

Clinical Study

Endplate defects, not the severity of spinal stenosis, contribute to low back pain in patients with lumbar spinal stenosis

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Received 18 May 2021; revised 10 September 2021; accepted 21 September 2021

Abstract

BACKGROUND CONTEXT: It is controversial whether lumbar spinal stenosis (LSS) itself contributes to low back pain (LBP). Lower truncal skeletal muscle mass, spinopelvic malalignment, intervertebral disc degeneration, and endplate abnormalities are thought to be related to LBP. However, whether these factors cause LBP in patients with LSS is unclear.

PURPOSE: To identify factors associated with LBP in patients with LSS.

STUDY DESIGN/SETTING: Cross-sectional design.

PATIENT SAMPLE: A total of 260 patients (119 men and 141 women, average age 72.8 years) with neurogenic claudication caused by LSS, as confirmed by magnetic resonance imaging (MRI).

OUTCOME MEASURES: Ratings of LBP, buttock and leg pain, and numbness on a numerical rating scale (NRS), 36-Item Short Form Survey (SF-36) scores, muscle mass measured by bioelectrical impedance analysis, and radiographic measurements including slippage and lumbopelvic alignment. The severity of LSS, endplate defects, Modic endplate changes, intervertebral disc degeneration, and facet joint osteoarthritis were assessed on MRI.

METHODS: The presence of LBP was defined as an NRS score ≥ 3 . The demographic data, patient-reported outcomes, and radiological and MRI findings were compared between patients with and without LBP. Multivariate logistic regression analysis was used to identify the factors that were independently associated with the presence of LBP.

RESULTS: There were significant differences between patients with and without LBP for buttock and leg pain and numbness on the NRS, general health on the SF-36, presence of endplate defects, presence of Modic changes, disc degeneration grading, and disc height grading (all $p < .05$). Multivariate logistic regression analysis showed significant associations between LBP and diabetes (OR 2.43; 95% CI 1.07–5.53), buttock and leg numbness on the NRS (OR 1.34; 95% CI 1.17–1.52), general health on the SF-36 (OR 0.97; 95% CI 0.95–0.99), and the presence of erosive endplate defects (OR 3.04; 95% CI 1.51–6.11) (all $p < .05$).

FDA device/drug status: Not applicable.

Author disclosures: **MM:** Nothing to disclose. **MK:** Nothing to disclose. **MT:** Nothing to disclose. **SM:** Nothing to disclose. **TS:** Nothing to disclose. **MN:** Nothing to disclose. **YY:** Nothing to disclose. **TN:** Nothing to disclose. **WN:** Nothing to disclose. **YN:** Nothing to disclose.

This study was reviewed and approved by the Institutional Review Board at Wakayama Medical University.

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CONCLUSIONS: These results suggest that LBP in patients with LSS should be carefully assessed not only for spinal stenosis but also clinical factors and endplate defects. © 2021 Elsevier Inc. All rights reserved.

Keywords: Lumbar spinal stenosis; Spondylolisthesis; Low back pain; Modic changes; Endplate defects; Disc degeneration; Facet joint osteoarthritis; Muscle mass; Spinal alignment; Magnetic resonance imaging

Introduction

Degenerative lumbar spinal stenosis (LSS) describes a condition in which there is diminished space available for the neural and vascular elements in the lumbar spine secondary to degenerative changes in the spinal canal. When symptomatic, LSS causes variable clinical syndromes of gluteal and/or lower extremity pain and/or fatigue, which may occur with or without back pain [1]. In patients with LSS, the relationship between abnormal magnetic resonance imaging (MRI) findings and pain, especially low back pain (LBP), is debated, although several studies have shown improvements in LBP after decompression surgery without fusion [2–4]. Degenerative spondylolisthesis has been reported to be related to symptomatic LSS, although no association has been found between spondylolisthesis and the presence of LBP [5].

Common sources of LBP include the intervertebral disc, facet joint, sacroiliac joint, ligament, vertebral body, nerves, and paraspinal musculature [6,7]. Disc degeneration has long been suspected of playing an essential role in the pathogenesis of LBP. However, strong associations between disc degeneration and LBP have not been observed in population-based studies [8,9]. Recent attention has been focused on the endplate, which is more vascular and neural than the disc, as a potential source of LBP [10].

