Clinical Study

ASJ Asian Spine J. 2024;18(3): 425-434
https://doi.org/10.31616/asj.2023.0064
PISSN 1976-1902 • eISSN 1976-7846

Association between ligamentous stenosis at spondylolisthetic segments before fusion surgery and symptomatic adjacent canal stenosis at follow-up in patients with degenerative spondylolisthesis

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Received Feb 28, 2023; **Revised** Jul 4, 2023; **Accepted** Jul 9, 2023
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Study Design: A retrospective case-control propensity score-matching study.
Purpose: This study aimed to longitudinally evaluate whether preoperative ligamentous stenosis at the spondylolisthetic segments could affect the incidence of symptomatic adjacent canal stenosis following one-segment fusion surgery.
Overview of Literature: Several risk factors for symptomatic adjacent canal stenosis following fusion surgery have been assessed. Patients with lumbar canal stenosis mainly due to ligamentum flavum (LF) hypertrophy (ligamentous stenosis) also have LF hypertrophy in other segments.
Methods: In total, 76 patients participated in this case-control study (neurologically symptomatic adjacent canal stenosis, n=33; neurologically asymptomatic cases at follow-up, n=43). Their risk factors during surgery and magnetic resonance (MR) images before the surgery and at follow-up were evaluated. Data from the two groups (n=25 each) were matched using propensity scores for age, sex, time to MR imaging at follow-up, surgical procedure, and LF hypertrophy in adjacent segments before the surgery and analyzed.
Results: Compared with the asymptomatic group, the symptomatic adjacent canal stenosis group had a significantly larger LF area/spinal canal area in the spondylolisthetic segments before the surgery. During the follow-up periods (in months), they had a larger LF area/spinal canal area in the adjacent segments: the two values were significantly correlated. The sensitivity, specificity, and positive and negative predictive values for determining symptomatic adjacent canal stenosis were high compared with on the cutoff value for the LF area/spinal canal area at the spondylolisthetic segments before the surgery. These results were the same after matching.
Conclusions: Symptomatic adjacent canal stenosis is mainly caused by LF hypertrophy. Ligamentous stenosis at the spondylolisthetic segments before fusion surgery might be strongly associated with symptomatic adjacent canal stenosis at follow-up.

Keywords: Adjacent segment disease; Spinal stenosis; Spondylolisthesis; Flaval ligament; Spinal fusions

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Introduction

Neurologically symptomatic canal stenosis in adjacent segments can compromise long-term clinical results following fusion surgery in patients with degenerative spondylolisthesis. Therefore, risk factors for symptomatic adjacent canal stenosis following fusion surgery must be evaluated clinically.

Previous studies have evaluated several risk factors for symptomatic adjacent disease following surgery, including patient factors (older age, higher body mass index [BMI], and global malalignment) and radiographic factors in adjacent segments before the surgery (disc and facet degeneration, facet tropism, and horizontal lamina). These previous studies have also assessed surgical factors such as long fusion, posterolateral lumbar fusion (PLF) or posterior lumbar interbody fusion (PLIF), and superior facet violation with pedicle screws [1-12]. These factors could increase mechanical stress on the adjacent segments. However, adjacent canal stenosis does not always occur because of mechanical stress. In addition, responses to the mechanical stress in adjacent segments might differ among individuals.

Sakai et al. [13] reported two types of lumbar spinal canal stenosis: one mainly due to ligamentum flavum (LF) hypertrophy (ligamentous stenosis) and the other due to causes other than LF hypertrophy (nonligamentous stenosis). Axial magnetic resonance (MR) images showed an increased LF area/spinal canal area in patients with ligamentous stenosis compared with those with nonligamentous stenosis [13]. LF hypertrophy-associated stenosis was found throughout the lumbar spine in patients with ligamentous stenosis. Therefore, ligamentous stenosis might have a genetic predisposition [13].

These findings suggested that the type of stenosis in spondylolisthetic segments before the surgery and adjacent segments at follow-up might be the same. The etiology of adjacent canal stenosis following fusion surgery remains unclear. However, if symptomatic adjacent canal stenosis is mainly ligamentous, then patients with ligamentous stenosis in spondylolisthetic segments have a stronger likelihood of developing adjacent canal stenosis following surgery than those with nonligamentous stenosis. In response to mechanical stress, patients with a predisposition to ligamentous stenosis in spondylolisthetic segments before fusion surgery are at greater risk of developing complications to adjacent segments. To date, no longitudinal analysis of adjacent segments according to the types of stenosis in spondylolisthetic segments has been conducted.

