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Degenerative changes in the interspinous ligament

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Objective: The aim of this study was to investigate the imaging assessment of interspinous ligament degeneration (ISLD) in patients with or without low back pain (LBP).

Methods: Sixty patients with LBP were enrolled in Group A and 60 subjects frequency-matched by age and sex in Group B. An MRI-based grading system for ISLD was scored and ranged from Type A (normal) to Type D (severe). The lumbar disc was also graded according to degeneration at four lumbar levels.

Results: Type A ISLD was the most prevalent type with 161 levels (67.1%) in Group A and 172 (71.7%) in Group B. Type D was the least frequent, seen in 13 levels in Group A and 3 in Group B. There was a significantly higher incidence of Type D ISLD in Group A than Group B (5.4% vs. 1.3%, $p<0.05$). The average age of patients with Type D ISLD in Group A was higher than Types A, B and C (Type A and B $p<0.01$, Type C $p<0.05$). In Group B, the age of patients with Type D ISLD was significantly higher than those with Type A ($p<0.05$). Although disc grade increased in advanced ISLD in both groups, only the difference between Type D and Types A and B in Group A were statistically significant ($p<0.05$).

Conclusion: More advanced ISLD grades were less common in patients with or without LBP. Advanced change of the ISL was more common in patients with LBP. ISLD occurred in more severe disc degeneration.

Key words: Interspinous ligament; intervertebral disc; lumbar spine; magnetic resonance imaging (MRI).

Low back pain (LBP) is a common health problem with appreciable socioeconomic effects resulting from morbidity, increased health costs and lost productivity. Degenerative changes of the lumbar spine have generally been accepted as one of the major causes of LBP. In addition to the disc alterations, degeneration also occurs in the nondiscal structures of the spinal column, such as the facet joints, paraspinal muscles and spinal ligaments.

The interspinous ligament (ISL) is one of the major components of the posterior ligamentous system in the spinal column. Some authors consider the ISL to play an important role in spinal stability by resisting the separation of the spinous processes and opposing lumbar flexion.[1-4] Degenerative changes of the ISL start as early as the late second decade. When bending, the change in loading moves posteriorly and the posterior elements of the lumbar spine become denser with greater load bearing. The ISL is the first to be injured in flexion with sufficient force, resulting in an unstable spinal column.[1,5] However, previous studies have usually ignored interspinous ligament degeneration (ISLD) due to the diagnostic limitations. Indeed, plain radiography or computed

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tomography simply reveals ligamentous disruption by indirect signs, such as a widening/splaying of the spinous processes and/or facet diastasis.[6]

Recently, due to superior soft tissue delineation and multiplanar capabilities, magnetic resonance imaging (MRI) is considered the modality of choice in assessing ligamentous pathology.[7] According to the signal intensity, Fujiwara et al. described an MRI-based classification correlated with the specific histological findings of the ISL.[8] Keorochana et al. modified Fujiwara's criteria into four types (Table 1).[9,10] However, these studies referred to ISLD only in patients with LBP. Similar to disc degeneration, such changes in the ISL may also be observed in the asymptomatic population; however, little information is available. Additionally, the influence of the ISL on the cascade of disc degeneration and the interactions of these processes is still uncertain.

The aim of this study was to investigate the frequency and distribution of ISLD in symptomatic and asymptomatic populations and to correlate these findings with disc degeneration on MRI.

Patients and methods

This cross-sectional, frequency-matched, case-control study was carried out at a tertiary spine care center with the approval of the Ethics Committee of the university hospital. All participants were provided oral and written explanations on the details of the present study and a written informed consent was obtained from each participant. The recruitment period was one year; from July 2008 to June 2009.

A total of 178 eligible patients who complained of LBP with or without radicular pain for at least 6 months were identified. Patients with (1) history of previous spine surgery or traumatisms, (2) psychiatric diseases or symptoms, (3) rheumatic disease or inflammatory spine diseases such as ankylosing spondylitis or spondyloarthritis, (4) comorbidities such as diabetes and (5) other MRI features considered to be a potential source of non-discogenic LBP, such as disc herniation with symptomatic root compression, spinal stenosis, spondylolisthesis, transitional lumbosacral vertebra or degenerative scoliosis were excluded. A total of 60 patients (34 males, 26 females; mean age: 46.2±7.8 years, range: 23 to 74 years) with LBP were enrolled in this study as the symptomatic group (Group A). The average visual analog scale (VAS) score in Group A was 5.6±2.6.