Modic endplate changes and endplate lesions have been found to be closely related to LBP [9,11]. Modic endplate changes appear as changes in the signal intensity of the endplate and vertebral bone marrow seen on MRI. Endplate lesions can appear on MRI as endplate defects of different shapes, and a previous MRI study classified endplate defects into focal, corner, and erosive defects [12]. The presence of endplate defects identified on MRI was associated with LBP after adjusting for the effects of Modic endplate changes and disc degeneration in a population-based sample [13]. However, no studies have examined the association between endplate defects and LBP in patients with LSS.

Trunk muscle mass has also been associated with LBP in patients attending spinal outpatient clinics [14]. Another study found that patients with LSS and sarcopenia reported more severe LBP compared with those without sarcopenia [15]. Furthermore, previous meta-analysis has demonstrated a strong relationship between LBP and decreased lumbar lordosis when compared with age-matched healthy controls [16]. To date, no studies have fully assessed these possible contributing factors for LBP in patients with LSS. The purpose of this study was to clarify which factors are associated

with LBP. We investigated clinical and radiological factors, including not only severity of spinal stenosis but also endplate abnormalities, disc degeneration, facet joint osteoarthritis, and lumbopelvic alignment in patients with LSS.

Materials and methods

Study design and population

This study was a cross-sectional study conducted at the Spine Care Center of Wakayama Medical University Kihoku Hospital from September 2017 to March 2020. The study was approved by the Institutional Review Board at Wakayama Medical University (No. 2378). All patients were required to provide written informed consent before enrollment.

The inclusion criteria were age >50 years, presence of neurogenic intermittent claudication and pain and/or numbness in the lower extremities with or without LBP, LSS confirmed by MRI, and referral to physical therapy. The exclusion criteria were previous spine surgery, foraminal stenosis, spondylolysis, osteoarthritis of the knee and/or hip, cognitive impairment, history of psychiatric illness, prostheses or metal implant, or implants or devices that are contraindications for body composition analysis such as the presence of an electronic implant (eg, heart pacemaker or brain stimulator). Consecutive patients who met the inclusion criteria and agreed to participate were enrolled in the study.

Measurements

A numerical rating scale (NRS) was used to assess the intensity of LBP, buttock and leg pain, and buttock and leg numbness. Patients completed a questionnaire and were asked, “Please show the degree of your pain (numbness) from 0 (no pain) to 10 (worst imaginable pain) when your symptom was at its worst during the past week?” Health-related quality of life was evaluated using the Medical Outcomes Study 36-Item Short-Form General Health Survey (SF-36) [17]. Appendicular and trunk skeletal muscle mass were measured by bioelectrical impedance analysis (BIA) using an InBody S10 device (InBody Co. Ltd, Seoul, South Korea).

Radiographic measurements

Radiographic measurements and MRI findings were evaluated by orthopedic spine surgeons who were certified as specialists by the Japanese Orthopedic Association and

Japanese Society for Spine Surgery and Related Research Spine. These surgeons were unaware of the study aims and patient information. The intrarater and interrater reliabilities of the radiographic and MRI evaluations were evaluated in 30 randomly selected patients by two orthopedic surgeons (Table 1). Each vertebral body from L1 to L5 was identified from bottom up by orthopedic surgeons who evaluated radiographic measurements.

Lumbopelvic alignment was measured using standing lateral radiographs and included measures of lumbar lordosis, pelvic incidence, pelvic tilt, and sacral slope. The presence of slippage and percent of slip (% slip) were assessed using lumbar flexion–extension radiographs of the patient in the standing position.

MRI evaluation

Lumbar spine MRI was performed using a 1.5-Tesla scanner (Signa HDxt 1.5T, GE Healthcare, Chicago, IL, USA). The sagittal *T1-weighted (T1W)* images were acquired using a fast spin-echo sequence with a repetition time (TR) of 670 ms and echo time (TE) of 14.2 ms. The sagittal *T2-weighted (T2W)* images were acquired with a TR of 4300 ms and TE of 102 ms. The matrix size was 384 × 256, field of view 300 × 300 mm, slice thickness 4 mm, and intersection gap 1 mm. The axial images were acquired with a TR/TE of 775 ms/14 ms and 3950 ms/110 ms for T1W and T2W, respectively, a matrix size of 320 × 224, and field of view of 200 × 200 mm. The slice thickness was 4 mm, and intersection gap was 1 mm.

