LIGAMENTUM FLAVUM HYPERTROPHY IN ELDERLY PATIENTS WITH LOW BACK PAIN: A MRI STUDY

Abstract

Introduction: Lumbar spinal canal stenosis (LSCS) is the most common spinal disorder in elderly patients. Ligamentum flavum (LF) hypertrophy contributes to the development of this disorder. We examined the correlations between LF thickness, measured on the magnetic resonance images, and age, gender, LSCS, disc degeneration, and disc herniation.

Materials and Method: Three hundred and forty patients complaining of low back pain were evaluated (mean age: 57.8±11.2 years). LF thickness was measured at the L2-3, L3-4, L4-5, and L5-S1 levels using axial T1-weighted magnetic resonance images at the facet joint level. All patients were examined for spinal stenosis, disc degeneration, and disc herniation.

Results: LF thickness was significantly greater in the LSCS group at the L2-3, L3-4, and L4-5 levels (p<0.05). LF thickness at all levels increased with age (p<0.05). No effect of gender was observed on LF thickness (p>0.05). LF thickness at all levels was significantly greater in patients with degeneration of Pfirrmann grades IV-V than in those with degeneration of grades I-III (p<0.05). There was no association between lumbar disc herniation and LF thickness at the lumbar intervertebral levels (p>0.05).

Conclusion: In elderly patients, LF hypertrophy was correlated with age, LSCS, spinal level, and disc degeneration, and not with disc herniation and gender.

Key Words: Ligamentum Flavum; Intervertebral Disc Degeneration; Spinal Canal; Magnetic Resonance Imaging.
**INTRODUCTION**

Lumbar spinal canal stenosis (LSCS) is one of the most common spinal disorders in elderly patients (1-4). Ligamentum flavum (LF) hypertrophy is considered a major contributor to the development of LSCS. LF thickening can decrease the spinal canal diameter, thereby compressing the dural sac and nerve roots. This compression can cause symptoms even in the absence of osseous spurs, herniated nucleus pulposus, or a bulging annulus fibrosus (1,2,5,6). The only therapeutic maneuver for these patients is surgical removal of the hypertrophied ligament. Reducing its thickness might increase the segmental space available for the dural sac (5).

The vertebral column comprises separate functional vertebral units. Apart from the vertebral bone, each unit also contains facets joints, intervertebral discs, and ligaments (7). LF extends from the second cervical vertebra to the first sacral vertebra, covering the posterior and lateral walls of the spinal canal (1,2,5,8,9). The posterior ligamentous complex of the spinal column—where LF is prominently involved in constraining posterior flexion—plays an important role in the stabilization of segmental movements (7). As a connective tissue, LF affects and/or controls the intervertebral movements and is also involved in the maintenance of smooth surfaces of the posterior dural sac (1,6).

Histological studies have demonstrated that LF hypertrophy is mainly caused by fibrosis (4,9,10). Although factors such as aging, mechanical stress, and physical activity are involved in LF hypertrophy, the precise pathomechanisms underlying LF hypertrophy remain unknown (2,6,9-11).

We aimed to analyze the relationship of LF thickness, obtained using magnetic resonance (MR) imaging (MRI), with age, gender, spinal level, disc degeneration, and disc herniation. We hypothesized that LF would be positively correlated with the aforementioned variables.

**MATERIALS AND METHOD**

**Patients**

This was a retrospective study, and the need for informed consent was waived by our institutional review board. All patients who were admitted to our hospital with low back pain and underwent MRI evaluation between January 2014 and December 2014 were recruited. The exclusion criteria were any history of lumbar spinal surgery, lumbar and sacral mass lesion, discovevertebral infection, lumbar vertebral fracture, current radiotherapy or spinal deformities such as spondylolisthesis and scoliosis. Three hundred and forty patients (mean age, 57.8±11.2 years) were included; among them, 205 (60.3%) were female and 135 (39.7%) were male.

