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Original Article

Surgical treatment for thoracic spinal stenosis

UK Chang[1], WJ Choe[1], CK Chung[1] and HJ Kim*,[1]

[1]*Department of Neurosurgery, Neuroscience Research Institute, Seoul National University College of Medicine Clinical Research Institute, Seoul National University Hospital, Seoul, Korea*

Objectives: To describe the underlying causes, surgical results and prognostic factors in thoracic stenosis causing myelopathy.

Methods: The underlying causes and surgical results were analyzed retrospectively in 28 cases of thoracic spinal stenosis which caused myelopathy. Degenerative spondylosis was the most common cause, and three cases were associated with systemic diseases. Decompressive laminectomy was performed in 24 cases, anterior decompression in five cases, and combined decompression in one case. Ossification of ligamentum flavum was found in 18 cases, facet hypertrophy in 13, ossification of posterior longitudinal ligament in six, and ventral spur in four. Postoperatively 16 patients improved and four patients worsened. Follow-up ranged from 2 months to 5 years and 8 months). Statistical analysis was performed using a χ^2 test to investigate the relationship between subjects. Multivariant analysis (general linear model) was used to determine the factors which influence surgical outcome.

Results: There were neurological improvements in 16 patients, in whom Nurick grade changed from 3.3 preoperatively to 1.8 postoperatively. Eight patients showed no significant change in functional grade and four patients deteriorated after decompressive laminectomy. The group of which initial symptom duration was less than 2 years showed better results ($P=0.006$). The group with sufficient decompression and no additional proximal stenosis had better treatment outcome ($P=0.005$, $P=0.002$).

Conclusion: Chronic severe myelopathy caused by thoracic spinal stenosis can be reversible with appropriate decompression. Surgical outcome was dependent on initial symptom duration, sufficient decompression and presence of additional proximal stenosis.

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Introduction

Even though spinal stenosis in the cervical or lumbar region is well known, thoracic spinal stenosis is less recognized.

After Marzluff *et al*[1] reported four cases of thoracic stenosis due to facet hypertrophy, many authors identified degenerative hypertrophy of the posterior elements as the pathologic cause of thoracic stenosis. Degenerative disc disease causes physical stresses on the posterior elements, produces hypertrophy of these structures, and it encroaches posteriorly on the thoracic cord.[2] Symptoms in thoracic stenosis are related to later development of posterolateral spondylotic changes in the narrow spinal canal.[3] Thoracic cord compression can also result from anterior disc protrusion, spondylotic spurs, an ossified posterior longitudinal ligament, ligamentum flavum thickening or ossification, or a combination of these factors.[2] Degenerative thoracic stenosis is known to develop at the lower thoracic level, where flexion and extension movements frequently occur.[4]

Symptomatic thoracic spinal stenosis is also found in systemic metabolic diseases such as acromegaly, achondroplasia, osteochondrodystrophy, Scheuermann's disease, hypophosphatemic vitamin D-refractory rickets or Paget's disease.[2,5] Secondary thoracic stenosis due to metabolic diseases tends to involve longer segments and leads to circumferential narrowing of the spinal canal.

*Correspondence: HJ Kim, Department of Neurosurgery, Seoul National University Hospital, 28 Yongon-dong, Chongno-ku, Seoul, Korea 110-744

It is known that symptom manifestation present differently according to the patients' age, causes of stenosis and the level of stenosis.[6] However, surgical outcome and its prognostic factors are not well established. We performed surgery on 28 cases of thoracic spinal stenosis caused by various pathologic entities, most of which presented with thoracic myelopathy. To describe surgical outcome and its prognostic factors, medical records and radiological data were analyzed retrospectively.

Materials and methods

Patient population

From 1987 to 1997, 28 consecutive patients with thoracic stenosis underwent decompressive surgery at the authors' institute. There were 15 males and 13 females with a mean age of 49 years, with a range from 4 to 77 years. Postoperative follow-up ranged from 2 months to 68 months (mean 30.6 months). Postoperative follow-up evaluation was done with clinical exam at outpatient clinic.

Diagnosis and definitions

The initial symptoms were paraparesis (eight cases, 29%), sensory change (five cases, 18%), tingling sense (12 cases, 43%) or truncal or back pain (three cases, 10%) (Table 1). The duration of initial symptoms ranged from 2 months to 20 years (median, 12 months).