The severity of spinal stenosis was examined using Schizas's seven-grade classification based on the morphology of the dural sac as observed on axial T2W images based on the rootlet/cerebrospinal fluid ratio [18]. Grades A1–A4 were defined as no stenosis or minor stenosis, B as moderate stenosis, C as severe stenosis, and D as extreme stenosis.

Endplate defects were classified into focal, corner, or erosive defects, according to the Feng classification on sagittal T2W images [12]. Focal defects were defined as follows: local hollow or discontinuity on the endplate with nucleus protrusion into the subchondral bone; corner defect involving the anterior or posterior corner of the vertebral body with locally disrupted or absent vertebral trabeculae; and (E, F) erosive defect characterized by extensive alteration of the endplate depicted as an irregular, serrated, or worm-eaten appearance.

body with locally disrupted or absent vertebral trabeculae; and erosive defect characterized by extensive alteration of the endplate depicted as an irregular, serrated, or worm-eaten appearance (Figure). Ten endplates in the lumbar spine (L1–S1) were evaluated for the presence or absence of any type of defects.

Modic endplate changes were classified into three types: type 1 was defined as hypointense on T1W images and hyperintense on T2W images; type 2 as hyperintense on both T1W images and T2W images; and type 3 as hypointense on both T1W images and T2W images [19].

Disc degeneration was evaluated on T2W images using the 5-point Pfirrmann grading system, with grade I representing a normal or nondegenerated disc and grade V representing end-stage disc collapse [20].

Disc bulging and disc height narrowing were examined using a 0–3 rating scale, with 0 defined as normal and 1–3 representing progressive degrees of abnormality [21].

Facet joint osteoarthritis was assessed on axial T1W images using a 4-point grading system, with grade 1 representing a normal and grade 4 representing marked osteophytes [22].

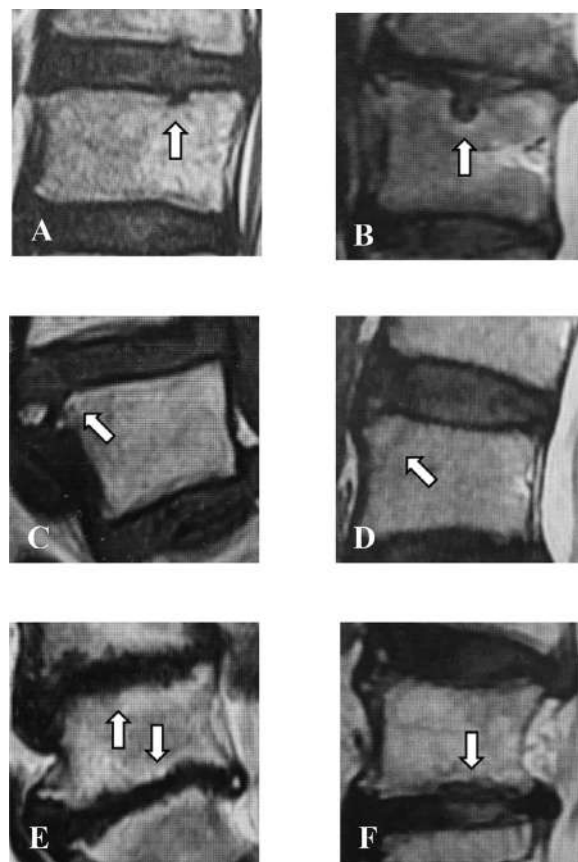


Fig. Morphological characteristics of three types of endplate defects. (A, B) Focal defects were defined as follows: local hollow or discontinuity on the endplate with nucleus protrusion into the subchondral bone; (C, D) corner defect involving the anterior or posterior corner of the vertebral body with locally disrupted or absent vertebral trabeculae; and (E, F) erosive defect characterized by extensive alteration of the endplate depicted as an irregular, serrated, or worm-eaten appearance.