**Imaging Parameters**

All MRI examinations were performed using a 1.5-T unit (Sigma; GE Medical System, Milwaukee, Wisconsin, USA), employing a phased-array coil. The imaging protocol included:

- Sagittal T2-weighted fast spin echo (FSE): (TR: 2996 ms, TE: 105.98 ms, matrix: 30x30 cm, slice thickness: 4 mm)
- Sagittal T1-weighted FSE: (TR: 668 ms, TE: 7.93 ms, matrix: 30x30 cm, slice thickness: 4 mm)
- Axial T1-weighted FSE: (TR: 500 ms, TE: 8.97 ms, matrix: 18x18 cm, slice thickness: 4 mm)
- Axial T2-weighted FSE: (TR: 4431 ms, TE: 98.48 ms, matrix: 19x19 cm, slice thickness: 4 mm) for all MRI examinations.

**Image Analysis**

A single radiologist retrospectively evaluated MR images of the patients using our Picture Archiving and Communication System (PACS). The thickness of LF was measured at 4 levels (L2-3, L3-4, L4-5, L5-S1). Also, intervertebral disc degeneration, anteroposterior diameter, and the area of the lumbar spinal canal and disc degeneration were evaluated at these levels for all patients. LF thickness at each intervertebral level (L2-3, L3-4, L4-5, and L5-S1) was obtained by measuring it at half the length of LF on axial T1-weighted MR images at the facet joint level, using the measuring tool of the PACS (Figure 1). If LF was asymmetrical, we used the thicker one.

The severity of the intervertebral disc degeneration was graded for the four different intervertebral levels (L2-3, L3-4, L4-5, and L5-S1). The evaluation was performed using T2-weighted midsagittal images, employing the five-grade classification introduced by Pfirrmann et al. (12) (Table 1). LF thicknesses were compared between degeneration grade I–III group and grade IV–V group.

The anteroposterior diameter of the spinal canal and the area of the canal for each intervertebral level were measured on the axial T2-weighted images at the midline between the posterior margin of the disc and the anterior border of the vertebral arch. The spinal canal area was obtained as the area of the projection of the canal onto the plane of the facet joint. LSCS was assumed if the anteroposterior diameter of the spinal canal was less than 11.5 mm or the area of the spinal canal was less than 1.5 cm² (13).
To assess the reliability of the measurements, the same radiologist performed the measurements twice, with an interval of 3 weeks, in 100 lumbar spines randomly selected from a 340-spine collection.

**Statistical Analysis**

Statistical analyses were evaluated using the SPSS (v15.0; SPSS Inc., Chicago, IL) program. We tested the relationship between LF thickness and the age of the patients with Student’s t-test. The relationship between LF thickness and the spinal level was evaluated using Pearson’s correlation test. The independent-samples t-test was used for the relationships between LF thickness and gender, LSCS, disc degeneration, and disc herniation. p < 0.05 was considered statistically significant. The intra-class correlation coefficient (ICC) was used to estimate the intra-observer reliability for LF thickness evaluation.

**RESULTS**

LF thickness were greater in the LSCS group at levels of L2-3, L3-4, and L4-5, but not at L5-S1, compared with the group without LSCS (p<0.05) (Table 2).

The thickness of LF, starting from the level of L2-3, up to L4-5 tended to increase (p<0.05). LF thickness in study group was 3.49±0.56 mm, 3.86±0.63 mm, 4.15±0.72 mm, and 3.07±0.5 mm for the L2-3, L3-4, L4-5, and L5-S1 levels, respectively. It was the greatest at L4-5.

LF thickness increased by age group (p<0.05) (Table 3). No effect of gender on LF thickness was observed (p>0.05).

**Table 1— Classification of Disc Degeneration**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Signal Intensity of The Nucleus Pulposus and Structure</th>
<th>Distinction of Nucleus and Anulus</th>
<th>Height of Intervertebral Disc</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Homogenous, bright white; hyperintense to isointense to CSF</td>
<td>Clear</td>
<td>Normal</td>
</tr>
<tr>
<td>II</td>
<td>Inhomogeneous with or without horizontal bands; hyperintense to isointense to CSF</td>
<td>Clear</td>
<td>Normal</td>
</tr>
<tr>
<td>III</td>
<td>Inhomogeneous, gray, intermediate to CSF</td>
<td>Unclear</td>
<td>Normal to slightly decreased</td>
</tr>
<tr>
<td>IV</td>
<td>Inhomogeneous, gray to black; hypointense to CSF</td>
<td>Lost</td>
<td>Normal to moderately decreased</td>
</tr>
<tr>
<td>V</td>
<td>Inhomogeneous, black; hypointense to CSF</td>
<td>Lost</td>
<td>Collapsed disc space</td>
</tr>
</tbody>
</table>