Spastic paraparesis was the most common chief complaint (24 cases, 86%); there were also two cases of sensory change and two of back pain. The mode of progression was classified as rapid (motor weakness was apparent within 6 months after initial symptoms), or insidious (paraparesis appeared more than 6 months after initial symptoms). Eleven cases (39%) showed a rapid course and 17 (61%) an insidious course.

Clinical features were divided into two subtypes; typical myelopathy, or mixed type with myelopathy and radiculopathy. Typical myelopathy was seen in 24 cases (86%), in whom spastic paraparesis, hyperreflexia, and sensory loss below the stenotic level were noted. Bilateral myelopathy was more frequent than unilateral involvement (18 *vs* six cases). Mixed myelopathy with radiculopathy was seen in four cases (14%), in whom paraparesis, radicular pain and normal deep tendon reflex were found.

Table 1 Initial symptoms of the 28 patients

Initial symptom	*Number of the cases* (%)	*Duration* (months)
Tingling sense	12 (43)	55.8
Paraparesis	8 (29)	41.5
Sensory change	5 (18)	48.6
Truncal pain	3 (10)	61.0

Stenotic levels were classified as upper (T1–T4), middle (T5–T8) or lower (T9–T12) thoracic segment. The most frequently involved segment was the lower thoracic (T9–T12) (15 cases, 54%), followed by the midthoracic (T5–T8) and upper thoracic (T1–T4) (Table 2). Additional significant stenosis in other separate segments was seen proximally and distally in three patients each, at the time of surgery. The other two cases with additional stenosis were treated before or long after thoracic spine surgery (Table 3). In 20 cases (71%), spinal ligaments were ossified in some segments and diffuse idiopathic skeletal hyperostosis (DISH) was found in two cases.

Magnetic resonance imaging (MRI) and metrizamide myelographic computed tomography (MMCT) were used to diagnose the causes of compression of the spinal cord. MRI was performed in 26 and MMCT in 12 patients.

Causes of thoracic stenosis were thickening or ossification of the ligamentum flavum (18 patients, 64%), facet joint hypertrophy (13, 46%), ossification of the posterior longitudinal ligament (six, 22%), and ventral spur or disc protrusion (four, 14%). Patients were thus divided into three groups according to the stenotic site, namely anterior lesions ($n=5$), posterior lesions ($n=18$), and combined anterior and posterior lesions ($n=5$). Anterior lesions included ossification of the posterior longitudinal ligament (OPLL) and spondylotic spur of the vertebral body with or without disc protrusion. Posterior lesions included facet hypertrophy and/or thickening or ossification of the ligamentum flavum (OLF). Severe disc degeneration or protrusion was observed in 10 cases, about 59% of the 17 cases for which MRI films were available. In the remaining seven cases, disc degeneration was not found in stenotic level.

For the evaluation of preoperative and postoperative neurological status, Nurick's functional classification of myelopathy was applied.[7] Mean preoperative Nurick's grade was 3.2, with grades III and IV the most common. Postoperative outcome was classified as improvement, aggravation or no change. Improvement was defined when postoperative Nurick's grade was better than preoperative Nurick's grade.

Surgery

Surgical treatment was performed with posterior (23 patients) and/or anterior decompression (four patients) including one case of combined anterior and posterior decompression. Posterior decompression comprised laminectomy and bilateral medial facetectomy. The

Table 2 The level of involvement in thoracic stenosis

Level	*Cases* (%)
Upper (T1–T4)	6 (21)
Middle (T5–T8)	7 (25)
Lower (T9–T12)	15 (54)

Table 3 The cases with significant stenosis at other segments

No.	*Sex/age*	*CC*	*Level*	*Stenotic cause*	*Associated stenosis*	*Operation*	*Postoperative course*
1	M/69	para	T1–3	FH	L-stenosis	① C7–T3 TL ② L2–4 TL and fusion	Improved
2	F/43	para	T9–12	FH (T11–12) Spur (T9–11) OPLL (T9–11)	Upper T-stenosis	① T8–12 TL ② T7L1–2 TL (8 years later) ③ T2–4 TL	Improved Aggravated Paraplegic
3	F/39	para	T2–3	FH and LF thickening	C-OPLL	① T2,3 TL and T1,4 PL ② C1–7 TL (7 years later)	Improved Rt. hemi
4	M/52	para	T8–10	OLF	C-OPLL (DISH)	Simultaneous TL C2–C7 and T8–T10 C4,5 corpectomy and AIF (3 years later)	Good
5	M/17	para	T3–4	FH	L-stenosis (achondroplasia)	T2–4 TL	Slightly improved
6	F/46	para	C7–T3	OPLL	C-OPLL (DISH)	① C7–T5 TL ② C1–C6 TL	Quadriparesis
7	M/32	para	T11–L1	Compression fracture (T11,12)	L-stenosis	Ant. Decompression	Improved
8	M/77	para	T11,12	OLF and FH	L-stenosis	① L2–5 TL ② T1,12 TL	Paraparesis Improved