Table 1
Reliability of radiographic and MRI measurements

Measurements	Intrarater reliability	Interrater reliability
Lumbopelvic alignment*	0.98	0.96
Presence of slippage†	0.80	0.74
Severity of stenosis†	0.77	0.68
Endplate defects†	0.78	0.65
Modic changes†	0.82	0.69
Disc degeneration†	0.77	0.71
Disc bulging†	0.71	0.57
Disc height narrowing†	0.80	0.70
Facet joint osteoarthritis†	0.76	0.65

* Intra- and interclass correlation coefficient,

† Kappa coefficient.

Statistical analysis

Each description of spinal stenosis (more than grade B) [23], Modic endplate changes, endplate defects, and disc degeneration (more than grade IV) [24], and facet joint osteoarthritis (more than grade 3) [25] was evaluated from L1/2 to L5/S. The scores for the grade of disc degeneration (1–5) [26], disc height, and disc bulging (0–3) [21] were summed from L1/2 to L5/S, because disc degeneration summary score has been reported to be associated with the presence of LBP [27]. The presence of LBP was defined as an NRS score ≥ 3 , as used in a previous study that defined a poor outcome for pain as an NRS score ≥ 3 in adults with recent-onset LBP [28].

The demographic data, patient-reported outcomes, and radiological and MRI findings were compared between patients with and without LBP using the Mann–Whitney *U* test for nonparametric variables, Student's *t* test for parametric variables and the χ^2 test. Post-hoc power analysis was performed to identify the power obtained for the Mann–Whitney *U* test and Student's *t* test. Multivariate logistic regression analysis using the forward stepwise likelihood ratio method was used to identify the factors that were independently associated with the presence of LBP (NRS ≥ 3). The following variables were included as independent variables: age, sex, body mass index, duration of symptoms, presence of comorbidities, trunk muscle mass, appendicular skeletal muscle mass, NRS, SF-36 score, and radiological and MRI findings. To test the quality of the logistic models, the Hosmer–Lemeshow goodness-of-fit test and Nagelkerke R-squared were used. Multicollinearity was measured by variance inflation factors (VIF) to prove that the independent variables do not correlate among each other. A *p* value $> .05$ indicated a good model fit in the Hosmer–Lemeshow goodness-of-fit test. All statistical tests were two-tailed, and the significance level was fixed at .05 throughout. Missing data were not imputed and assumed to be missing completely at random. All analyses were performed using IBM SPSS Statistics for Windows (version 27.0; IBM Corp., Armonk, NY, USA).

Results

A total of 260 patients (119 men and 141 women, average age 72.8 years) were enrolled. The demographic data and radiographic and MRI findings are summarized in (Tables 2–5), respectively. A total of 1,300 segments and 2,600 endplates from L1/2 to L5/S1 were assessed on MRI.

209 patients had LBP (NRS score ≥ 3), and 51 patients did not. 6 patients with LBP and 2 patients without LBP had missing data in the SF-36. There were significant differences between patients with and without LBP in the following factors: presence of hypertension and buttock and leg pain and numbness on the NRS, general health on the SF-36, presence of endplate defects, total number of endplate defects and erosive type of endplate defects, presence of Modic changes, total number of Modic endplate changes

and Modic type 3 endplate changes, disc degeneration grade, disc height grade, and summary score of disc height grade ($p < .05$) (Tables 2–5). The post-hoc power analysis for the Mann–Whitney *U* test and Student's *t* test showed that their powers were 0.88 and 0.89, respectively.

Stepwise multivariate logistic regression analysis showed significant associations between LBP (NRS score ≥ 3) and diabetes (odds ratio (OR) 2.43; 95% confidence interval (CI) 1.07–5.53; $p = .03$), buttock and leg numbness on the NRS (OR 1.34; 95% CI 1.17–1.52; $p < .01$), general health on the SF-36 (OR 0.97; 95% CI 0.95–0.99; $p = 0.01$), and the presence of the erosive type of endplate defects (OR 3.04; 95% CI 1.51–6.11; $p < 0.00$) (Table 6). VIF values among these independent variables were 1.011 to 1.033, indicating that there was no problem of multicollinearity. The model explained 25.0% (Nagelkerke R^2) of the variance in the presence of LBP and correctly classified 83.7% of cases.