CSF: Cerebrospinal fluid

**Table 2— Mean Thickness of the LF in with and without Lumbar Spinal Canal Stenosis (LSCS) Group [mean thickness of the LF (mm) ±sd]**

<table>
<thead>
<tr>
<th>Lumbar Level</th>
<th>Group With LSCS</th>
<th>Group Without LSCS</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>L2-3</td>
<td>3.37±0.66</td>
<td>3.15±0.48</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>L3-4</td>
<td>3.77±0.63</td>
<td>3.58±0.52</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>L4-5</td>
<td>4.23±0.78</td>
<td>3.90±0.54</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>L5-S1</td>
<td>3.02±0.48</td>
<td>2.95±0.45</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>
LF thickness in patients with degeneration of grades IV-V was significantly greater than that in degeneration of grades I-III (p<0.05) (Table 4). At L2-3, L3-4, L4-5, and L5-S1, no significant differences were found between LF thicknesses in the individuals with and without lumbar disc herniations (p>0.05).

The ICC for intra-observer reliability was 0.89 for LF thickness.

**DISCUSSION**

The main finding of the present study was the significantly higher LF thickness among subjects with LSCS. We also observed a positive correlation between LF thickness and age, spinal level, and disc degeneration among elderly patients. Overall, LF thickness did not depend on gender or disc herniation.

The pressure exerted on the nerves by the adjacent tissues such as the intervertebral disc, LF, or facet joint is considered the major cause of low back pain or leg pain in elderly patients (6,8,11,14). LF hypertrophy was first reported in 1913 as a cause of stenosis leading to the low back pain (15). Computed tomography and MRI study evaluating LF thickness have indicated that LF is one of the significant factors responsible for compression (3,5,10,16,17).

LF, a key spinal ligament, is also called the yellow ligament because it is rich in yellow elastin. The normal LF is a well-defined elastic structure composed of 80% of elastic fibers and 20% of collagen fibers (3,6,9). The increased collagen-to-elastin ratio has also been considered a sign of developing LSCS in elderly patients (4,10).

Aging affects all the structures of the spine (18). We observed that LF thickness at all the lumbar levels increased with age. This observation was in agreement with the previous research suggesting that LF thickness is age dependent (1,5,10,17,19,20). Abbas et al. (5) have reported that significant changes in LF thickness after the age of 60 occur only at L3-4. In our study, we observed a significant increase in LF thickness at all levels after the age of 70. In addition, in agreement with the previous findings, we found no association between gender and LF thickness (1,5,19).

LF thickness increases cranio-caudally except for L5-S1 (5). We found significant differences between LF thicknesses at each level. LF at L5-S1 was thinner than at the other levels. It was thickest at the level L4-5 and thicker at L4-5 and L3-4 than at L2-3 and L5-S1. Our results for the spinal level were in agreement with those of the previous reports (1,2,5,19).

Disc degeneration and LF thickening are very common conditions in the aging population (2). Degenerated discs provide to release inflammatory cytokines, which contribute

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**Table 3**—Mean Thickness of the LF at Each Spinal Level in Patient with Low Back or Leg Pain Distributed by Age [mean thickness of LF (mm) ±sd]

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>n</th>
<th>L2-3</th>
<th>L3-4</th>
<th>L4-5</th>
<th>L5-S1</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>40-49</td>
<td>95</td>
<td>2.78±0.29</td>
<td>3.25±0.37</td>
<td>3.58±0.37</td>
<td>2.69±0.36</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>50-59</td>
<td>88</td>
<td>3.14±0.22</td>
<td>3.58±0.42</td>
<td>3.91±0.53</td>
<td>2.91±0.56</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>60-69</td>
<td>99</td>
<td>3.28±0.33</td>
<td>3.66±0.42</td>
<td>4.04±0.48</td>
<td>3.06±0.34</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>70-79</td>
<td>46</td>
<td>3.72±0.51</td>
<td>4.26±0.60</td>
<td>4.70±0.84</td>
<td>3.84±0.54</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>80 and over</td>
<td>12</td>
<td>4.49±0.68</td>
<td>4.61±0.41</td>
<td>4.73±0.21</td>
<td>3.46±0.30</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