*para = paraparesis; FH = facet hypertrophy; OLF = ossified ligamentum flavum; ant = anterior; TL = total laminectomy; PL = partial laminectomy; hemi = hemiparesis; AIF = anterior interbody fusion; OPLL = ossification of posterior longitudinal ligament; CC = chief complaint

Table 4 Number of decompressions compared with surgical approach

	Stenotic segments	*Decompressed segments*
Anterior decompression group ($n=4$)	1.8	1.2
Posterior decompression group ($n=23$)	2.2	3.6

mean numbers of stenotic and decompressed segments were 2.2 and 3.6 respectively (Table 4). Asymmetrical cord compression by the posterior element was observed in 11 cases; predominant right-sided compression ($n=6$) was slightly more than left sided ($n=5$). Eighteen patients in the dorsal lesion group, four in the combined lesion group and one in the anterior (OPLL) group underwent posterior decompression. Four underwent anterior decompression. In the anterior decompression group, the mean number of stenotic and decompressed segments was 1.8 and 1.2 respectively (Table 4). Anterior decompression included the anterolateral approach (thoracotomy) in one case of ventral spur due to old compression fracture and one of OPLL, the posterolateral approach (costotransversectomy) in one case of ventral spur due to old compression fracture, and the posterior approach (transpedicular) in one case of OPLL. Combined anterior (transpedicular approach) and posterior decompression was performed in one patient with disc herniation and a thickened ligamentum flavum. Bony fusion was done with anterior approach and not with the posterior approach (Table 5).

Factor analysis

Data analysis was performed using a computer statistical program (SPSS for Windows; SPSS Inc., Chicago, IL, USA). Statistical analysis was performed using a χ^2 test to investigate the relationship between subjects. The analyzed factors were age (below 49 and over 49 years), initial symptoms, duration of initial symptoms (up to 2 years, over 2 years), chief complaints, duration of chief complaints (up to 2 months, from 2–4 months, over 4 months), clinical type, preoperative Nurick's grade, stenotic level, stenotic cause, additional proximal stenosis and sufficient decompression. Multivariant analysis (general linear model) was used to determine the factors which influence surgical outcome. For these analyses, the criterion for statistical significance was set at a probability value of 0.05 or less.

Results

Clinical manifestations

The mean duration of initial symptoms was 51.0 months (median, 12.0 months) and mean duration of chief complaint was 24.1 months (median, 4 months). There was no significant difference between degenerative and secondary stenosis group (Table 6).

The mean interval from initial symptoms to paraparesis was 29.7 months; in the group with ventral pathologic lesions the interval was shorter than in the group in which stenosis was attributed only to posterior elements, which was statistically significant ($P=0.032$). Sphincter change was present in 14 cases (50%).

Table 5 The stenotic causes and surgical approach

Compression site	*Stenotic cause*	*Cases (%)*	*Surgical approach*
Posterior	Facet hypertrophy	4 (14)	Post. decompression
	Ossified LF	7 (25)	Post. decompression
	Combined	7 (25)	Post. decompression
Anterior	OPLL	3 (11)	Ant. decompression (2)
			Post. decompression (1)
	Ventral spur	2 (7)	Ant. decompression
Ant. and Post.		5 (18)	Post. decompression (4)
			Combined decompression (1)

*Ant = anterior; Post = posterior; OPLL = ossified posterior longitudinal ligament; LF = ligamentum flavum

Table 6 Comparison between degenerative and systemic stenosis

	Age	*Duration of CC (months)*	*Duration of initial Sx (months)*	*Interval btw CC and initial Sx (months)*
Degenerative Stenosis	52.3	26.2	52.0	29.0
Systemic Stenosis	29.7	11.8	45.0	33.3
Total	49.1	24.1	51.0	29.7

*CC = chief complaint; Sx = symptoms; btw = between

Radiological findings

Signal change in the spinal cord was found in eight cases (32%) on preoperative MRI, but did not influence postoperative outcome ($P = 0.208$). On MR T1-weighted images, thickening or ossification of the ligamentum flavum was seen as a central hyperintense triangular structure with marginal hypointensity along the posterior surface of the spinal cord. The characteristic feature was a beak-like, bilaterally thickened projection into the spinal canal. Ossification of the ligamentum flavum was seen on CT as linear high density. Thickening with or without ossification, was often associated with facet joint degeneration. Parasagittal T1- or T2-weighted images showed apophyseal impingement on epidural fat or the subarachnoid space and axial views more clearly showed compression of the spinal cord by a hypertrophied superior articular process (Figure 1).