Discussion

The factors associated with LBP, including demographic data, comorbidities, patient-reported outcomes, muscle mass, and radiographic measurements, were investigated in patients with LSS. This study showed that diabetes, buttock and leg numbness, general health, and presence of the erosive type of endplate defects, but not the severity of spinal stenosis, were associated with the presence of LBP.

In this study, spinal stenosis assessed according to the morphology of the dural sac was not associated with LBP. Other studies have analyzed the relationship between MRI findings and LBP in patients with LSS, but its use remains controversial [29,30]. However, no study to date has investigated the relationship between MRI findings, including endplate defects and Modic endplate changes, and LBP in patients with LSS. Although patients with LSS and LBP reported a higher degree of buttock and leg pain and numbness than those without LBP, diabetes, buttock and leg numbness, general health, and the presence of the erosive type of endplate defects, but not the severity of spinal stenosis assessed on MRI, were associated with LBP. These findings suggest that vertebral endplate defects are an independent factor related to LBP and neurogenic symptoms but not spinal stenosis itself contribute to LBP in patients with LSS.

The endplate serves as a physical shield that prevents the nucleus pulposus from penetrating the adjacent vertebral body while also acting as the gateway for nutrient transport between the vertebral marrow and the intervertebral disc. Damaged endplate regions can be sites of reactive bone marrow lesions that include proliferating nerves, which are susceptible to chemical sensitization and mechanical stimulation [31]. A previous study of a population-based sample reported that the erosive type of endplate defects was associated with a history of lifetime back pain and the intensity of the worst back pain [13]. Although endplate defects have been reported to be strongly associated with the presence of

Table 2
Characteristics of patients

	Total N = 260	LBP (+) (N = 209)	LBP (-) (N = 51)	p
Age (years)	72.8 ± 8.4	72.9 ± 8.3	72.4 ± 9.0	.63*
Sex (male:female)	119:141	93:116	26:25	.41 [†]
Height (cm)	157.8 ± 9.0	157.4 ± 8.9	159.5 ± 9.2	.17*
Weight (kg)	58.7 ± 11.0	58.3 ± 11.2	60.6 ± 10.2	.09*
Body mass index (kg/m ²)	23.5 ± 3.6	23.5 ± 3.8	23.8 ± 3.2	.41*
Appendicular skeletal muscle mass (kg)	16.7 ± 4.3	16.5 ± 4.2	17.4 ± 4.6	.16*
Trunk muscle mass (kg)	18.2 ± 4.0	18.1 ± 3.9	18.8 ± 4.2	.25*
Duration of symptoms (months)	42.0 ± 47.6	43.2 ± 48.0	36.9 ± 46.1	.08*
Comorbidities (number (%))				
Hypertension	164 (63.1)	138 (66.0)	26 (51.0)	<.05 [†]
Diabetes	80 (30.8)	67 (32.1)	13 (25.5)	.36 [†]
Dyslipidemia	66 (25.4)	52 (24.9)	14 (27.5)	.71 [†]
Heart disease	56 (21.5)	44 (21.1)	12 (23.5)	.70 [†]
Pulmonary disease	22 (8.5)	19 (9.1)	3 (5.9)	.46 [†]
Number of comorbidities	1.5 ± 1.1	1.5 ± 1.1	1.3 ± 1.0	.24*
NRS				
Back pain	5.0 ± 2.6	6.0 ± 1.9	1.1 ± 0.8	<.01*
Buttock and leg pain	5.8 ± 2.6	6.1 ± 2.5	4.6 ± 3.0	<.01*
Buttock and leg numbness	5.1 ± 2.9	5.5 ± 2.8	3.4 ± 2.8	<.01*
SF-36				
Physical functioning	53.7 ± 22.5	52.7 ± 22.4	57.9 ± 22.8	.19*
Bodily pain	38.2 ± 20.2	36.9 ± 19.4	43.4 ± 22.6	.06*
Role physical	49.4 ± 27.7	49.1 ± 27.2	50.8 ± 30.0	.70*
Role emotional	55.5 ± 28.9	54.4 ± 27.9	60.0 ± 32.7	.26*
Mental health	59.6 ± 21.6	59.2 ± 21.5	61.0 ± 22.2	.34*
Social functioning	62.8 ± 27.7	62.0 ± 27.7	66.1 ± 27.7	.38*
Vitality	48.7 ± 22.0	47.5 ± 22.1	53.8 ± 21.2	.08*
General health	49.9 ± 16.8	48.5 ± 16.7	55.5 ± 15.8	<.01*