Thus, this case-control propensity score-matching study was conducted to investigate (1) whether LF hypertrophy could be the main cause of symptomatic adjacent canal stenosis, (2) whether the degree of LF thickening in spondylolisthetic segments before fusion surgery and adjacent segments at follow-up were related, and (3) whether a greater degree of LF hypertrophy in spondylolisthetic segments before fusion surgery was a risk factor for symptomatic adjacent canal stenosis at follow-up.

Materials and Methods

Ethical considerations

The study protocol was approved by the Committee on the Ethics of Human Research of Hamawaki Orthopaedic Hospital (approval no., 202007-15), and informed consent was obtained from all patients. All procedures were conducted in accordance with the 1964 Helsinki Declaration and its later amendments.

Patients

The medical records of 498 consecutive patients with degenerative spondylolisthesis treated with fusion surgery at one segment at Hamawaki Orthopaedic Hospital between 2003 and 2016 were retrospectively reviewed. Patients who were followed up clinically for at least 3 years (average follow-up time, 84.6±40.3 months) were included (n=352 [71%]). Patients who underwent decompression for pre-existing stenotic adjacent segments simultaneously with fusion surgery, as in cases of primary stenosis, were excluded from the study. Those who had coexisting degenerative scoliosis (Cobb angle at the standing radiograph ≥20°), history of lumbar surgery, or infection, tumor, rheumatoid arthritis, or major trauma were also excluded. Symptomatic cases at follow-up with causes other than adjacent canal stenosis (herniation and facet cysts) and those with facet violation in adjacent segments during surgery were also excluded. In total, 76 patients (55%) with follow-up MR images were enrolled, including 33 patients with neurologically symptomatic adjacent canal stenosis (symptomatic canal stenosis group) and 43 patients with neurologically asymptomatic stenosis (asymptomatic group). In both groups, MR images were taken before the surgery and at follow-up when neurological symptoms appeared in the symptomatic adjacent canal stenosis group and after 3 years in the

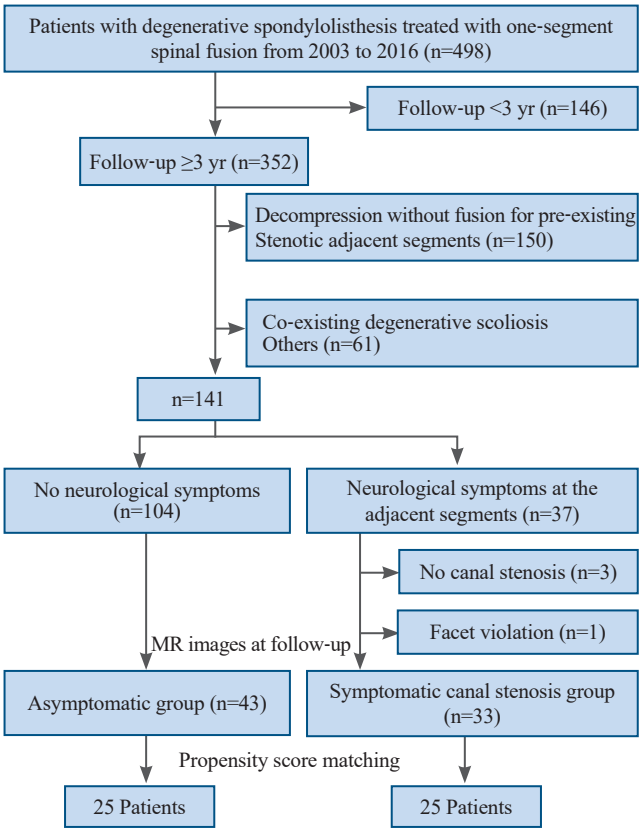


Fig. 1. Patient selection flowchart. MR, magnetic resonance.

asymptomatic group. To reduce confounding bias, data of 25 patients from each group, matched for age, sex, time from surgery to MR imaging at follow-up, surgical procedure (PLF or PLIF), and LF area/spinal canal area in adjacent segments before the surgery (caliper width: 0.2×standard deviation of the propensity score), were also analyzed (Fig. 1).

Facet violation was determined based on whether pedicle screws destroyed the facets on computed tomography (CT) scans within 1 month after the surgery. In all patients, damage to the upper intervertebral facets was avoided during fusion surgery to prevent denervation. In both groups, a Visual Analog Scale (VAS) questionnaire (scores of 0–10) was used to assess low back pain and pain in the buttocks and lower limbs before the surgery and at follow-up MR imaging.

Spinal fusion surgery

The patients underwent two types of spinal fusion surgery, namely, PLF or PLIF. In this series, PLF was conducted early and PLIF late. The techniques and instruments used for PLF and PLIF were identical. Although the surgeries were not performed by a single surgeon, they were all performed by the same surgical team.

