Identification and Surgical Treatment of Primary Thoracic Spinal Stenosis

John R. Dimar II, MD, Kelly R. Bratcher, RN, CCRP, Steven D. Glassman, MD, Jennifer M. Howard, MPH, and Leah Y. Carreon MD, MSc

Abstract

We report the surgical treatment results for 7 patients (4 men, 3 women; mean age, 49 years) who presented with myelopathy caused exclusively by primary thoracic spinal stenosis, predominantly in the lower thoracic spine. (Patients with concurrent ascending lumbosacral degenerative disease were excluded.) All patients received extensive nonoperative treatment before referral to our center. Surgical treatment consisted of wide posterior decompression and instrumented fusion (5 cases), anterior vertebrectomy and fusion (1), and anterior vertebrectomy with autograft strut followed by wide posterior decompression and instrumented fusion (1). Mean operative time was 313 minutes, mean blood loss was 944 mL, and there were no major postoperative complications.

Minimum follow-up was 2 years. Five patients had significant improvement in myelopathy and were ambulating normally, 1 had modest improvement in ambulation, and 1 remained wheelchair-bound. All patients achieved solid radiographic fusions.

After presenting these case studies, we review the current literature on treatment effectiveness. Primary thoracic spinal stenosis should be considered in patients who present with isolated lower extremity myelopathy, particularly when no significant pathologic findings are identified in the cervical or lumbosacral spine. Expedient wide decompression with concurrent instrumented fusion is recommended to prevent late development of spinal instability and recurrent spinal stenosis.

Dr. Dimar is Associate Professor, Department of Orthopaedic Surgery, University of Louisville School of Medicine, Louisville, Kentucky, and Attending Surgeon, Leatherman Spine Center, Louisville, Kentucky.
Ms. Bratcher is Senior Clinical Research Coordinator, Leatherman Spine Center, Louisville, Kentucky.
Dr. Glassman is Associate Professor, Department of Orthopaedic Surgery, University of Louisville School of Medicine, Louisville, Kentucky, and Attending Surgeon, Leatherman Spine Center, Louisville, Kentucky.
Ms. Howard is Research Assistant, Leatherman Spine Center, Louisville, Kentucky.
Dr. Carreon is Clinical Research Director, Leatherman Spine Center, Louisville, Kentucky.

Address correspondence to: Leah Y. Carreon, MD, MSc, Leatherman Spine Center, 210 E Gray St, Suite 900, Louisville, KY 40202 (tel, 502-584-7525; fax, 502-584-6851; e-mail, lcarreon@spinemds.com).

Am J Orthop. 2008;37(11):564-568.

Spinal stenosis is spinal canal narrowing that causes compression of the neural elements. Cervical stenosis typically results in upper extremity radicular symptoms, myelopathy, or both; lumbar spinal stenosis results in lower extremity radicular symptoms or neurogenic claudication; thoracic spinal stenosis (TSS) is unique in that it often produces only myelopathy.

Isolated TSS is an extremely rare condition[1-11] and thus is seldom considered as the cause of myelopathy, leading to a delay in diagnosis. The precise etiology of TSS is unknown. Certain cases appear to be of congenital origin (symptoms develop early in life), whereas others involve age-related degeneration of the discs, facets, and ligamentous structures. Other reported causes of TSS are ossification of the posterior longitudinal ligament (OPLL), Paget's disease, ankylosing spondylitis, acromegaly, achondroplasia, osteochondrodystrophy, diffuse idiopathic skeletal hyperostosis (DISH), and various forms of rickets.[8,9,11]

In this article, we report the surgical treatment results for 7 patients who presented with myelopathy caused exclusively by primary TSS. We also review the current literature on treatment effectiveness.

Materials and Methods

We reviewed the charts of 7 patients (4 men, 3 women; mean age, 49 years; age range, 36-80 years) who had been surgically treated for primary TSS between 1998 and 2006 (Table). All patients were followed for a minimum of 2 years (range, 24-68 months). All cases presented with significant myelopathy and a delay in diagnosis. We excluded cases that involved ascending lumbosacral degenerative disease with spinal stenosis, whether primary or secondary to adjacent-level degeneration from a prior decompression and fusion.