Values are mean ± SD.

* Mann–Whitney U test.

[†] χ^2 test.

NRS, numerical rating scale; SF-36, Medical Outcomes Study 36-Item Short-Form General Health Survey.

Modic endplate changes and disc degeneration [12,32], the presence of endplate defects has also been associated with LBP after adjusting for the effects of Modic endplate changes and disc degeneration [13]. Another study of a population with LBP demonstrated that endplate defects are an independent risk factor for the progression of disc

degeneration and Modic endplate changes [33]. Our study showed that the presence of the erosive type of endplate defects was also associated with LBP in patients with LSS when considering the effects of demographic factors, patient-reported outcomes, and radiological findings such as Modic endplate changes and disc degeneration.

This study has some limitations. A cross-sectional design limits causal inference. Several studies have shown improvements in LBP after decompression surgery [3,4]. 1 possible explanation for this is that LBP and buttock pain might be not assessed separately. A previous study showed that buttock pain was significantly associated with neuropathic pain regardless of the presence of leg pain, and LBP without buttock pain was associated with nociceptive pain rather than neuropathic pain in patients with chronic lumbar spinal disorders [34]. Therefore, LBP and buttock pain have different pain properties. In this study, LBP and buttock pain, including leg pain, were assessed separately. Longitudinal studies are needed to identify whether preoperative endplate defects affect postoperative LBP after decompression surgery.

In this study, buttock and leg numbness, but not the severity of spinal stenosis assessed on MRI, was associated with LBP. It is possible that radiological assessment of

Table 3
Radiological findings

	Total N = 260	LBP (+) (N = 209)	LBP (-) (N = 51)	p
Spinal alignment				
LL (°)	20.6 ± 12.5	20.2 ± 12.6	22.1 ± 11.8	.33*
PI (°)	52.2 ± 11.0	52.3 ± 10.8	52.0 ± 11.6	.84 [†]
PT (°)	25.8 ± 9.2	26.2 ± 9.2	24.3 ± 8.9	.18 [†]
SS (°)	26.4 ± 9.1	26.1 ± 9.1	27.6 ± 9.0	.37 [†]
PI-LL (°)	31.6 ± 13.2	32.1 ± 13.3	29.8 ± 13.0	.29*
Presence of slippage N (%)	111 (42.7)	86 (41.1)	25 (49.0)	.31 [‡]
% Slip (%)	16.5 ± 6.2	16.3 ± 5.7	17.2 ± 7.5	.15 [†]

LL, lumbar lordosis; PI, pelvic incidence; PT, pelvic tilt; SS, sacral slope.

Values are mean ± SD.

* Student's t test.

[†] Mann–Whitney U test.

[‡] χ^2 test.