Asymptomatic control patients were screened from 376 individuals presenting to our hospital for routine health examinations during the same period and recruited from informational bulletins placed in the Department of Radiology at our hospital. Subjects with no history of relevant LBP or related complaints (e.g. no medical consultation or work absence because of LBP) were considered as potential controls. Forty-eight individuals (12.8%) refused to participate in further clinical examination and MRI. Volunteers with structural spinal abnormalities found on MRI were also excluded. Asymptomatic individuals had a comparable risk factor profile in terms of age (±3 years), gender, smoking and physical job characteristics with the eligible patients in Group A. A total of 60 patients (34 males, 26 females; mean age: 46.9±8.4 years, range: 20 to 76 years) were included in the asymptomatic group (Group B).

Table 1. The grading system of the ISLD on MRI.[9,10]

Type	Characteristics
A	Mixed or low signal intensity on both T1- and T2-weighted MRI
B	High signal intensity on both T1- and T2-weighted MRI
C	Low signal intensity on T1-weighted and high signal intensity on T2-weighted MRI
D	Iso- or low signal intensity on both T1- and T2-weighted MRI with spinous process hypertrophy or narrowing of interspinous space

All examinations were performed on a 1.5 Tesla lumbar coil MRI (Siemens AG, Munich, Germany). The imaging protocol included sagittal T1-weighted (TR: 560 msec, TE: 12 msec) and T2-weighted spin echo sequence (TR: 3000 msec, TE: 100 msec) with the following parameters; matrix: 512×225, field of view: 300×225 mm, thickness: 4.0 mm, interslice gap: 1.0 mm and number of excitations: 3. MRI was performed with the patient in the supine position so the ligament was not tensioned.

Images were read blindly by two spine surgeons (one junior fellow and one consultant). Each observer analyzed all images on two separate occasions with a minimum interval of 4 weeks. To obtain the reference grades for the second part of the study, a consensus opinion was reached in all cases of disagreement after the data were collected.

Two parameters (ISL and intervertebral disc) were graded according to degeneration at four lumbar spinal levels (L2-3, L3-4, L4-5, and L5-S1). On the basis of a comprehensive literature review, ISLD was divided into 4 categories using the criteria proposed by Fujiwara et al.[8] and modified by other authors.[9,10] Type A represented 'normal', Type B 'mild', Type C 'moderate' and Type D 'severe' (Table 1).

Table 2. MRI classification of disc degeneration.[11]

Grade	Disc structure	Distinction between nucleus and annulus	Signal intensity	Disc height
1	Bright white, homogeneous	Clear	Hyperintense	Normal
2	Inhomogeneous with or without horizontal bands	Clear	Hyperintense	Normal
3	Gray, inhomogeneous	Unclear	Intermediate	Normal to slightly decreased
4	Gray to black, inhomogeneous	Lost	Intermediate to hypointense	Normal to moderately decreased
5	Black, inhomogeneous	Lost	Hypointense	Collapsed

Table 3. Frequency of ISLD grade at each spinal level.

Level	Group A				Group B			
	Type A	Type B	Type C	Type D	Type A	Type B	Type C	Type D
L2-3	56*	3	1	0	53†	6	1	0
L3-4	43	8	5	4	46	12	2	0
L4-5	33	13	7	7	40	11	8	1
L5-S1	29	26‡	3	2	33	21§	4	2

*Significantly greater prevalence at L2-3 ($p<0.01$ vs. L3-4, L4-5, and L5-S1).
†Significantly greater prevalence at L2-3 ($p<0.01$ vs. L4-5 and L5-S1).
‡Significantly more prevalence at L5-S1 ($p<0.05$ vs. L2-3, L3-4, and L4-5).
§Significantly more prevalence at L5-S1 ($p<0.01$ vs. L2-3, $p<0.05$ vs. L4-5).

The grading system of disc degeneration was a five-point scale designated by Pfirrmann et al.[11] It was determined by the disc structure, distinction between the nucleus and annulus, signal intensity and disc height on T2-weighted sagittal images (Table 2). Grade 1 indicated normal whereas Grade 5 corresponded to the end-stage degeneration.

Interobserver and intraobserver reliability were calculated using Cohen's kappa method.[12] The interpretation of reliability coefficients were performed as follows: kappa 0 to 0.20 showed slight agreement, 0.21 to 0.40 fair agreement, 0.41 to 0.60 moderate agreement, 0.61 to 0.80 substantial agreement, and 0.81 to 1.0 excellent agreement.[13] The frequency of the ISLD type at each spinal level was analyzed using the chi-square test. The relationship of the ISLD type with age was evaluated using the Spearman correlation test. The correlation between the ISLD and disc degeneration was examined using the Mann-Whitney U test. Statistical analyses were performed using SPSS v.14, (SPSS Inc., Chicago, IL, USA) and a p value of less than 0.05 was considered statistically significant.