Mean duration of preoperative myelopathic symptoms was 13 months. Before referral to our center, all patients received extensive preoperative conservative treatment, which consisted of pain medication, muscle relaxants for spasticity, physical therapy, bed rest, epidural injections, and chiropractic care.

For all patients, we obtained plain anteroposterior radiographs of the thoracic spine, magnetic resonance imaging (MRI) scans, and water-soluble isoniazide myelogram followed by axial computed tomography (CT) scan. TSS was predominantly in the lower thoracic spine, with pathologic narrowing at T11–T12 (4 cases), T10–T11 (2 cases), and

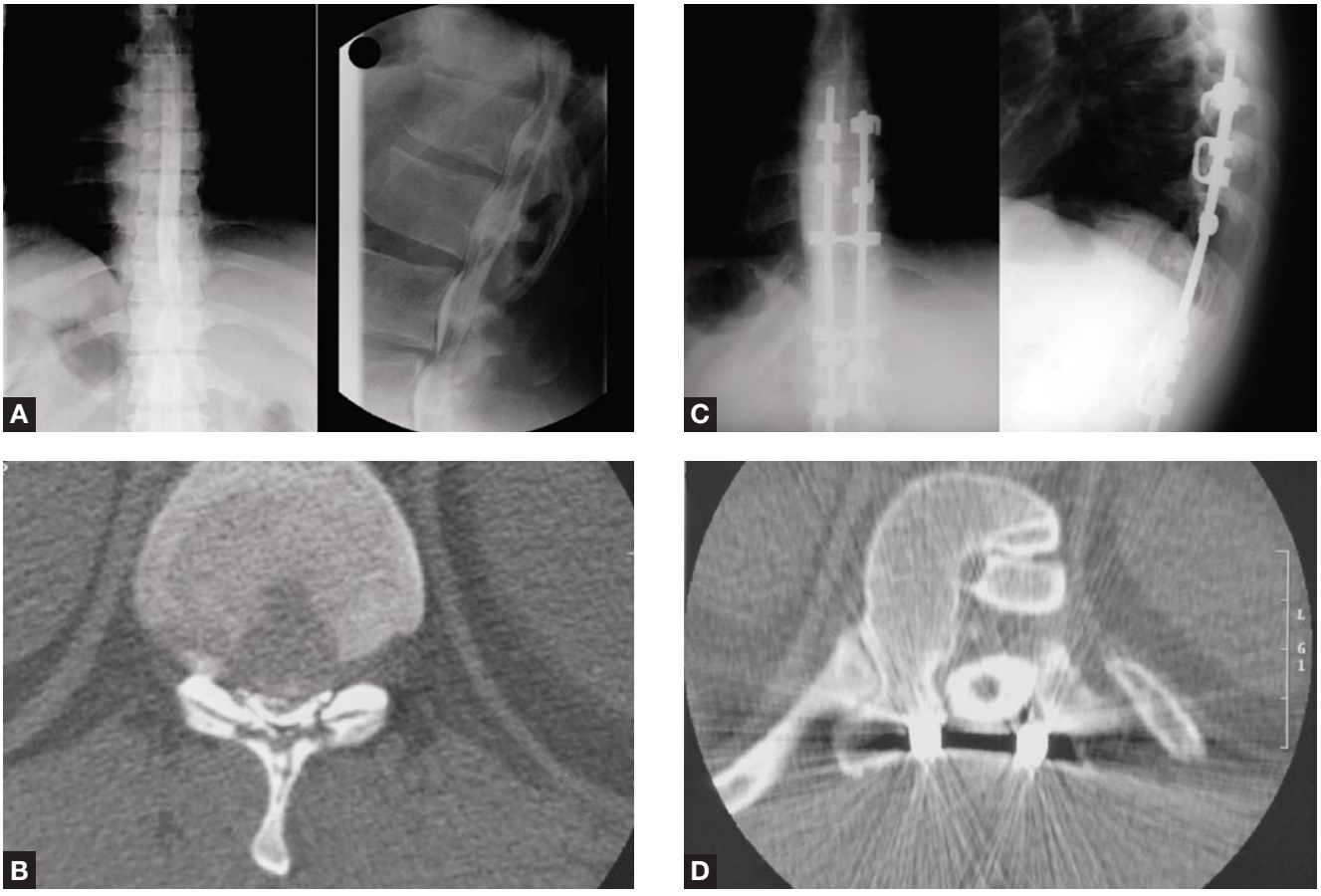


Figure 1. (A) Anteroposterior and lateral myelograms show spondylitic changes and segmental kyphosis with stenosis at T11–T12. (B) Axial computed tomography scan shows severe spinal stenosis at T11–T12. (C) Anteroposterior and lateral radiographs after posterior decompression and instrumented fusion. (D) Axial computed tomography scan shows anterior decompression and restoration of anterior and posterior canal diameter.

T9–T10 (1 case). Myelographic blocks were caused by degenerative discs, shortened pedicles, facet hypertrophy, ligamentous and capsular hypertrophy, or a combination of these findings.