Patient and surgical factors

The following factors were analyzed as possible confounding factors: age, sex, BMI, and lumbar lordosis (LL) at surgery, time to MR imaging at follow-up, surgical procedure (PLF/PLIF ratio), and fused level. LL was measured as the angle between the upper values of the L1 and S1 vertebrae in the standing lateral lumbar radiograph obtained before the surgery.

Radiographic factors

Disc degeneration, facet joint degeneration, angle between the facets, and translatory and rotatory displacements in spondylolisthetic segments were evaluated before the surgery, and the same factors along with facet tropism were assessed in adjacent segments before the surgery.

Before the surgery, disc degeneration was assessed using the Pfirrmann classification (1–5) on T2-weighted MR images in a midlateral slice [14]. Facet joint degeneration was assessed using the Weishaupt classification (0–3) on preoperative CT (TSX-021B/4A; Toshiba, Tokyo, Japan) [15]. The angle between the facets and facet tropism were measured on preoperative CT images. Translatory and rotatory displacements were measured as the difference in the slippage length and angle in the functional lateral lumbar radiographs using the methods described by White and Panjabi [16].

LF hypertrophy in spondylolisthetic and adjacent segments

The symptomatic adjacent canal stenosis level was adjacent to the fusion level. The levels at follow-up were dependent on the fusion level in the symptomatic adjacent canal stenosis group. In cases the fusion level was at L4–5, the adjacent level was L3–4 (L4–5 → L3–4 [upper segments]). Other levels were comparable (L3–4 → L4–5 [lower segments], L5–S1 → L4–5 [upper segments]). In the symptomatic adjacent canal stenosis group, 12 of 33 patients had double stenosis at follow-up. However, most stenotic segments showed the same level as above. Therefore, the adjacent levels in the asymptomatic group were determined in the same manner as in the symptomatic adjacent canal group.

The LF area/spinal canal area was evaluated in spondylolisthetic and adjacent segments using T1-weighted MR images in a slice at the level of the most stenotic dural sac area. The increase in LF hypertrophy during

follow-ups (months) in adjacent segments was calculated as follows: (LF area/spinal canal area before the surgery−LF area/spinal canal area at follow-up)/time to MR imaging at follow-ups.

MR images were captured digitally and viewed on a Synapse viewer (Fujifilm Medical Co., Tokyo, Japan). Measurements were obtained using digital measuring tools included in the software package. A single surgeon who was blinded to patient data performed the measurements. To calculate intraclass correlation coefficients, the evaluation was repeated for the same 50 patients in 1 month, and Pearson correlation coefficients were calculated.

Receiver operating characteristic analysis

To determine the cutoff value for predicting LF thickening in spondylolisthetic segments before the surgery for symptomatic adjacent canal stenosis, MR results were plotted on a receiver operating characteristic (ROC) curve, the area under the curve (AUC) and 95% confidence intervals (CIs) were calculated, and the cutoff value was obtained. Using the cutoff value, the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for determining symptomatic adjacent canal stenosis were calculated.

Statistical analysis

Between-group differences were determined using the paired or unpaired *t*-test or chi-square test. The correlation between the groups was analyzed using Pearson's method. Differences in variables with $p<0.05$ were considered significant. Statistical analysis and propensity-score matching were performed using SPSS Statistic for Windows ver. 14.0 (SPSS Inc., Chicago, IL, USA).

Results

Patient characteristics

In the symptomatic adjacent canal stenosis group before matching, all patients (n=33) had new neurological symptoms at follow-up (average time, 87.9±44.0 months after the surgery), even if they were asymptomatic for some time after the surgery. The symptoms included unilateral leg pain and cauda equina in 12 and 21 patients, respectively. Reoperation was needed in 22 patients, and conservative treatment was continued in 11. Among patients undergoing reoperation, 15 underwent decompression only and seven underwent fusion in addition to decompression. All 22 patients who underwent reoperation achieved some neurologic recovery following reoperation. Three of them underwent a third surgery at an average of 35.3±19.9 months following reoperation.

In the asymptomatic group, the average VAS scores for pains in low back, buttocks, and lower limbs were significantly lower at follow-up than before the surgery, before matching, and after matching. By contrast, no significant differences were found in the VAS scores in the symptomatic adjacent canal stenosis group (Fig. 2).

Patient and surgical factors

No significant differences were found between the two groups in terms of age, sex, time to MR imaging at follow-up, PLF/PLIF ratio, fused levels, BMI, and LL before and after matching (Table 1).