Table 4
MRI findings of spinal stenosis and endplate abnormalities

	Total N = 260	LBP (+) (N = 209)	LBP (–) (N = 51)	p
Spinal stenosis (1,300 segments analyzed)				
Stenosis grading (Schizas classification), N segments (%)				.40*
Grade A1	128 (9.8)	107 (10.2)	21 (8.2)	
Grade A2	313 (24.1)	259 (24.8)	54 (21.2)	
Grade A3	212 (16.3)	162 (15.5)	50 (19.6)	
Grade A4	188 (14.5)	154 (14.7)	34 (13.3)	
Grade B	251 (19.3)	194 (18.6)	57 (22.4)	
Grade C	207 (15.9)	168 (16.1)	39 (15.3)	
Grade D	1 (0.1)	1 (0.1)	0 (0)	
Total number of stenoses (more than grade B), N segments	1.8 ± 1.2	1.7 ± 1.2	1.9 ± 1.2	.43 [†]
Endplate defects (2,600 endplates analyzed)				<.01*
Focal defects, N (%)	445 (17.1)	369 (17.7)	76 (14.9)	
Corner defects, N (%)	260 (10.0)	210 (10.0)	50 (9.8)	
Erosive defects, N (%)	514 (19.8)	460 (22.0)	54 (10.6)	
Total number of focal defects, N endplates (from L1 to S1, per patient)	1.7 ± 1.6	1.8 ± 1.6	1.5 ± 1.7	.13 [†]
Total number of corner defects, N endplates (from L1 to S1, per patient)	1.0 ± 1.6	1.0 ± 1.5	1.0 ± 1.9	.32 [†]
Total number of erosive defects, N endplates (from L1 to S1, per patient)	2.0 ± 2.2	2.2 ± 2.4	1.1 ± 1.8	<.01 [†]
Total number of endplate defects, N endplates (from L1 to S1, per patient)	4.7 ± 2.7	5.0 ± 2.7	3.5 ± 2.7	<.01 [†]
Modic endplate changes (1,300 segments analyzed)				<.01*
Type 1, N (%)	49 (3.8)	45 (4.3)	4 (1.6)	
Type 2, N (%)	228 (17.5)	187 (17.9)	41 (16.1)	
Type 3, N (%)	65 (5.0)	62 (5.9)	3 (1.2)	
Total number of Type 1, N segments (from L1/2 to L5/S, per patient)	0.2 ± 0.5	0.2 ± 0.5	0.1 ± 0.3	.07 [†]
Total number of Type 2, N segments (from L1/2 to L5/S, per patient)	0.9 ± 1.0	0.9 ± 1.0	0.8 ± 0.9	.77 [†]
Total number of Type 3, N segments (from L1/2 to L5/S, per patient)	0.3 ± 0.5	0.3 ± 0.6	0.1 ± 0.2	<.01 [†]
Total number of Modic endplate changes, N segments (from L1/2 to L5/S, per patient)	1.3 ± 1.2	1.4 ± 1.2	0.9 ± 0.9	0.02 [†]

Values are mean ± SD.

* χ^2 test.

[†] Mann–Whitney *U* test.

spinal stenosis is insufficient because the results of the present study were obtained using conventional MRI with the patient in the supine position. Imaging studies of patients in this position is another limitation because symptoms are typically aggravated during walking or in the upright position in patients with LSS. The timing of MRI also might affect the relationship between radiological findings and pain, because LSS symptoms are often secondary to inflammatory flareups that occur affecting any of structures, such as intervertebral disc herniation, synovial cysts, and capsular and ligament hypertrophy. Although total number of spinal stenosis was included as independent variable in the multiple logistic regression analysis, presence of the erosive type of endplate defects, but not spinal stenosis was associated with presence of LBP in this study. However, a specific study of single-level LSS might better illustrate the role of endplate defects in LBP.

Finally, the Nagelkerke R-squared value in our stepwise logistic regression analysis was low. 4 variables were chosen as independent variables in the logistic regression analysis using the forward stepwise likelihood ratio method. This met the rule of ten events per variable, minimal criterion for sample size in logistic regression analysis, although evidence supporting events per variable rules for binary

logistic regression has been reported to be weak [35]. LBP appears to be a multifactorial problem associated with several biophysical factors, including inflammatory cytokines, altered pain-processing mechanisms and central sensitization, and psychological, social, and genetic factors [6,36].

Conclusions

Diabetes, buttock and leg numbness, general health, and the presence of the erosive type of endplate defects, but not spinal stenosis were associated with LBP in patients with LSS. These results suggest that LBP in patients with LSS should be carefully assessed not only for spinal stenosis but also clinical factors and endplate defects. Future studies should investigate whether the preoperative endplate defects are related to postoperative LBP after decompression surgery.