Surgical treatment to correct spinal canal narrowing consisted of wide posterior decompression and instrumented fusion (5 cases), anterior vertebrectomy and fusion (1), and anterior vertebrectomy with autograft strut followed by wide posterior decompression and instrumented fusion (1). As is routine, all patients received perioperative antibiotics, used a cell saver, and were monitored with somatosensory evoked potentials. Mean operative time was 309 minutes (range, 170-527 minutes), and mean operative blood loss was 944 mL (range, 300-1900 mL). Mean duration of postsurgical hospitalization was 7 days (range, 4-13 days); there was a subsequent period of inpatient rehabilitation.

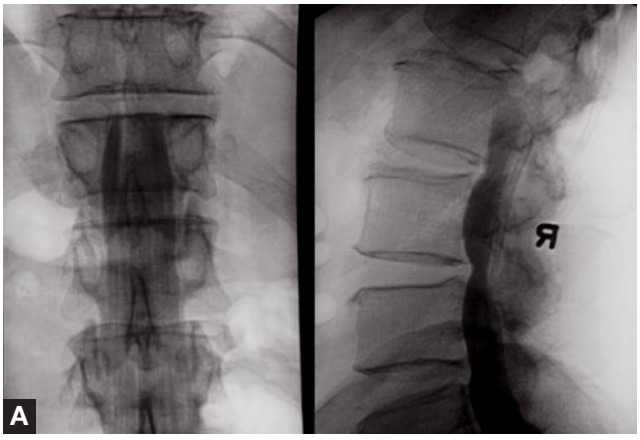
Case Studies

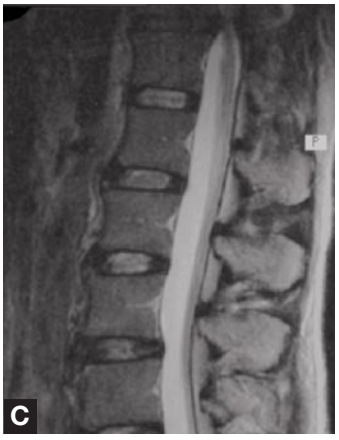
Case 1. A 35-year-old man with progressive leg weakness was referred to our institution. He had been treated conservatively by his family doctor and a chiropractor but after 4 months was referred to a neurosurgeon, who performed decompression on the left side. Symptoms improved slightly, but weakness and numbness continued progressing in both legs, without loss of bladder or bowel function. Physical examination at our institution revealed bilateral lower extremity weakness, sustained clonus, spasticity, loss of proprioception, broad-based gait, and positive Romberg sign. Neurologic status was graded Frankel D.