Radiographic factors

No significant differences were found between the two groups in terms of disc degeneration, facet degeneration, angles between the facets, translatory displacements, and rotatory displacements in spondylolisthetic segments and the same factors along with facet tropism in adjacent segments before the surgery, before matching, and after matching (Table 2).

LF hypertrophy

Intraclass correlation coefficients for the spinal canal and LF areas were 0.863 and 0.819, respectively. The symptomatic adjacent canal stenosis group had a significantly larger LF area/spinal canal area in the spondylolisthetic segments than the asymptomatic group before the surgery, before matching, and after matching (Fig. 3, Table 3).

No significant difference in the LF area/spinal canal area in adjacent segments before the surgery was observed between the two groups. Compared with the asymptomatic group, the symptomatic adjacent canal stenosis group had a significantly larger LF area/spinal canal area at follow-up and a greater increase in LF hypertrophy during follow-up in adjacent segments before and after matching (Fig. 3, Table 3).

Correlations between LF hypertrophy in spondylolisthetic segments and those at adjacent segments

In all patients, the LF area/spinal canal area in spon-

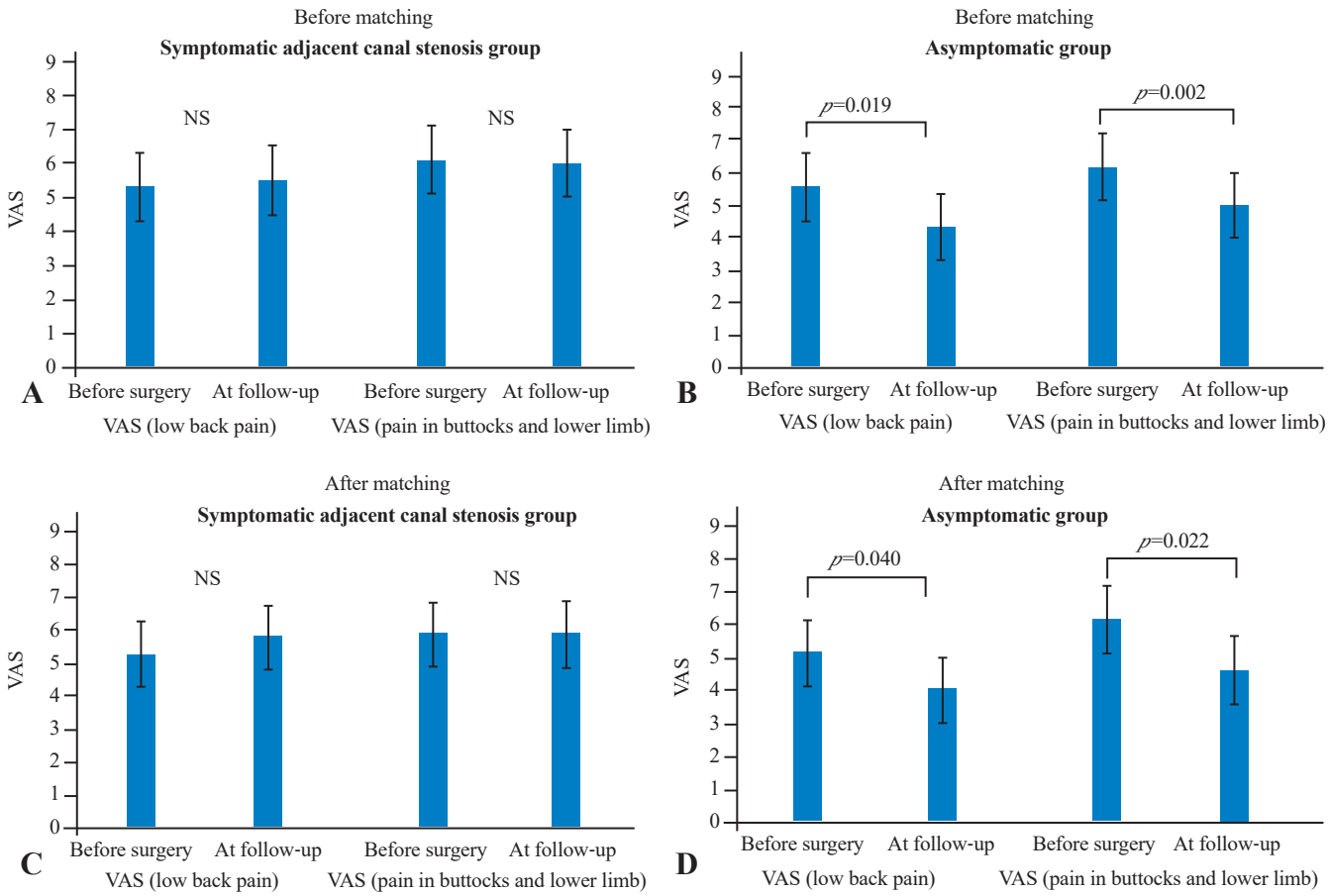


Fig. 2. The average Visual Analog Scale (VAS) scores for low back pain and pain in the buttocks and lower limb are significantly lower at follow-up than these before surgery in the asymptomatic group, before and after matching **(B, D)**. In contrast, no significant differences are found in the symptomatic adjacent canal stenosis group **(A, C)**. NS, not significant.