Surgical outcome

Most patients improved after decompression. There was symptomatic improvement in 22 cases (79%); motor function improved in 16 (57%) and sensory impairment or leg pain was alleviated in the remaining six (22%). No neurological change was seen in two cases in which the duration of symptoms was so long (4 years and 14 years, respectively).

Functional grade was aggravated in four cases of posterior decompression. One patient had whole-spine OPLL, and only the upper thoracic spine was decompressed posteriorly (Table 3, case 6). Another underwent posterior decompression in the lower thoracic spine where the spinal cord was compressed by OPLL and hypertrophied facet, although there was additional upper thoracic stenosis (Table 3, case 2). Two others had an ossified ligamentum flavum at midthoracic level. Overall mean postoperative Nurick's grade was 2.6, while mean preoperative Nurick grade was 3.2.

Table 7 Statistical significance of prognostic factors

	Multi-variant analysis (P-value)
Age	0.355
Chief complaint	0.255
Duration of chief complaint	0.107
Initial symptom	0.133
Duration of initial symptom	0.003
Preoperative Nurick Grade	0.889
Proximal stenosis	0.001
Stenotic cause	0.728
Stenotic level	0.144
Clinical type	0.512
Sufficient decompression	0.002

Factor analysis

The result of χ^2 test and multi-variant analysis was summarized in Table 7. Patients in whom the duration of initial symptoms was less than 2 years showed a better postoperative outcome than those whose initial symptoms had lasted for more than 2 years ($P = 0.003$).

Additional proximal stenosis was inversely related to surgical outcome ($P = 0.001$). In seven patients, there was additional stenosis – either preoperatively or at the time of surgery – at other levels. In four patients, in whom proximal decompression was

performed first, the results were good. These were three cases of additional lumbar stenosis and one case of additional cervical OPLL (Table 3). In case 4, posterior decompression for cervical OPLL and lower thoracic OLF were performed simultaneously.

In three cases in which distal decompression was carried out first, the outcome was poor. One was a case of whole spine OPLL, in which symptomatic upper thoracic segments were decompressed posteriorly. Postoperatively, symptoms of proximal cord compression appeared (Table 3, case 6). The remaining two cases were associated with cervical OPLL (Table 3, case 2) and lumbar stenosis (Table 3, case 8). In case 8, neurological status was aggravated after initial lumbar decompression but improved after late thoracic decompression.

In the group in which causes of stenosis were sufficiently removed, the results were better than in the group with residual compression ($P=0.002$). In 23 cases, operations were done for the entire removal of stenotic causes, but posterior decompression was carried out in one case of OPLL and four of circumferential stenosis (Table 5).

Discussion

Clinical manifestations

Symptomatic thoracic spinal stenosis is often associated with skeletal involvement of systemic endocrine disease. Degenerative spinal stenosis at the thoracic level, however, is found increasingly in patients who undergo MR imaging. The most frequent initial symptom was a tingling sensation, and the most frequent chief complaint was paraparesis, which was usually spastic. The number of cases with rapid progression was higher in the ventral lesion group than in posterior lesion only group, with statistical significance ($P=0.032$). Sphincter change was present in half of all cases, and more frequently in the rapid course group than in the insidious course group, but there was no significant difference between the rapid course group and insidious course group ($P=0.653$). This was not influenced by accompanied disc protrusions. Those with thoracic stenosis rather than disk herniations are known to present with more chronic course and rare sphinchteric deficits,[1,8] a finding consistent with that of the authors, though the number of cases with sphincter change was somewhat higher in our data.