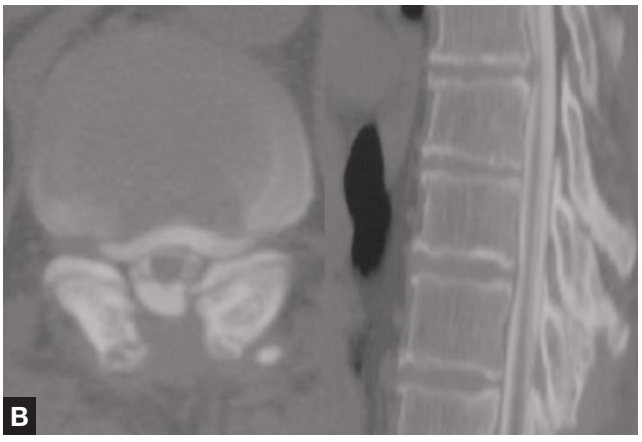
MRI and CT myelogram showed severe spinal stenosis at T11–T12, changes consistent with the patient's recent surgery (Figure 1). The patient now underwent T11 vertebrectomy and autograft strut grafting for the large anterior osteophyte at T11–T12, plus posterior wide decompression (including the facets) and posterior instrumented autograft fusion (T9–L2).

The postoperative course was uneventful, and there were no complications. Lower extremity muscle strength, sensation, and proprioception improved, and gait returned to normal. Radiographs obtained at 2 years showed solid fusion, and the patient's neurologic condition was graded Frankel E.

Case 2. A 45-year-old man presented 4 months after sustaining a lifting injury. He felt a sharp pain in the lower back and developed progressive numbness and diffuse tingling through the lower extremities. He also noted difficulty ambulating, coordination problems, and generalized weakness, without loss of bowel or bladder function. A







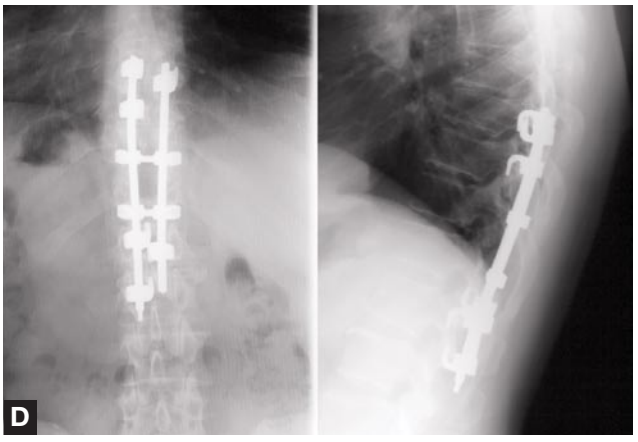


Figure 2. (A) Anteroposterior and lateral myelograms show myelographic block at T11–T12. (B) Axial and sagittal reconstruction computed tomography scan shows residual stenosis at T11–T12. (C) Sagittal magnetic resonance imaging shows high-grade stenosis at T11–T12. (D) Postoperative anteroposterior and lateral radiographs show wide compression at T11–T12 with posterior instrumented fusion.

neurosurgeon recommended conservative care, including epidural blocks and physical therapy. On referral of the patient to our institution, physical examination revealed a multidermatomal pattern of decreased sensation through the right lower extremity: right-side muscle weakness with marked atrophy of the quadriceps and gastrocnemius muscles, bilateral Babinski sign, sustained clonus, and hyperreflexia of the Achilles tendon on the right. Overall neurologic function was graded Frankel D. Plain radiographs were normal. MRI revealed severe spinal stenosis at T11–T12, confirmed by myelogram and CT scan, which showed a myelographic block at T11–T12 caused by shortened pedicles and facet hypertrophy (Figure 2). As anterior compression was minimal, selective wide posterior decompression (T10–T12) and then instrumentation autograft fusion (T8–L2) were performed.