Table 1. Patient and surgical factors

Characteristic	Symptomatic adjacent canal stenosis					
	Before matching			After matching		
	+ (n=33)	– (n=43)	*p*-value	+ (n=25)	– (n=25)	*p*-value
Age (yr)	63.2±9.0	63.9±8.8	0.726	63.4±9.5	62.7±8.9	0.772
Sex			0.212			0.529
Male	12	10		6	8	
Female	21	33		19	17	
Time to MR images at FU (mo)	87.9±44.0	87.3±36.9	0.947	91.1±47.0	87.6±37.8	0.772
Types of spinal fusion surgery			0.578			0.564
PLF	19	22		14	16	
PLIF	14	21		11	9	
Fused levels			0.189			0.543
L3–4	5	2		4	2	
L4–5	26	40		19	22	
L5–S1	2	1		2	1	
Body mass index (kg/m^2)	24.7±3.5	23.9±4.1	0.355	24.6±3.8	23.9±4.5	0.582
Lumbar lordosis (º)	41.8±12.0	41.7±12.1	0.996	41.5±13.3	42.8±12.9	0.731

Values are presented as mean±standard deviation or number of patients.
MR, magnetic resonance; FU, follow-up; PLF, posterolateral lumbar fusion; PLIF, posterior lumbar interbody fusion.

Table 2. Radiographic factors before surgery

Variable	Symptomatic adjacent canal stenosis					
	Before matching			After matching		
	+ (n=33)	– (n=43)	*p*-value	+ (n=25)	– (n=25)	*p*-value
Spondylolisthetic segments						
Disc degeneration	3.5±0.6	3.4±0.5	0.922	3.4±0.6	3.2±0.5	0.310
Facet joint degeneration	2.8±0.4	2.7±0.5	0.139	2.9±0.3	2.7±0.5	0.215
Angles between the facets (°)	66.1±18.5	69.7±21.0	0.441	64.7±18.8	71.3±20.5	0.244
Translatory displacements (mm)	2.0±1.1	1.8±1.4	0.482	1.9±1.2	1.7±1.4	0.523
Rotatory displacements (°)	6.4±4.2	5.1±3.7	0.158	5.7±3.7	4.6±3.0	0.290
Adjacent segments						
Disc degeneration	2.9±0.5	2.6±0.6	0.051	2.8±0.4	2.6±0.6	0.089
Facet joint degeneration	1.2±0.5	1.1±0.5	0.627	1.2±0.5	1.2±0.6	0.602
Angles between the facets (°)	68.2±17.5	68.3±16.0	0.978	65.9±17.4	70.5±16.9	0.348
Facet tropism (°)	7.1±6.5	7.3±7.1	0.906	7.4±7.2	5.6±6.3	0.349
Translatory displacements (mm)	1.5±1.1	1.9±1.6	0.285	1.4±1.2	1.6±1.4	0.521
Rotatory displacements (°)	2.6±2.8	3.5±3.3	0.249	2.0±2.1	3.2±2.9	0.113

Values are presented as mean±standard deviation.