Results

Intraobserver agreement was substantial or excellent, with a kappa value of 0.79 to 0.87 for the ISLD and 0.72 to 0.88 for disc degeneration. As expected, the interobserver agreements were lower than intraobserver agreements for both ISLD and disc degeneration, with kappa values between 0.74 and 0.78 (substantial) and 0.69 and 0.74 (substantial), respectively.

The frequency of each ISLD type is shown in Table 3. ISLD distribution was similar in Group A and B. More advanced ISLD grades were less commonly observed.

Type A was the most prevalent type, with 161 levels (67.1%) in Group A and 172 (71.7%) in Group B. Type A was significantly more common at L2-3 when compared with the other levels in Group A ($p<0.01$), and L4-5 and L5-S1 in Group B ($p<0.01$).

Type B was commonly observed at L5-S1, with a statistical difference when compared with the other levels in Group A ($p<0.05$), and L2-3 ($p<0.01$) and L4-5 ($p<0.05$) in Group B.

For Type C, there was no statistical difference between each level, with the exception of L2-3 and L4-5 ($p<0.05$) in Group B.

Type D was the least common type and often occurred at the lower lumbar spine (L4-5 and L5-S1). Type D was significantly more prevalent in Group A than in Group B (5.4% vs. 1.3%, $p<0.05$) (Fig. 1). Type D (13 levels in Group A and 3 in Group B) was observed only in patients older than 65 years. The average age of Type D in Group A was significantly greater than other

Table 4. Average age in each ligament type in Group A and Group B.

Type	Average Age (yrs)	
	Group A	Group B
	Mean±SD	Mean±SD
A	46.14±12.91	44.67±9.40
B	43.76±10.34	46.52±9.66
C	46.38±9.40	47.46±7.38
D	55.85±12.82*	53.43±10.18†

*The average age of Type D was significantly higher than that of Type A and B ($p<0.01$) and Type C ($p<0.05$).
†The average age of Type D was significantly higher than that of Type A ($p<0.05$).

Table 5. Disc degeneration grade in each ligament type.

		Ligament Type			
		Type A	Type B	Type C	Type D
		Mean±SD	Mean±SD	Mean±SD	Mean±SD
Disc Degeneration	Group A	3.41±0.79	3.38±0.72	3.77±0.60	3.82±0.72*
	Group B	2.89±0.76	2.98±0.76	3.07±0.77	3.29±0.76

*The grading of disc degeneration in Type D was significantly higher than that in Type A and B ($p<0.05$).

types ($p<0.01$ vs. Type A and B, and $p<0.05$ vs. Type C). In Group B, there was a significant increase in age when Type D was compared to Type A ($p<0.05$) (Table 4).

The relationship between ISLD and disc degeneration is shown in Table 5. Although there tended to be increased disc degeneration grade in advanced ISLD in both groups, this difference was only significant between Type D and Types A and B in Group A ($p<0.05$).

Discussion

Spinal degeneration is considered an underlying etiology and source of spinal disability. Degenerative changes, including those of the disc and nondiscal structures, have been categorized into 3 consecutive functional phases; temporary dysfunction, instability and re-stabilization.[14-16]

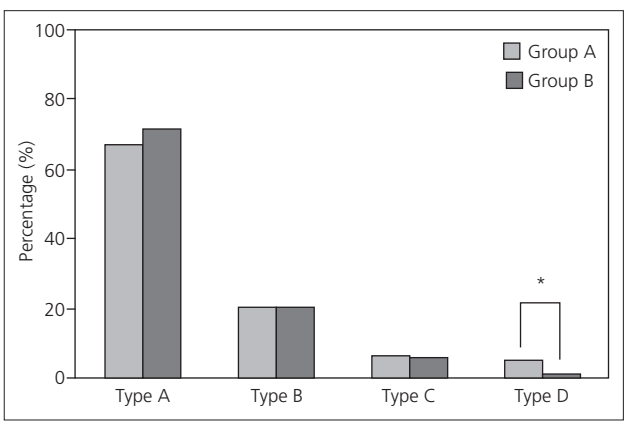

Fig. 1. The frequency of each type of ISL in Group A and Group B. Note: Type D was significantly more common in Group A than that in Group B (*$p<0.05$).

A grading system for ISLD was first described by Fujiwara et al.[8] and has since been modified.[9,10,17] Based on previous well-correlated radio-pathological studies, this classification represents the proper stages of spinal degeneration. Kong et al. performed kinetic MRI to investigate the segmental motion of the lumbar spine and found that the motion tended to increase in the early phases of ISLD and decrease in late phases in translation and angular planes.[17] Similar findings were also reported by Keorochana et al.[10] The authors considered that graduated inhibition of segmental motion of the lumbar spine may be interpreted by the features of ISLD, including the vertical plane collapse, spinous process hypertrophy and interspinous space narrowing.