The postoperative course was uneventful. Neurologic function remained stable, muscle strength increased in the right lower extremity, and sensory function and coordination improved. Painful dysesthesias continued in the lower extremities, but postoperative CT myelogram showed no residual stenosis. The pain was thought to result from a chronic spinal cord injury. Two years after surgery, instrumentation was removed, fusion was solid, and there was no residual stenosis. Six years after surgery, there was no deterioration in motor strength, but the patient complained of a chronic burning pain in the lower extremity. Final neurologic status was graded Frankel E.

Results

None of the 7 patients had any major postoperative complications. Five patients had significant improvement in myelopathy and were ambulating normally, 1 had modest improvement in ambulation, and 1 remained wheelchair-bound. The 2 patients who had incontinence before surgery regained control of their bowel and bladder function. All patients achieved solid radiographic fusions. One patient required instrumentation removal and exploration of the fusion, which was found to be solid and to have no residual stenosis.

Discussion

Spinal stenosis is a widely recognized condition that predominantly involves the cervical and lumbosacral spine. The precise etiology remains obscure, but there appears to be a developmental defect that may remain quiescent for decades until acquired degenerative changes occur within the discs and facets. These factors produce critical narrowing of the central spinal canal and foramen, and this narrowing causes the classic neurologic symptoms associated with the disease.[12] Cervical stenosis classically presents with either radicular symptoms secondary to foraminal stenosis or myelopathy resulting from the central stenosis. Early anatomical studies and later CT measurements have established that the normal anteroposterior cervical spinal canal diameter is at least 12 mm. Lumbosacral stenosis commonly presents with radicular symptoms, spinal claudication, or, seldom, acute cauda equina syndrome. Lumbosacral anatomical

Table. Summary of Results

Case No	Age/ Sex	Duration of PreOp Symptoms (months)	Type of Surgery	Surgical Time	Estimated Blood Loss (mL)	Length of Stay (days)	Complication	Length of Follow-Up (months)	PreOp Ambulatory Status	Outcome
1	36/M	10	Anterior vertebrectomy T10-T12, Wide posterior decompression, posterolateral instrumented fusion T8-L1	8h 47m	1000	7	None	67	Myelopathic, ambulating with cane	Ambulating independently
2	51/F	4	Anterior vertebrectomy and fusion T9-T10	2h 50m	300	4	None	53	Myelopathic, ambulating with walker	Ambulating independently
3	80/F	6	Wide posterior decompression, posterolateral instrumented fusion T7-L1	3h 52m	600	9	None	66	Myelopathic, ambulating with walker, incontinent	Ambulating independently
4	45/M	6	Wide posterior decompression, posterolateral instrumented fusion T9-L1	3h 37m	700	5	None	68	Myelopathic, ambulating with cane	Ambulating independently
5	53/M	7	Wide posterior decompression, posterolateral instrumented fusion T10-T11	3h 55m	700	5	None	37	Myelopathic, ambulating with walker, incontinent	Wheelchair
6	69/F	48	Wide posterior decompression, posterolateral instrumented fusion T11-L4	4h 49m	950	13	Ileus	40	Myelopathic, ambulating with walker	Ambulating independently
7	37/M	7	Wide posterior decompression, posterolateral instrumented fusion T11-T9	8h 12m	1900	7	None	24	Myelopathic, ambulating with cane	Ambulating independently

studies and CT measurements have established normal canal diameter as ranging from 12 to 17 mm.[12-14]

TSS, by contrast, is quite rare, having been described only in case reports and case series reporting surgical outcomes.[1-11] Although several generalized skeletal diseases have been implicated in the etiology of secondary spinal stenosis (eg, Paget's disease, achondroplasia and other forms of dwarfism, DISH, OPLL, rickets, acromegaly, ankylosing spondylitis), the etiology of primary TSS is similar to that of cervical and lumbosacral stenosis (ie, developmental narrowing combined with acquired degenerative changes).[12,13] In addition, dynamic spinal stenosis may play an important role, as it results in anterior, posterior, lateral, and rotatory instability, which may aggravate spinal canal narrowing.