The authors observed two different clinical types: typical myelopathy, and mixed type myelopathy with radiculopathy. It is reported that when the T9-T10 level or higher is involved, the manifestations are typical myelopathy, mixed type with common sphincter dysfunction is seen in cases below the T10 level.[6] However, such relationship between stenotic level and symptom manifestation was not evident in our data. Most patients (86%) showed typical myelopathy, while a small portion (14%) manifested mixed type. Mixed type with myelopathy and radiculopathy consists of lower motor neuron type weakness, a normal degree of deep tendon reflex, and radicular distribution of pain or sensory symptom. Severe motor but less sensory deficit can be explained by the fact that vascular supply to anterior gray matter, consisting of perforators from both anterior and lateral arteries in the watershed zone, was more tenuous than that to posterior sensory columns.[8]

The segments most frequently involved in thoracic stenosis were lower thoracic (T9–T12), in which there is great mobility and vulnerability to flexion, extension, and rotation.[9,10] Both the frequency and severity of ossification of ligamentum flavum increase as the thoracolumbar junction is approached; this is because the lower thoracic spine undergoes local mechanical stress from tensile force due to frequent motion.[11]

Cases in which there was significant stenosis in other segments are summarized in Table 3. Six cases of confirmed additional stenosis was found at the time of thoracic decompression; in case 8 lumbar decompression preceded thoracic decompression, while in case 3, cervical stenosis developed long after thoracic decompression.

Hyperostosis was seen in 20 cases (71%), and in two, there was associated diffuse idiopathic skeletal hyperostosis (DISH). This is a skeletal disease characterized by ossification of the anterolateral aspect of vertebral bodies, without disc degeneration or facet joint ankylosis, and often combined with ossification of the ligamentum flavum and OPLL.[12]

Radiological findings

Diagnostic methods comprised MMCT and MRI. MMCT defined the contour of ossification most precisely, and also demonstrated the presence of ossification of the dura mater in OLF and OPLL.[13] There are, however, three main disadvantages of CT. First, at the upper thoracic level, artifacts occurred due to the scapula or shoulder joint. Second, CT of the entire spine is impractical, and the level of scanning must therefore be previously decided on the basis of other neurological information. Third, the information provided by sagittal reconstruction was inferior to that obtained by MRI,[13] which is now widely accepted as the procedure of choice in suspected thoracic myelopathy. A relative disadvantage of MRI compared to CT is that cortical bone shows low signal intensity on MR images.[14] MRI, however, more accurately predicts the significance of spinal canal compromise by defining the spinal cord separately from the thecal sac. Moreover, sagittal MR images better define the extent of cephalocaudal involvement, which is an important surgical consideration. MRI is the imaging modality which best differentiates calcification and ossification of posterior elements,[14] and it can identify ventral pathologic lesions more accurately than CT. MRI has been more frequently used and is more useful in the authors' experience.

