

Spinal Cord Kinking in Thoracic Myelopathy Caused by Ossification of the Ligamentum Flavum

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Abstract

Background: Ossification of the ligamentum flavum (OLF) is being increasingly recognized as a cause of thoracic myelopathy. This study was to describe a rare clinical entity of spinal cord kinking (SK) in thoracic myelopathy secondary to OLF.

Methods: The data of 95 patients with thoracic myelopathy secondary to OLF were analyzed retrospectively. The incidence and location of SK were determined using preoperative magnetic resonance imaging (MRI). The clinical presentation and radiological characteristics in patients with SK were analyzed. Posterior *en bloc* laminectomy with OLF was performed, and the surgical results were evaluated.

Results: SK was found in seven patients (7.4%) based on preoperative MRI. The patients included one male and six females with an average age of 55.6 years (range, 48–64 years). Five patients presented with radiculomyelopathy and two presented with typical thoracic myelopathy of spastic paraparesis. In all cases, the kinking was located just above the end of the spinal cord where the conus medullaris (CM) was compressed by the OLF. The degree of SK varied from mild to severe. The tip of the CM was located between the upper third of T11 to the lower third of L1, above the lower edge of L1. With an average follow-up of 30.4 months, the modified Japanese Orthopedic Association score significantly improved from 5.7 ± 1.8 preoperatively to 8.9 ± 1.4 postoperatively ($t = 12.05$; $P < 0.0001$) with an improvement rate of $63.1 \pm 12.3\%$.

Conclusions: SK is a rare radiological phenomenon. It is typically located at the thoracolumbar junction, where the CM is compressed by the OLF. Our findings indicate that these patients may benefit from a posterior decompressive procedure.

Key words: Ossification of Ligamentum Flavum; Spinal Cord Kinking; Thoracic Myelopathy

INTRODUCTION

Ossification of the ligamentum flavum (OLF) is being increasingly recognized as a cause of thoracic myelopathy.^[1-6] It is characterized by heterotopic bone formation in the posterolateral sides of the spinal canal that progresses slowly and compromises the function of the spinal cord.^[7,8] Spinal cord kinking (SK) is a rare radiological phenomenon that is associated with idiopathic spinal cord herniation^[9] and spine shortening surgery.^[10] So far, only one case of SK in thoracic OLF has been described.^[11] In the present study, we analyzed consecutive cases of SK in thoracic myelopathy secondary to OLF.

METHODS

Between January 2006 and December 2011, 95 consecutive patients with thoracic OLF-induced myelopathy underwent surgery at our institution. Our review of this consecutive

series was conducted following approval by the institutional review board and the ethics committee of our hospital. The diagnosis was established by computed tomography (CT) and magnetic resonance imaging (MRI) and further confirmed during the operation. Patients with thoracic myelopathy caused by thoracic disc herniation or ossification of the thoracic posterior longitudinal ligament were excluded from this study.

The incidence and location of SK in these patients were examined using preoperative MRI. The definition of SK

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based on MRI was angulation of the spinal cord by more than 10° at the OLF. The clinical presentations, including symptom duration, numbness of the lower extremities (LE), leg pain, weakness of the LE, intermittent claudication, spastic paraparesis and sphincter dysfunction, were evaluated. Based on the preoperative CT, OLF at the level that had the most severe appearance was classified into either the nonfused type or the fused type. The location of OLF, SK and the tip of the conus medullaris (CM) were evaluated using preoperative MRI. Co-existence of spinal diseases such as lumbar canal stenosis (LCS), lumbar disc herniation and cervical posterior longitudinal ligament ossification was also investigated. Posterior *en bloc* laminectomies with pedicle screw fixations were performed in all cases. The modified Japanese Orthopedic Association (mJOA) scoring system was used to evaluate the neurological status before surgery and at follow-up. The maximum score of 11 indicates normal function. Postoperative neurological recovery was estimated on the basis of the recovery rate (RR) using the following equation:

$$RR = (\text{postoperative mJOA} - \text{preoperative mJOA}) / (11 - \text{preoperative mJOA}) \times 100\%.$$

All statistical analyses were performed using the SPSS software (version 17; SPSS Inc., Chicago IL, USA). A paired *t*-test was conducted to determine significant differences in preoperative and following-up clinical outcomes scores.

