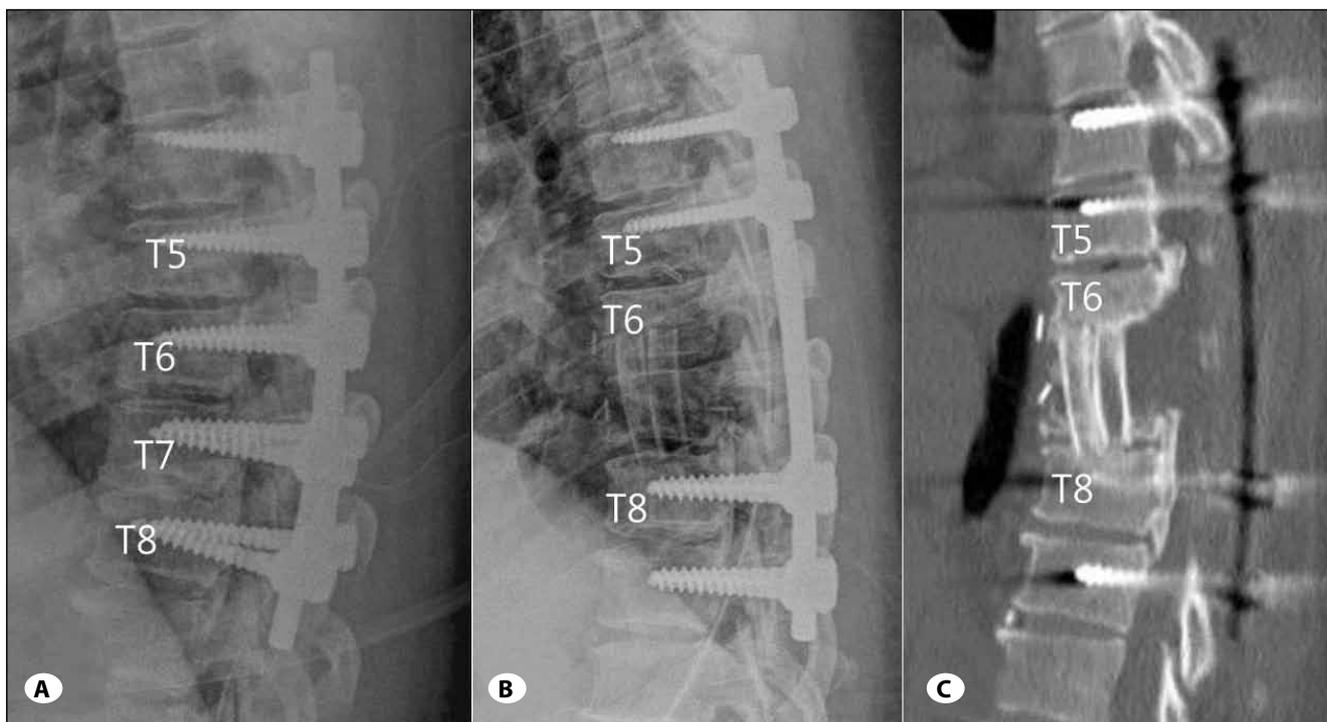


Figure 2: **A)** Postoperative lateral radiographs of a T5-7 posterior laminectomy and T4-8 posterior instrumented fusion. **B)** Second operation performed at T4-5 and T8-9: instrumented fusion, anterior decompression and interbody fusion of T6-8. **C)** Postoperative one-year follow-up midsagittal CT scans showing complete union and decompressed OPLL mass.

state. Two weeks after the operation, her MMT had improved to 3/5 on the right and 4 to 5/5 on the left lower extremity. She could stand with assistance at 1 month and walk with a cane at 3 months.

The 1-year follow up CT showed complete fusion of the implanted rib graft (Figure 2C). The patient could walk without any assistance. The follow up JOA score was improved to 9. Informed consent was obtained for publishing these results.

DISCUSSION

Previous reports have shown that the results of surgery for thoracic myelopathy caused by OPLL are less favorable than the results for cervical OPLL surgery (4, 15). These unfavorable outcomes are related to the following anatomic and pathophysiological factors inherent in the thoracic spine: the thoracic spine is kyphotic and posterior decompression is therefore less effective as backward movement of the spinal cord by indirect decompression is anatomically limited; the thoracic spinal cord is more vulnerable because of its relative avascularity (6); and the presence of strong adhesion of the ossified ligament to the ventral dural sac increases the risk of spinal cord injury during decompression procedures. A number of surgical methods have been introduced to manage thoracic OPLL in consideration of the surgical approach, fusion or non-fusion, and the use or absence of instrumentation. An anterior approach has been recommended by some authors, as it allows for direct decompression by excising or floating the OPLL (4). However, the anterior transthoracic

approach is technically demanding and may place more surgical stress on patients than a posterior approach. Takahata et al. examined the clinical outcomes of circumferential spinal cord decompression through a posterior approach in thoracic OPLL (11). Although this procedure enables effective neurological recovery, a high rate of complications (33%) including neurological deterioration has been observed.

Using the posterior approach for thoracic OPLL, decompression surgery alone often results in neurological deterioration after surgery. The reason is that decompression destabilizes the thoracic spine and the spinal cord is therefore more prone to compression via ventral thoracic OPLL (7, 8, 15, 16). To reduce the incidence of postoperative neurological deterioration, Yamazaki et al. recommended posterior decompression with instrumented fusion for thoracic OPLL, rather than decompression alone (15). In their study, none of the 24 patients who underwent posterior decompression and fusion developed persistent paralysis after surgery, and only 1 patient (3.85%) had transient paralysis immediately after surgery. Further, they suggest that additional anterior decompression surgery is not desirable during the recovery period (16). However, Matsumoto et al. showed that postoperative neurological deterioration was observed in 27.5% patients after posterior decompression and fusion surgery in thoracic OPLL patients (8). Our case also showed neurological deterioration following posterior decompression and fusion.

Several studies have reported that anterior decompression through thoracotomy does not necessarily produce favorable

results when performed as a rescue surgery on thoracic OPLL patients, whose myelopathy worsens after laminectomy (10). Worsening myelopathy might indicate severe damage to the spinal cord resulting from the laminectomy, in which case the spinal cord may not withstand an additional anterior decompression procedure. However, a multicenter analysis has reported that neurological recovery was achieved in all 6 cases of secondary intervention. Among the 14 patients who did not undergo surgical intervention for neurological deterioration, 11 experienced spontaneous neurological recovery. Hioki et al. also reported that thoracic paraparesis after laminectomy recovered when anterior decompression was performed (5). In our case, paraplegia occurred 36 h after the operation. Partial recovery occurred immediately after the subsequent anterior decompression and a near-complete recovery was achieved by the time of the final follow-up.

Spinal cord circulation insufficiency or micromotion of the operative segment after decompression may be associated with postoperative paraplegia even after combined posterior instrumentation is performed (2, 9, 16). Some authors speculate that postoperative neurological deterioration may be attributable to the reperfusion mechanism rather than to iatrogenic spinal cord injury (1).

Our patient recovered from paraplegia caused by posterior decompression and fusion performed to treat thoracic OPLL after anterior decompression was performed. We recommend that an additional anterior decompression procedure be considered when posterior decompression and fusion lead to neurological deterioration.

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