Jaspan and colleagues[4] and Ilkko and colleagues[15] reported on patients with severe myelopathy secondary to severe primary TSS treated with surgical decompression. Barnett and colleagues[1] reported on 6 myelopathic patients presenting with isolated primary TSS (diagnosed with CT myelography and MRI) who had neurologic improvement after being treated with surgical decompression. No mention of long-term follow-up or the possibility of symptom recurrence was made.

Yamamoto and colleagues[11] reported on 7 patients with primary TSS who presented with myelopathy and were treated with wide decompression without fusion. These patients were followed for 5 months to 6 years. Two patients required repeat decompression for symptom recurrence.

Epstein and Schwall[2] reported on the surgical treatment and neurologic outcomes of 9 patients with primary TSS and presented an excellent literature review featuring another 19 patients. The diagnosis, made with CT myelogram, MRI, or both, was defined as a congenitally narrowed spinal canal (<10 mm). Mean duration of symptoms was 2.3 months, and mean patient age was 53. In all but 2 patients, neurologic symptoms improved after wide decompression. There is no mention of follow-up duration, and 7 patients had concurrent decompression to the sacrum. Palumbo and colleagues[9] reported on the surgical treatment and 2- to 9-year follow-up of 12 cases of TSS. Surgical decompres-

sion was performed in the thoracic spine in 4 cases and in the thoracolumbar spine and sacrum in 8 cases. In 5 cases, early improvement deteriorated because of recurrent stenosis, instability, or deformity.

The cases described by both Epstein and Schwall[2] and Palumbo and colleagues[9] most likely are not isolated primary TSS but, instead, ascending lumbosacral spinal stenosis in which progressive, ascending acquired spinal stenosis in the lumbosacral spine extends proximally into the lower thoracic spine. Their and our results confirm the common delay in diagnosis and the need for surgical treatment in patients with primary TSS. Patients also need to be informed that they may not improve neurologically. Long-term follow-up is also necessary, as clearly demonstrated by the time-related degradation of surgical results—challenging previous findings that spinal instability or deformity does not develop after wide decompression.

Our results confirm the previously reported variable nature of neurologic recovery in these cases but also the appropriateness of early surgical intervention and the importance of long-term follow-up. Concurrent stabilization and fusion prevent development of instability or deformity and recurrence of myelopathy (reported in earlier case series). Given these findings, the usual recommendation regarding wide posterior decompression with facet removal is to concurrently perform posterior fusion and instrumentation in order to avoid potential late instability and recurrence of secondary TSS.

Clinical Evaluation of Myelopathy. Primary TSS is a rare cause of myelopathy, and the diagnosis is often delayed, with the result being potential worsening of the neurologic injury. Clinical evaluation of myelopathy requires a high index of suspicion and careful clinical examination to differentiate cervical and thoracic compression. Cervical myelopathy usually causes both upper and lower extremity weakness, imbalance, and loss of sensation and proprioception and may have the classic upper extremity pathologic reflexes (eg, positive Hoffmann reflex, reverse radial reflex) and the pathologic lower extremity reflexes (eg, clonus, positive Babinski sign). These findings should be contrasted to those of thoracic myelopathy, which usually presents with only lower extremity weakness, numbness, loss of proprioception, bowel and bladder dysfunction (rare), and clonus or positive Babinski sign (possible). Of importance, when stenosis is at the lowest levels of the thoracic canal, the tip of the conus (L1–L2) may have mixed lesions because of both cord compression and concurrent compression of the anterior horns of the nerve roots forming the cauda equina, thus presenting with some radicular symptoms. Careful attention to these clinical findings will guide the clinician in ordering diagnostic testing of the appropriate area of the spine. Consequently, in a patient presenting with myelopathy, strong consideration should be given to the diagnosis of primary TSS, particularly if no significant pathologic findings are identified in the cervical or lumbosacral spine.