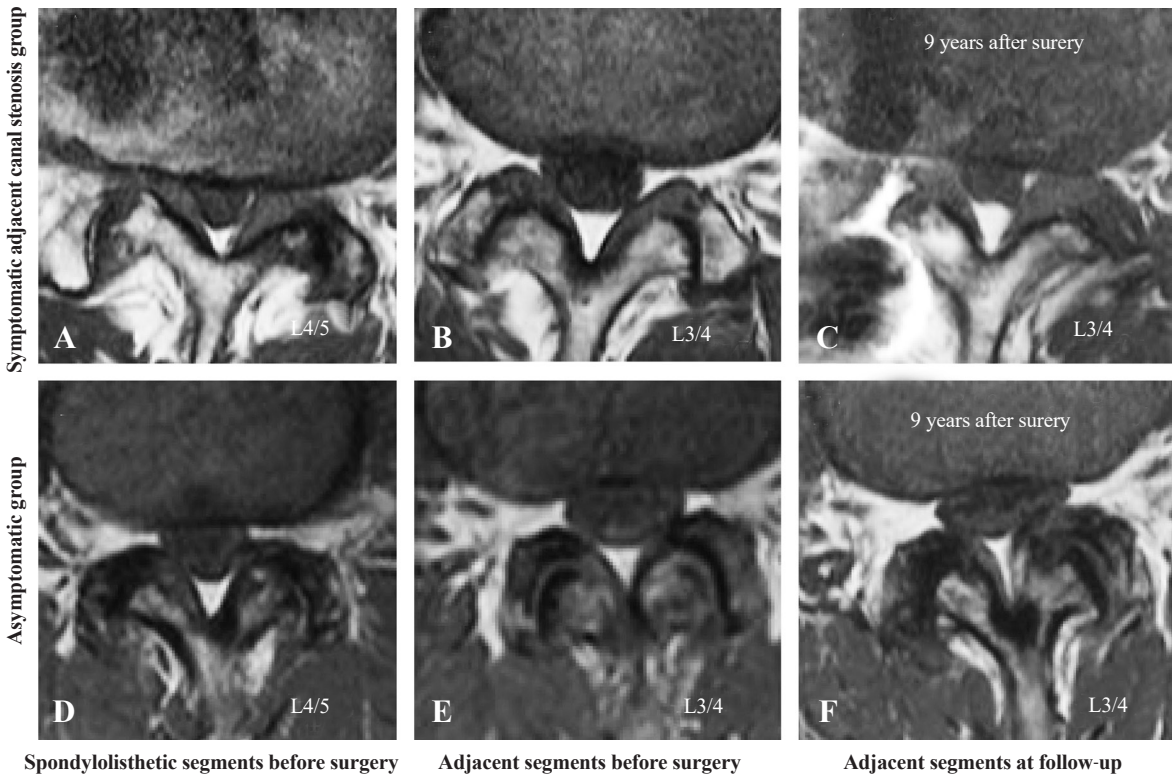


Fig. 3. The symptomatic adjacent canal stenosis group has a greater ligamentum flavum (LF) area/spinal canal area than the asymptomatic group at the spondylolisthetic segment **(A, D)**, and not pre-operatively **(B, E)** but at follow-up **(C, F)** at the adjacent segment.

dylolisthetic segments before the surgery significantly positively correlated with the LF area/spinal canal area at follow-up and increased LF hypertrophy during follow-up in adjacent segments before and after matching. In the symptomatic adjacent canal stenosis group, both correlations were observed before matching, and the latter was observed after matching. In the asymptomatic group, the former correlation was found before

Table 3. LF hypertrophy

LF area/spinal canal area	Symptomatic adjacent canal stenosis					
	Before propensity score matching			After propensity score matching		
	+ (n=33)	– (n=43)	*p*-value	+ (n=25)	– (n=25)	*p*-value
Spondylolisthetic segments before surgery: LF area/spinal canal area	0.55±0.11	0.40±0.11	<0.001	0.53±0.10	0.41±0.13	<0.001
Adjacent segments before surgery: LF area/spinal canal area	0.33±0.10	0.29±0.11	0.131	0.30±0.07	0.28±0.08	0.514
Adjacent segments at FU: LF area/spinal canal area	0.52±0.12	0.37±0.12	<0.001	0.49±0.11	0.39±0.13	0.008
Adjacent segments during the FU periods: increased LF area/spinal canal area, measured in months (×100)[a)]	0.29±0.24	0.10±0.18	<0.001	0.30±0.25	0.14±0.17	0.012

Values are presented as mean±standard deviation.
LF, ligamentum flavum; FU, follow-up.
[a)](LF area/spinal canal area before surgery–LF area/spinal canal area at the FU)/magnetic resonance imaging interval (mo).

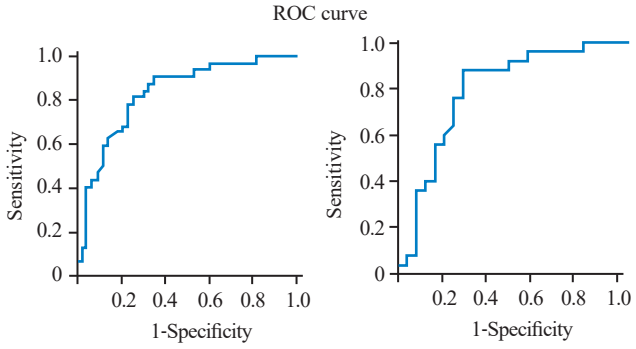
Table 4. Correlation between LF hypertrophy at spondylolisthetic segments and those at adjacent segments