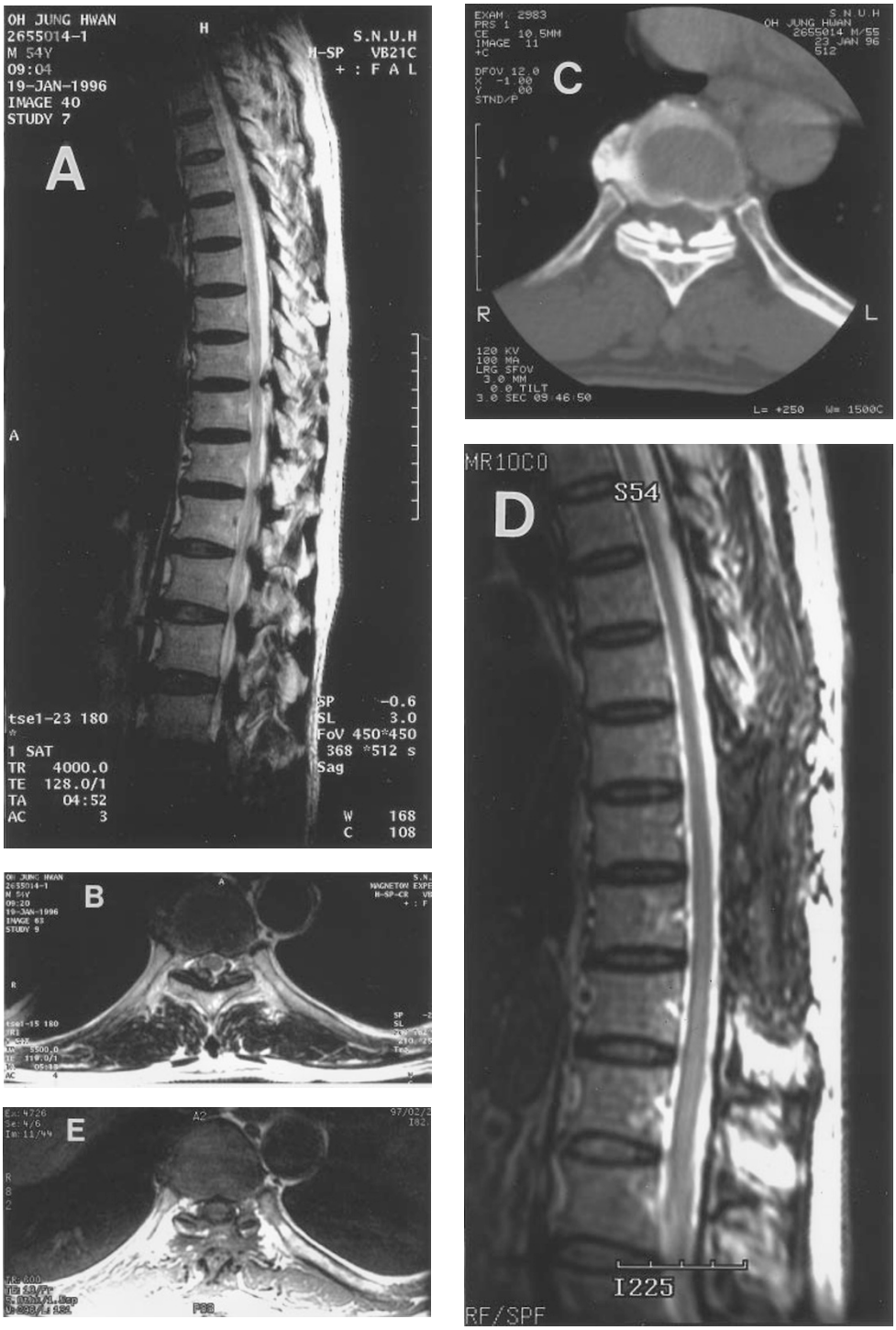

Figure 1 A 55 year-old man presented with progressing paraparesis and tingling sense of his legs. T2-weighted sagittal (**A**), axial (**B**) magnetic resonance (MR) image and computed tomography (**C**) showed thoracic stenosis due to ossification of ligamentum flavum in T9–10 level. Decompression through posterior approach was done. Follow-up MR images after 12 months after operation demonstrate restoration of cerebrospinal fluid space on T2-weighted sagittal (**D**) and T1-weighted axial (**E**) image. His neurological problem gradually improved during the follow-up period

In our series, the most frequent pathologic lesion was OLF. This develops as part of the aging process; the radiologically based prevalence rates of thoracic OLF were found to be 6.2% in males and 4.8% in females.[15] The condition occurs mainly in intervertebral segments at lower thoracic levels, protruding inferiorly from inferior facets into projections of intervertebral foramina. It shows low signal intensity on T2-weighted image and marrow signal within the central aspect of the calcific mass on T1-weighted image.[13,16] Thickening or ossification of the ligamentum flavum is often associated with facet joint hypertrophy. The orientation of facet joint in thoracic spine is in the coronal plane, so hypertrophy of the superior articular process can compress posterolateral spinal cord and which assumes trefoil appearance. It is important to appreciate that facet hypertrophy is clearly visualized paramedially, and that true-sagittal MR images may be misleading.[2]

OPLL is a rare cause of thoracic myelopathy; in our series it involved six female patients. A Japanese study, based on a fixed population sample, revealed that the prevalence of thoracic OPLL was 0.6%, and that patients were nearly always asymptomatic.[17] In OPLL, proton density MR imaging demonstrated ossified lesions with high sensitivity.[18] High signal intensity seen on T1 weighted images within ossified lesions indicates the presence of fatty marrow and corresponds with a radiolucent area within the ossified lesions seen on conventional tomograms.[18]

Degenerative stenosis is often said to be associated with disc degeneration, especially stenosis due to ligamentum flavum ossification.[19] Disc protrusion was detected at the stenotic segments in 50% of degenerative stenosis in the authors' cases and disc degeneration was found in about 82%. Marginal osteophyte formation and ossification of the ligamentum flavum is known to develop at the same level as the affected disc in more than 80% of patients with herniated disc at the thoracic level.[19] This indicates the importance of mechanical factor in the pathogenesis of thoracic stenosis.

Surgery

The choice of surgical approach depends on the site of the compressive lesion, its level and the patients' neurological status. A decision regarding the extent of decompression depended on the maximally compressed segments in anterior decompression, and is focused on how long decompression was necessary to include entire stenotic segments in posterior decompression.