RESULTS

Clinical presentation

Among the 95 patients with thoracic myelopathy caused by OLF, seven cases (7.4%) were found to have SK by preoperative MRI. The seven patients included one male and six females with a mean age of 55.6 years (range, 48–64 years). The median duration of symptoms before surgery was 13.1 months (range, 3–24 months). Five patients presented with radiculomyelopathy, and two patients presented with typical myelopathy of spastic paraparesis. The primary complaints on admission were lower limb numbness and sensory dysfunction (7 patients), tingling or pain in the legs (3 patients), weakness of the LE (7 patients), intermittent claudication (7 patients, 100%), spastic paraparesis (2 patients), and sphincter

dysfunction (3 patients). The symptoms are summarized in Table 1.

Radiological characteristics

SK was located at the thoracolumbar junction (T10-T12), where the CM was involved in the compression caused by OLF. The location of OLF with the most severe compression was between T10 and L1. The tip of the CM was located from the upper third (UT) of T11 to the lower third of L1, above the lower edge of L1 [Table 2]. A re-evaluation of the 95 cases showed that the CM was involved in the compression by OLF in 13 patients. Therefore, the rate of SK was 53.8% (7/13) when the conus medullaris was involved.

CT at the involved level revealed a hyperdense, typical V-shaped configuration of the OLF [Figure 1]. Nonfused type OLF was diagnosed in three cases, and fused type OLF was diagnosed in four cases. The T2-weighted sagittal MRI scan showed that the degree of SK varied from mild to severe [Figure 2]. MRI revealed the OLF to be a beak-like excrescence situated posterior to the thecal sac and impinging the CM [Figures 1 and 2]. The co-existing spinal disease included LCS in two cases, lumbar disc herniation in one case and cervical posterior longitudinal ligament ossification in one case.

Surgical outcome

The preoperative mJOA score was 5.7 ± 1.8 . After the operation, the clinical status of all the patients improved, and no neurological deterioration occurred. Postoperative MRI in two cases showed that the SK disappeared after posterior *en bloc* laminectomy of the OLF [Figure 1]. With an average follow-up of 30.4 months (range, 25–36 months), the mJOA score significantly improved to 8.9 ± 1.4 ($t = 12.05$; $P < 0.0001$) and the improvement rate was $63.1 \pm 12.3\%$ [Table 3].

DISCUSSION

Thoracic myelopathy secondary to OLF has been primarily reported in the east asian region.^[1-6,8-13] It commonly affects the lower thoracic spine, especially T9–L1.^[1,4-6,14,15] Although its pathogenesis remains unclear, the morphology of OLF in the thoracic canal has been well described.^[16] The ligamentum flavum is comprised of two parts: The medial interlaminar portion and the lateral capsular portion.

Table 1: Clinical characteristics of the patients

Patient	Age (years), sex	Symptom duration (months)	Numbness in LE	Pain in LE	Weakness in LE	Claudication	Spastic paraparesis	Bladder bowel dysfunction
1	56, female	6	Yes	No	Yes	Yes	No	No
2	59, female	3	Yes	Yes	Yes	Yes	No	No
3	51, female	24	Yes	Yes	Yes	Yes	No	Yes
4	64, female	24	Yes	No	Yes	Yes	No	No
5	48, male	18	Yes	No	Yes	Yes	Yes	Yes
6	50, female	5	Yes	No	Yes	Yes	Yes	Yes
7	61, female	12	Yes	Yes	Yes	Yes	No	No

LE: Lower extremities.

Table 2: Radiological findings

Patient	Location of OLF	Maximum compression	Location of SK	Classification of OLF	End tip of CM	Coexisting spinal disease
1	T12-L1	T12-L1	T12	Nonfused	LT of L1	LCS
2	T11-T12	T11-T12	T11	Nonfused	T12-L1 disc	LCS
3	T10-T12	T11-T12	T11	Fused	LT of T12	None
4	T10-T12	T10-T11	T10	Fused	UT of T11	None
5	T10-T12	T11-T12	T11	Fused	LT of T12	None
6	T10-T12	T11-T12	T11	Fused	LT of T12	Cervical OPLL
7	T11-T12	T11-T12	T11	Nonfused	T12-L1 disc	LDH

SK: Spinal cord kinking; OLF: Ossification of the ligamentum flavum; CM: Conus medullaris; LT: Low third; UT: Upper third; LCS: Lumbar canal stenosis; OPLL: Ossification of posterior longitudinal ligament; LDH: Lumbar disc herniation.

Table 3: Surgical findings

Patient	Level of decompression	Preoperative mJOA	Follow-up mJOA	Improvement mJOA	Follow-up duration (months)
1	T12-L5	7	10	3	36
2	T11-L1	8	10	2	25
3	T9-L1	4	7	3	28
4	T10-T12	7	10	3	36
5	T10-T12	4	8	4	32
6	T10-L1	3	7	4	29
7	T11-T12	7	10	3	27

mJOA: Modified Japanese Orthopedic Association.