Correlation with LF area/spinal canal area at the spondylolisthetic segments before surgery	Total patient group		Symptomatic adjacent canal stenosis group		Asymptomatic group	
	Correlation coefficient	*p*-value	Correlation coefficient	*p*-value	Correlation coefficient	*p*-value
Before matching						
LF area/spinal canal area at the adjacent segments at FU	0.578	<0.001	0.508	0.003	0.336	0.028
Increased LF area/spinal canal area, measured in months, at the adjacent segments during the FU periods	0.423	<0.001	0.405	0.019	0.113	0.472
After matching						
LF area/spinal canal area at the adjacent segments at FU	0.457	<0.001	0.353	0.083	0.334	0.103
Increased LF area/spinal canal area, measured in months, at the adjacent segments during the FU periods	0.399	0.004	0.459	0.021	0.109	0.603

LF, ligamentum flavum; FU, follow-up.

matching (Table 4).

No significant difference was observed between male and female patients or between PLF and PLIF with respect to the increase in LF hypertrophy during follow-up in adjacent segments in all patients before and after matching. Furthermore, in all patients, the increased LF hypertrophy during follow-up in adjacent segments did not correlate with other possible factors including other patients (excluding time to MR imaging at follow-up), surgical factors, radiographic factors; and LF area/spinal canal area in adjacent segments before the surgery before matching and all the above factors, except the angle between the facets in adjacent segments before the surgery after matching. Multiple regression analysis including the angle between the facets in adjacent segments and the LF area/spinal canal area in spondylolisthetic segments before the surgery demonstrated that the latter was the only factor that correlated with the increased LF hypertrophy during follow-up in adjacent segments after matching (partial regression coefficient, 0.607; 95% CI, 0.127–1.086; p=0.014).



(A) Before matching **(B)** After matching

Fig. 4. The receiver operating characteristic (ROC) curve for preoperative ligamentum flavum area/spinal canal area at the spondylolisthetic segments before **(A)** and after **(B)** matching.

ROC analysis

The AUCs were 0.833 (95% CI, 0.741–0.926; p<0.001) and 0.793 (95% CI, 0.662–0.924; p<0.001) before and after matching, respectively (Fig. 4). The cutoff values of the LF area/spinal canal area in spondylolisthetic segments were 0.4520 and 0.4295 before and after matching, respectively. Regarding the cutoff values, the

sensitivity, specificity, PPV, and NPV were 82%, 74%, 71%, and 84% before and 88%, 72%, 76%, and 86% after matching, respectively.

Discussion

In this study, compared with the asymptomatic group, the symptomatic adjacent canal stenosis group had a significantly larger LF area/spinal canal area in spondylolisthetic segments before the surgery and a greater degree of increased LF area/spinal canal area during follow-up in adjacent segments. In addition, the two values were significantly correlated in the total patient group and symptomatic adjacent canal stenosis group. Therefore, ligamentous stenosis in spondylolisthetic segments before fusion surgery might be strongly associated with symptomatic adjacent canal stenosis at follow-up.

Spinal fusion surgery applies excessive mechanical stress to the adjacent segments, which induces LF hypertrophy along with facet joint enlargement and disc protrusion, resulting in the compression of neural tissues. LF hypertrophy was expressed as increased LF area/spinal canal area during follow-up [13]. The symptomatic adjacent canal stenosis group had a significantly greater degree of the above value (months) in adjacent segments than in the asymptomatic group. Thus, LF hypertrophy was strongly correlated with symptomatic adjacent canal stenosis. The actual area of stenosis during follow-up due to LF hypertrophy and facet joint enlargement and disc protrusion was expressed as follows: (LF area at follow-up−LF area before the surgery) (mm^2)/time to MR imaging at follow-up and (spinal canal area before the surgery−spinal canal area at follow-up) (mm^2)/time to MR imaging at follow-up, respectively. In the symptomatic adjacent canal stenosis group, the average area, in months, of the former was more than twice that of the latter before (0.529 versus 0.254, p=0.021) and after (0.536 versus 0.222, p=0.016) matching. Therefore, LF hypertrophy could be the main cause of the symptomatic adjacent canal stenosis in this study.