In thoracic myelopathy, a ventral lesion has been treated by posterior decompression, but in a patient with a large anterior lesion, or one in the middle or lower thoracic spine, posterior decompression should be avoided; because of the physiologic kyphosis of the thoracic spine, the effect of posterior decompression is uncertain.[20] Anterior decompression is more suitable than posterior; it permits adequate anterior spinal cord decompression and good thoracic spinal stability by means of bone graft.[21] Anterior decompression can be achieved by one of three approaches, posterior (transpedicular), lateral (costotransversectomy), or anterolateral (transthoracic).[22] Although requiring thoracotomy, the transthoracic approach provides extensive exposure of the thoracic vertebral bodies between T4 and the thoracolumbar junction and as well as the direct access required for anterior pathologic investigation. It can, moreover, more effectively deal with diffuse lesions involving more than three consecutive levels, and provide optimal exposure of midline lesions, thus enabling the repair of dural tears.[22] Both posterior and anterior decompression appeared effective for the treatment of upper thoracic ventral pathology, though for OPLL of the mid or lower thoracic level, anterior decompression is thought to be the best approach.[23] This is consistent with the author's experience.

Posterior decompression was carried out in cases involving OLF or facet joint hypertrophy. Thickening or ossification of the ligamentum flavum (LF), which is composed mainly of elastic connective tissue fiber in a longitudinal array[24] is one of the major causes of spinal stenosis. Thickening is produced by increased amounts of fibrous tissue within the ligaments and mucoid swelling and hyalinization of inter-elastic fibrous connective tissue.[24,25] In addition, LF thickening is also thought to be caused by the buckling of ligaments secondary to degenerative facet joint changes and spondylosis.[24] Ossification of the LF developed along the superficial layer of thickened LF, arising from both sites in the capsular and interlaminar portions.[11] Asymmetric cord compression by posterior elements was therefore common, occurring in about 50% of the cases in this series. Histological study revealed the presence of numerous fibrocartilaginous cells in the increased and swollen collagen fibers, indicating that developmental mode in the OLF is mainly endochondral ossification.[11]

Surgical outcome

After thoracic cord decompression, symptomatic improvement was seen in all but six cases. To investigate the prognostic factors influencing surgical outcome, many factors were analyzed.

Patients in whom the duration of initial symptoms was less than 2 years showed a better postoperative outcome than those whose initial symptom had lasted for more than 2 years ($P=0.003$). Thirteen cases showed improved functional outcome in 16 cases of which initial symptom duration was less than 2 years. However, only three cases improved functionally in the group in which initial symptom duration was over 2 years, four aggravated, and five cases of no change.

The presence of additional proximal stenosis was inversely related to surgical outcome ($P=0.001$). To explain neurological deterioration after distal decompression with proximal compression, several mechan-

isms have been proposed. The caudal movement of CSF into the expanded distal thecal sac might decrease CSF buffer space between the stenotic cause and the spinal cord, thereby allowing cord compression.[26] It has also been postulated that the displacement of CSF below the proximal stenotic lesion could reduce intrathecal pressure, allowing epidural venous engorgement. This would then compromise venous drainage resulting in cord swelling and additional cord compression.[26]

In the group in which stenosis were sufficiently decompressed, the results were better than in the group with residual compression ($P=0.002$). The causes of postoperative neurological deterioration in two cases of midthoracic ossified ligamentum flavum might be residual compression caused by the proximal end of decompressed segments and spinal cord injury during laminectomy. In one case, spinal cord strangulation by a proximal laminectomy margin was proved during reoperation; in the other case immediate postoperative MR showed a high signal in the intramedullary region of segments which had undergone surgery; this lesion was located on the left side which corresponds to the preoperative compression site.

Conclusion

Chronic myelopathy with thoracic spinal stenosis could be relieved by appropriate surgical decompression. In cases where thoracic decompression was done with proximal stenosis, the results tended to be poor, even if there had been preoperatively no sign of proximal cord compression. When surgeons encounter cases of thoracic stenosis, evaluation of the cervical region, therefore, appears necessary. In addition, in cases of circumferential stenosis, sufficient decompression for all causes of stenosis is recommended.