Figure 1: (a) Axial computed tomography scan showing the typical V-shaped configuration of the ossification of the ligamentum flavum in case 2. (b) Sagittal T2-weighted magnetic resonance imaging scan showing impingement of the conus medullaris by the beak-like ossification of the ligamentum flavum in the same case. (c) Sagittal T2-weighted magnetic resonance imaging scan in case 2 showing the disappearance of spinal cord kinking after posterior *en bloc* laminectomy with ossification of the ligamentum flavum.

Ossification usually starts in the capsular portion and then gradually extends to the interlaminar area. With enlargement of the ossification, the spinal cord might be compressed from the posterior and lateral sides. The characteristic V-shaped mound of OLF was observed in our preoperative CT scans.

The basis for SK in thoracic OLF is unclear. SK has been reported in idiopathic spinal cord herniation^[9] and spinal column shortening surgery.^[10] Miyazaki *et al.*^[11] reported that focal adhesive arachnoiditis might be the cause of SK in a patient with thoracic OLF. In our seven cases of SK, the compression as a result of OLF occurred at the CM. As the CM tapers toward the end of the spinal cord, we believe that



Figure 2: Sagittal T2-weighted magnetic resonance imaging scan in case 3 (a), case 4 (b), and case 5 (c) showing that the degree of spinal cord kinking varied from mild to severe. The kinking was located at the thoracolumbar junction, where the conus medullaris was involved in the compression by ossification of the ligamentum flavum.

the tapering of the CM may be the cause of SK. Although the CM is stabilized in the spinal canal by the denticulate ligament, nerve root, and the filum terminale,^[17] there is significant evidence that it moved along the cranio-caudal axis.^[18] With the narrowing of the thoracic canal caused by the insidious progression of OLF, the CM may be crushed upward because of its tapering form. The upward shift of the CM may result in SK. It is generally accepted that the CM terminates in the lower third of L1. However, in our seven cases of SK, the tips of the conus medullaris were located from the UT of T11 to the lower third of L1, above the lower edge of L1. This might be the result of the upward shift of the CM caused by OLF.

Thoracic myelopathy caused by OLF predominantly involves static compression.^[19] However, when SK occurs, other causes may also contribute to the resulting neurological deficits. It is rational to suggest that excessive morphological deformation

of the cord would adversely affect the blood flow. Kawahara *et al.*^[20] described SK in acute spinal column shortening in a canine model. They found that shortening of more than two-third of a single vertebral segment was characterized by SK and neurological dysfunction. They suggested that the causes of neurological dysfunction in SK might be morphologic deformation of the cord, decreased spinal cord blood flow, and anterior spinal artery obstruction. With the upward shift of the CM by compression from OLF, the tethering effect of the dentate ligament, nerve roots, and the filum terminale may also contribute to the neurological deficits. With change in position, the repeated dynamic mechanical damage to the CM and nerve roots may also play an important role. Therefore, it is likely that a combination of these factors caused the neurological symptoms observed in our patients.

Based on the symptoms, thoracic myelopathy can be divided into two clinical types: typical myelopathy and mixed-type myelopathy with radiculopathy.^[5,12,14] When OLF occurs at the lowest levels of the thoracic canal, the resulting compression can produce mixed upper and lower motor neuron lesions due to compromise of both the CM and caudal nerve roots. Only two cases in our report had presented with spastic paraparesis, and the other five cases presented with radiculomyelopathy. The symptoms of these five patients mimic LCS may result in misdiagnosis.^[5]

A nonsurgical approach for symptomatic patients with thoracic OLF is not effective.^[13,19] Surgery is the only treatment that can adequately address the significant compression of neurologic structures caused by OLF.^[13,16] Miyazaki *et al.*^[11] reported that the symptomatic SK in OLF was due to focal adhesive arachnoiditis. They recommended that adhesiolysis of the arachnoid be performed to release the SK after laminectomy and OLF resection. In our seven cases, only *en bloc* laminectomy with OLF was performed for decompression. The kinking disappeared after the operation, and all the patients showed satisfactory improvement in their neurological deficit. Therefore, we believe there is no need to perform intradural arachnoid release, as it may be associated with complications. In these patients, significant improvements can only result from posterior *en bloc* laminectomy.

In conclusion, SK is a rare radiological phenomenon typically located at the thoracolumbar junction where the CM is compressed by OLF. The tapering of the CM may be the cause of SK when it is involved in progressive compression by OLF. Further, patients with SK may show significant improvement after a posterior decompressive procedure.

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Conflicts of interest

There are no conflicts of interest.

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