In the current study, the grading system of ISLD was revealed to provide sufficient reliability and reproducibility. Currently, only one study has been reported evaluating intra- and interobserver agreements. Keorochana et al. found that a difference of 1 grade occurred more often than that of 2 grades, particularly between Types A and B.[9] The frequent misinterpretation may be explained by the high percentage of these two types and the difficulty in discriminating between bright (Type B) and intermediate signal intensities (Type A) on their 0.6 Tesla MRI. Furthermore, the disagreement was relatively less frequent in Type D ISL, mainly due to the easier

identification of the specific characteristics of the interspinous space and spinous process.

The clinical significance of the ISL has been elucidated in a number of studies. It has been reported that the ISL is often transected in patients undergoing disc surgery and that disc herniation may be secondary to the ligamentous damage.[5,18] In a review of thousands of plain radiography in patients with LBP, Scapinelli[19] found that the interspinous space diminishes both from the enlargement of the spinous process and exaggeration of the lumbar lordosis. Histological examination also revealed that a degenerative ISL with elastic fiber hyperplasia occurs frequently in elderly subjects. Moreover, the ligamentous pathology is thought to be related with increased nociception.[20,21] In a prospective observational study, Jinkins[22] demonstrated that intrinsic spinal muscle degeneration is relatively rare, but all cases of intrinsic spinal muscle degeneration had associated ISLD. The author considered that both acute and chronic ISL rupture may theoretically result in LBP. Recently, Tsao et al. applied a single bolus injection of hypertonic saline into the L4-5 ISL or paraspinal muscle in 10 volunteers and reported that the pain response, including acute and consistent LBP, was of longer duration and greater intensity and size after ISL injection.[23]

However, the cascade of ISLD has not been well documented. Fujiwara et al. reported that two-thirds of ISLD in patients with LBP present with a signal intensity similar to that of Type A or B ISLD in our study, and is mostly observed at L1-2 or L2-3.[8] This is consistent with previous findings that no ISL ruptures occurred at the upper lumbar levels.[24] Keorochana et al.[10] reported Type A and B ISLD to be most prevalent at L2-3 and L5-S1, respectively. The authors suggested that the upper lumbar levels are the least affected sites, whereas the lower levels that experience the greatest amount of loading and motion may develop a severe degree of degeneration. The signal intensity in Type C was mimicked as that in the interspinous bursitis, with a pathological correlation of increased vascularity, eburnation, and bursa formation.[21] As an inflammatory stage, the incidence of Type C was less than 10%. In addition, Type D represented the end-stage of degeneration, which was rarely identified in patients.

The frequency and distribution of ISLD in patients with LBP in this study were consistent with previous reports.[8,10] To the best of our knowledge, however, little information is available in the asymptomatic population. Our results showed that the tendency of ISLD in asymptomatic subjects was similar to those with LBP, with the more advanced ISLD grade less commonly observed. When comparing the two groups, it was noteworthy that Type D was less prevalent in asymptomatic subjects. Furthermore, all Type D cases were in elderly patients and the average age of patients with this type was much higher than that of other types. This can likely be explained by the same hypothesis applied to the previous findings: various alterations, including biochemical, morphological and mechanical changes, occurred in the spinal ligament as a patient aged.[25-27] In addition, at the end-stage of degeneration, Type D required more time to develop.

Studies evaluating the degenerative changes in the same spinal unit found a strong correlation between ISLD severity and advanced disc degeneration.[8,10,17] It was considered that a consonant loss of interspinous space may occur with increased axial loading stresses on the remnants of the ISL when the pathological aberration of the ISL is accompanied by a progressive height loss in the disc.[22] As a whole spinal unit, ISLD might adversely affect disc degeneration and vice versa. In this study, although there tended to be increased disc degeneration grade in severe ISLD, there was no statistical significance in the asymptomatic population.

Possible limitations of this study include the exclusion of ISLD at the L1-2 level as the narrowing of the interspinous space nearer the adjacent apices makes observation at this level on MRI less evident.[28] Second, although we evaluated the relationship between ISLD and disc degeneration, we were unable to determine which one played a predominant role in patients' symptoms. Further investigations are warranted to determine its definitive role in LBP and the specific clinical correlation of different types of the ISLD. Third, although the sample size was sufficient, the subgroup (Type D ISLD) was relatively small. Further study is strongly needed with a larger sample size in the future.

In conclusion, the MRI-based grading system of ISLD appears to provide sufficient reliability and reproducibility. The tendency of ISLD in asymptomatic population was similar to that in patients with LBP, with the more advanced ISLD grade less commonly observed. However, advanced alteration of the ISL was more frequent in patients with LBP. Our findings also suggested that ISLD occurred in more severe disc degeneration.

Conflicts of Interest: No conflicts declared.

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