References

1 Marzluff JM *et al*. Thoracic myelopathy caused by osteophytes of the articular processes: thoracic spondylosis. *J Neurosurg* 1979; **50:** 779–783.
2 Barnett GH, Hardy Jr RM. Thoracic stenosis and spondylosis. In: Tarlov EC (ed) *Neurosurgical Treatment of Disorders of the Thoracic Spine*. Neurosurgical topics: American Association of Neurological Surgeons Publication Committee 1991, pp 45–51.
3 Epstein NE, Schwall G. Thoracic spinal stenosis; diagnostic and treatment challenges. *J Spinal Disord* 1994; **7:** 259–269.
4 Panjabi MM *et al*. Thoracic human vertebrae. Quantitative three-dimensional anatomy. *Spine* 1991; **16:** 888–901.
5 Parfitt AM, Duncan H. Metabolic bone disease affecting the spine. In: Rothman FH, Simeone FA (eds.) *The Spine*. 2nd edn. Philadelphia: WB Saunders, 1982; pp 856–867.
6 Yonenobu K *et al*. Thoracic myelopathy secondary to ossification of the spinal ligament. *J Neurosurg* 1987; **66:** 511–518.
7 Phillips DG. Surgical treatment of myelopathy with cervical spondylosis. *J Neurol Neurosurg Psychiatry* 1973; **36:** 879–884.
8 Yamamoto I *et al*. Thoracic spinal stenosis: experience with seven cases. *J Neurosurg* 1988; **68:** 37–40.
9 Barnett GH *et al*. Thoracic spinal canal stenosis. *J Neurosurg* 1987; **66:** 338–344.
10 Omojola MF *et al*. Thoracic myelopathy secondary to ossified ligamentum flavum. *J Neurosurg* 1982; **56:** 448–450.
11 Okada K *et al*. Thoracic myelopathy caused by ossification of the ligamentum flavum, Clinicopathologic study and surgical treatment. *Spine* 1991; **16:** 280–287.
12 Resnick D, Niwayama G. Radiographic and pathologic features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). *Radiology* 1976; **119:** 559–568.
13 Hanakita J *et al*. Neuroradiological examination of thoracic radiculomyelopathy due to ossification of the ligamentum flavum. *Neuroradiology* 1990; **32:** 38–42.
14 Yoshino MT, Seeger J, Carmody RF. MRI diagnosis of thoracic ossification of posterior longitudinal ligament with concomitant disc herniation. *Neuroradiology* 1991; **33:** 455–457.
15 Kudo S, Ono M, Russel WJ. Ossification of thoracic ligamenta flava. *AJR* 1983; **141:** 117–121.
16 Goodman JM, Kuzma BB. Ossification of the ligamentum flavum with myelopathy. *Surg Neurol* 1996; **46:** 396–397.
17 Ono M *et al*. Ossification of the thoracic posterior longitudinal ligament in a fixed population. *Radiology* 1982; **143:** 469–474.
18 Otake S *et al*. Ossification of the posterior longitudinal ligament: MR evaluation. *AJNR* 1992; **13:** 1059–1067.
19 Shiraishi T, Crock HV, Lewis P. Thoracic myelopathy due to isolated ossification of the ligamentum flavum. *J Bone Joint Surg Br* 1995; **77:** 131–133.
20 Fujimura Y *et al*. Long-term follow-up study of anterior decompression and fusion for thoracic myelopathy resulting from ossification of the posterior longidutinal ligament. *Spine* 1997; **22:** 305–311.
21 Ido K *et al*. Anterior decompression and fusion for ossification of posterior longitudinal ligament in the thoracic spine. *J Spinal Disord* 1995; **8:** 317–323.
22 Kojima T, Waga S, Kubo Y, Matsubara T. Surgical treatment of ossification of the posterior longitudinal ligament in the thoracic spine. *Neurosurgery* 1994; **34:** 854–858.
23 Kurosa Y, Yamaura I, Osamu N, Shinomiya K. Selecting a surgical method for thoracic myelopathy caused by ossification of the posterior longitudinal ligament. *Spine* 1996; **21:** 1458–1466.
24 Stollman A, Pinto R, Benjamin V, Kricheff I. Radiologic imaging of symptomatic ligamentum flavum thickening with and without ossification. *AJNR* 1987; **8:** 991–994.
25 Rosenbloom SA. Thoracic disc disease and stenosis. *Radiol Clin N Am* 1991; **29:** 765–775.
26 Valls PL, Naul LG, Kanter SL. Paraplegia after routine lumbar laminectomy: report of a rare complication and successful management. *Neurosurgery* 1990; **27:** 638–640.