Declarations of competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Table 5
MRI findings of disc degeneration and facet joint osteoarthritis

	Total N = 260	LBP (+) (N = 209)	LBP (-) (N = 51)	p
Disc degeneration (1,300 segments analyzed)				
Disc degeneration grading (Pfirrmann classification), N segments (%)				<.01*
Grade I	6 (0.5)	4 (0.4)	2 (0.8)	
Grade II	122 (9.4)	98 (9.4)	24 (9.4)	
Grade III	285 (21.9)	224 (21.4)	61 (23.9)	
Grade IV	663 (51.0)	518 (49.6)	145 (56.9)	
Grade V	224 (17.2)	201 (19.2)	23 (9.0)	
Summary score of disc grading	18.8 ± 2.7	18.9 ± 2.8	18.2 ± 2.5	.11 [†]
Total number of disc degeneration (more than grade IV), N segments	3.4 ± 1.4	3.4 ± 1.4	3.3 ± 1.3	.40 [†]
Disc bulging (1,300 segments analyzed)				
Disc bulging grading, N segments (%)				.25*
Grade 0	256 (19.7)	208 (19.9)	48 (18.8)	
Grade 1	496 (38.2)	386 (36.9)	110 (43.1)	
Grade 2	423 (32.5)	345 (33.0)	78 (30.6)	
Grade 3	125 (9.6)	106 (10.1)	19 (7.5)	
Summary score of disc bulging grading	6.6 ± 2.5	6.7 ± 2.5	6.3 ± 2.4	.33 [†]
Disc height (1,300 segments analyzed)				
Disc height grading, N segments (%)				<.01*
Grade 0	323 (24.8)	253 (24.2)	70 (27.5)	
Grade 1	388 (29.8)	288 (27.6)	100 (39.2)	
Grade 2	315 (24.2)	262 (25.1)	53 (20.8)	
Grade 3	274 (21.1)	242 (23.2)	32 (12.5)	
Summary score of disc height grading	7.1 ± 3.7	7.4 ± 3.7	5.9 ± 3.1	.02 [†]
Facet joint osteoarthritis (1,300 segments analyzed)				
Facet joint grading (Fujiwara classification), N segments (%)				.64*
Grade 1	35 (2.7)	28 (2.7)	7 (2.7)	
Grade 2	473 (36.4)	375 (35.9)	98 (38.4)	
Grade 3	632 (48.6)	517 (49.5)	115 (45.1)	
Grade 4	160 (12.3)	125 (12.0)	35 (13.7)	
Total number of facet joint osteoarthritis (more than grade 3), N segments	3.0 ± 1.4	3.1 ± 1.4	2.9 ± 1.6	.68 [†]

Values are mean ± SD.

* χ^2 test.

[†] Mann–Whitney *U* test.

Table 6
Multivariate logistic regression analysis to identify factors associated with the presence of LBP (NRS ≥ 3)

Baseline factor	OR	95% CI for OR		p
		Lower	Upper	
Diabetes	2.43	1.07	5.53	.03
NRS buttock and leg numbness	1.34	1.17	1.52	<.01
SF-36 general health	0.97	0.95	0.99	.01
Presence of erosive defects	3.04	1.51	6.11	<.01

OR, odds ratio; CI, confidence interval; BMI, body mass index; NRS, numerical rating scale; SF-36, Medical Outcomes Study 36-Item Short-Form General Health Survey.

The independent variables entered into model were age, sex, BMI, presence of comorbidities, trunk muscle mass, appendicular skeletal muscle mass, NRS, SF-36 score, and radiological and MRI findings.

Continuous variables; Age, BMI, duration of symptoms, number of comorbidities, truncal and appendicular skeletal muscle mass, NRS, SF-36, spinal alignments, % slip, number of stenoses (more than grade B), number of endplate defects (focal, corner, erosive, total), number of Modic changes (type 1, 2, 3, total), number of disc degeneration (more than grade IV), summary score of disc grading (disc degeneration, bulging, height), and number of facet joint osteoarthritis (more than grade 3).

Categorical variables; Sex, presence of comorbidities, presence of slippage, presence of endplate defects (focal, corner, erosive), and presence of Modic changes (type 1, 2, 3).

Nagelkerke R-squared: 0.25.

Hosmer–Lemeshow goodness-of-fit test: 0.59.

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