**Table 4**—Mean Thickness of the LF in Grades IV to V and Grades I to III Disc Degeneration [mean thickness of the LF (mm) ±sd]

<table>
<thead>
<tr>
<th>Lumbar Level</th>
<th>Grade IV-V Disc Degeneration</th>
<th>Grade I-II Disc Degeneration</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>L2-3</td>
<td>3.25±0.56</td>
<td>2.90±0.27</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>L3-4</td>
<td>3.68±0.57</td>
<td>3.27±0.41</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>L4-5</td>
<td>4.04±0.64</td>
<td>3.57±0.41</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>L5-S1</td>
<td>3.01±0.45</td>
<td>2.56±0.32</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>
to LF hypertrophy (14). Sakamaki et al. (2) have found no correlation between LF thickness and decrease in the disc height in elderly patients. In similarity with the findings of Altinkaya et al. (1), we found that LF at all the levels was significantly thicker in patients with degeneration of grades IV-V than in those with degeneration of grades I-III.

Lumbar disc degeneration, a commonly encountered reason of lower back pain, is mostly caused by the herniation of the spinal discs (20). Degenerative LSCS is described as a narrowing in the spinal canal and/or intervertebral neural foramina. It can occur as a consequence of progressive hypertrophy in surrounding ligamentous and osseocartilaginous elements (21). LSCS represents a significant cause of low back pain and/or leg pain and paresis (1,2,21). In our study, we found that LF was significantly thicker in the subjects with LSCS than in those without this disorder, at the levels of L2-3, L3-4, and L4-5. At the L5-S1 level, no significant difference was seen between these two groups. These results concur with the findings of the previous studies (1,5). However, our results do not support the findings of Abbas et al. (5). The study of Abbas et al. has suggested that this phenomenon is caused by the relative hypermobility of the L3-4 and L4-5 segments in comparison with the L5-S1 segment (which is stabilized by a large transverse process of the L5 vertebra and the iliolumbar ligaments). Tomkins-Lane et al. (22) have detected lumbar stenosis predominantly at the level L4-5, and at the level L3-4 in elderly patients. Our results suggest that the L4-5 and L3-4 levels are more susceptible to LF thickening.

In our study contrary to the findings by Altinkaya et al. (1), we found no correlation between lumbar disc herniation and LF thickness for any of the lumbar levels. Studies have found that LF in patients with LSCS is significantly thicker than in those with lumbar disc herniation (3,9,11,23). Spinal canal stenosis is a dynamic phenomenon that becomes apparent during spinal loading. Hansson et al. (24) have reported that in axially loaded spines, LF led to greater narrowing of the lumbar spinal canal than the disc herniation. Nguyen et al. (25) reported that by using upright MRI, LF was statically larger along all disc space levels in the patients with low back pain. For that reason, prospective studies investigating the effect of disc herniation on LF thickness are needed.

A limitation of the present study is its retrospective design. The subjects of this study presented with low back and/or leg pain. In addition, we had no data on subjects without low back or leg pain (potential control group). Our study focused on evaluating degenerative changes using transversal examination. Thus, our results might not reflect the actual nature and course of LF hypertrophy fully. The study would have benefited from the inclusion of a control group. The impact of this report would be increased by considering the level of pain, body mass index, MRI findings, and the level of physical activity for both the study group and the control group. Then, the necessary patient–control comparisons could have been performed. Additional studies are warranted to address these limitations.

In conclusion, LF is an important anatomical structure; its thickening can cause LSCS, resulting in low back pain or leg pain. We found that LF hypertrophy was associated with age, spinal level, disc degeneration, and LSCS in elderly patients. Gender and disc herniation were not correlated with LF thickness.

The authors declare that they have no conflict of interest.

REFERENCES