Diagnostic Testing. MRI was useful in screening the spine for TSS, but a myelogram followed by a finely cut sagittal and coronal reconstructed CT scan is superior in identifying areas of stenosis with a higher degree of precision, as the stenosis often involves bony structures, such as shortened pedicles, hypertrophied facets, or, occasionally, ossification of the posterior longitudinal ligament or the ligamentum flavum.

Management of Primary Thoracic Spinal Stenosis. Once primary TSS is identified, expedient wide decompression with concurrent instrumented fusion is recommended to prevent late development of spinal instability and recurrent spinal stenosis. Long-term follow-up is essential, as many of the postoperative complications noted in this review and in the literature may take many years to manifest.

Authors' Disclosure Statement

Dr. Dimar and Dr. Glassman wish to note that they receive royalties, research grant support, and consulting fees from Medtronic Sofamor Danek (Memphis, TN) with no actual conflict of interest in relation to this article. The other authors report no actual or potential conflict of interest in relation to this article.

References

1. Barnett GH, Hardy RW Jr, Little JR, Bay JW, Sypert GW. Thoracic spinal canal stenosis. *J Neurosurg*. 1987;66(3):338-344.
2. Epstein NE, Schwall G. Thoracic spinal stenosis: diagnostic and treatment challenges. *J Spinal Disord*. 1994;7(3):259-269.
3. Govoni AF. Developmental stenosis of a thoracic vertebra resulting in narrowing of the spinal canal. *Am J Roentgenol Radium Ther Nucl Med*. 1971;112(2):401-404.
4. Jaspan T, Holland IM, Punt JA. Thoracic spinal canal stenosis. *Neuroradiology*. 1987;29(2):217.
5. Kikuchi S, Watanabe E, Hasue M. Spinal intermittent claudication due to cervical and thoracic degenerative spine disease. *Spine*. 1996;21(3):313-318.
6. Marzluff JM, Hungerford GD, Kempe LG, Rawe SE, Trevor R, Perot PL Jr. Thoracic myelopathy caused by osteophytes of the articular processes: thoracic spondylosis. *J Neurosurg*. 1979;50(6):779-783.
7. Matsuyama Y, Yoshihara H, Tsuji T, et al. Surgical outcome of ossification of the posterior longitudinal ligament (OPLL) of the thoracic spine. *J Spinal Disord Tech*. 2005;18(6):492-497.
8. Okada K, Oka S, Tohge K, Ono K, Yonenobu K, Hosoya T. Thoracic myelopathy caused by ossification of the ligamentum flavum. Clinicopathologic study and surgical treatment. *Spine*. 1991;16(3):280-287.
9. Palumbo MA, Hilibrand AS, Hart RA, Bohlman HH. Surgical treatment of thoracic spinal stenosis: a 2- to 9-year follow-up. *Spine*. 2001;26(5):558-566.
10. Tokuhashi Y, Matsuzaki H, Oda H, Uei H. Effectiveness of posterior decompression for patients with ossification of the posterior longitudinal ligament in the thoracic spine. *Spine*. 2006;31(1):E26-E30.
11. Yamamoto I, Matsumae M, Ikeda A, Shibuya N, Sato O, Nakamura K. Thoracic spinal stenosis: experience with seven cases. *J Neurosurg*. 1988;68(1):37-40.
12. Naylor A. Factors in the development of the spinal stenosis syndrome. *J Bone Joint Surg Br*. 1979;61(3):306-309.
13. Epstein JA, Epstein BS, Lavine L. Nerve root compression associated with narrowing of the lumbar spinal canal. *J Neurol Neurosurg Psychiat*. 1962;25:165-176.
14. Verbiest H. Pathomorphologic aspects of developmental lumbar stenosis. *Orthop Clin North Am*. 1975;6(1):177-196.
15. Ilkko E, Pyhtinen J, Sajanti J. Thoracic spinal canal stenosis: a case report. *Eur J Radiol*. 1994;18(2):126-128.