Sairyo et al. [17,18] indicated that LF hypertrophy histologically comprised fibrosis and loss of elastic fiber. Fibrosis was also caused by the accumulation of mechanical stress and inflammation during aging. Some previous studies have demonstrated that a mechanical stretching force could promote transforming growth factor-β1 production by LF fibroblasts, which enhances the expression of types I and III collagen, resulting in LF hypertrophy [19-21]. In certain patients, radiographic and surgical factors could enhance adjacent canal stenosis by increased mechanical stress to the adjacent segments. However, Sakai et al. [13] showed that the cross-sectional area of the LF was significantly greater in patients with ligamentous stenosis not only in the lower lumbar spine but also in the upper spine than in those with nonligamentous stenosis. Thus, LF hypertrophy could be due not only to mechanical stress but also to other factors, such as genetic predisposition. Furthermore, the degree of ligamentous stenosis increased from L1/2 to L4/5, which meant that the ligamentous stenosis might have been also affected by mechanical stress.

In this study, no significant between-group differences were observed in patients or surgical or radiographic factors before and after matching. Therefore, LF hypertrophy in spondylolisthetic segments before the surgery and increased LF hypertrophy during follow-up in adjacent segments in the symptomatic adjacent canal stenosis group may not be due mainly to increased mechanical stress but also to an excessive response of LF cells to mechanical stress. In addition, the two aforementioned values were significantly correlated in the total patient and symptomatic adjacent canal stenosis groups. Accordingly, symptomatic adjacent canal stenosis correlated with a predisposition to LF thickening against mechanical stress. Furthermore, this predisposition affected the spondylolisthetic segments before and adjacent segments after the surgery.

In the asymptomatic group of this study, low correlations were found between LF hypertrophy in spondylolisthetic segments before the surgery and adjacent segments at follow-up. These correlations may be difficult to identify because of less responsiveness to LF thickening against mechanical stress.

A previous report indicated that PLIF was associated with a higher incidence of adjacent segment disease than did PLF because PLIF was thought to make a more rigid construct [8]. However, no significant difference was observed in the occurrence of symptomatic adjacent canal stenosis between PLF and PLIF in patients with and without follow-up MR images (total 137 patients), matched for age, sex, follow-up period, and LF area/spinal canal area in adjacent segments before the surgery.

Regarding the cutoff value, the sensitivity, specificity, PPV, and NPV were high before and after matching. Thus, if the LF area/spinal canal area in spondylolisthetic segments exceeds the cutoff value, adjacent canal stenosis is likely to occur after the surgery. Accordingly, decompression of adjacent segments must

be performed at the same time as fusion surgery.

This study has several limitations. First, the sample size was small. Patients with symptomatic adjacent canal stenosis following fusion surgery were not common and required long-term follow-up, whereas asymptomatic patients may have had difficulty obtaining MR images after long-term follow-up. Second, patients without follow-up MR images over 3 years in the asymptomatic group were not evaluated. Patients without MR images had more recent surgery (more PLIF cases) and shorter clinical follow-up periods than those with MR images over 3 years (69±28 months versus 103±44 months, $p<0.001$). Thus, the time may have been insufficient for adjacent canal stenosis to occur. However, no significant differences were found between patients with and without follow-up MRI images in the asymptomatic group regarding other patients and surgical factors, radiographic factors, and LF area/spinal canal area in spondylolisthetic and adjacent segments before the surgery. Moreover, the symptomatic adjacent canal stenosis group had a significantly larger LF area/spinal canal area than the asymptomatic group in spondylolisthetic segments before the surgery using the same propensity score-matching study (matched for follow-up period instead of time to MR imaging at follow-up) including patients without follow-up MRI images (total: 137 patients, $p=0.003$). Third, the effect of global malalignment on symptomatic adjacent canal stenosis other than LL could not be determined because whole-spine radiographs in the standing position were not obtained.

Conclusions

In this study, we found that symptomatic adjacent canal stenosis was mainly caused by LF hypertrophy. LF hypertrophy in spondylolisthetic segments before the surgery and adjacent segments during follow-up were significantly correlated. Therefore, a greater degree of LF hypertrophy in spondylolisthetic segments before fusion surgery could be a risk factor for symptomatic adjacent canal stenosis at follow-up. In addition, symptomatic adjacent canal stenosis might correlate with a predisposition to LF thickening against mechanical stress.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Funding

This work was supported in part by the Japanese Orthopaedic Association Grants-in-Aid for Scientific Research (E.N.). The sponsor had no role in the design or conduct of this study.

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Author Contributions

All authors contributed to the study conception and design. Material preparation and data collection were performed by Yosuke Oishi, Masaaki Murase, Katsumi Doi, Yoshinori Takeuchi, and Jun-ichi Hamawaki, study analysis was performed by Yosuke Oishi, Eiichiro Nakamura, and Akinori Sakai, and statistical analysis was performed by Yosuke Oishi. The first draft of the manuscript was written by Yosuke Oishi and all authors commented on previous versions of manuscript. All authors read and approved the final manuscript.

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