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Original Article

Predictors of improvement in low back pain after lumbar decompression surgery: Prospective study of 140 patients

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ABSTRACT

Background: Lumbar decompression surgery is often used to treat neurological symptoms of the lower extremity as a result of lumbar disease. However, this method also leads to the improvement of the accompanying low back pain (LBP). We studied the extent of LBP improvement after lumbar decompression surgery without fusion and the associated preoperative factors.

Methods: Patients (n = 140) with lumbar spinal stenosis (n = 90) or lumbar disc herniation (n = 50) were included. To evaluate the change in LBP, VAS scores and the Oswestry disability index scores were measured before surgery and 2 weeks, 3 months, and 6 months after surgery. The predictors of residual LBP were investigated using logistic regression analyses.

Results: In total, 140 patients were examined. The VAS scores for LBP before surgery and 2 weeks, 3 months, and 6 months after surgery were 4.4 ± 3.0 (mean ± standard deviation), 1.1 ± 1.5, 1.3 ± 1.8, and 1.9 ± 2.2, respectively. LBP significantly improved 2 weeks after surgery ($P < 0.001$), stabilized between 2 weeks and 3 months after surgery, but was significantly aggravated 3–6 months after surgery ($P < 0.001$). At 6 months after surgery, 67 (47.9%) patients had a VAS score of >1. The predictors of residual LBP included severe preoperative LBP, degenerative scoliosis and the size of the Cobb angle. The independent predictors, determined by multivariate analysis were degenerative scoliosis and the size of the Cobb angle.

Conclusions: LBP was alleviated at 2 weeks after lumbar decompression surgery for lumbar disc herniation and lumbar spinal stenosis. The predictors of residual LBP after decompression included more severe LBP at baseline, degenerative scoliosis and the size of Cobb angle.

Level of evidence: Level 3.

1. Introduction

Having a prevalence of 28.5% in one study [1], low back pain (LBP) is a common cause of morbidity and disability. LBP has become a major health problem in Western countries, with a point prevalence of 10.2% [2], a 1-year prevalence ranging from 22% to 65%, and a life-time prevalence of up to 84% [3]. There are many causes of chronic LBP, including lumbar spinal stenosis (LSS) and lumbar disc herniation (LDH) [4–6]. The common spinal surgeries include discectomy for radiculopathy with LDH and decompressive laminectomy (both with and without fusion) for symptomatic spinal stenosis with or without degenerative spondylolisthesis [7]. Moreover, surgery for radiculopathy with LDH and symptomatic spinal stenosis is shown to have short-term benefits, as compared to treatment without surgery [8,9]. Thus, the aim of these surgeries is to treat the radicular leg pain and to improve walking ability.

Some studies have reported an improvement in the associated LBP following decompression surgery [10–12]. A recent study reported that decompression without fusion significantly improved LBP [13]. However, to our knowledge, no studies have investigated

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the improvement of LBP at the very early stage (2 weeks) after the surgery. Moreover, predictors of the failure of LBP improvement after lumbar decompression surgery without fusion have not been reported. In the present study, we aimed to investigate the improvement in LBP at 2 weeks after lumbar decompression surgery without fusion, to identify the preoperative predictors of LBP improvement after lumbar decompression surgery. Therefore, we performed a prospective clinical observational study involving 140 patients.

2. Materials and methods

2.1. Patients

We submitted a plan for a prospective study of the postoperative change in LBP, lower extremity pain, and lower extremity numbness to the ethical committee of our facility, and approval was obtained in September 2013 (approval no. 25 2013-9). Patients who were scheduled to undergo laminectomy and lumbar disc surgery without fusion for LSS or LDH were explained the purpose and method of the study, and were informed that they would receive the same level of care even if they did not participate in the study.

A total of 140 patients (98 men, 42 women; mean age, 60.4 ± 17.6 years [range, 18–85 years]) who provided consent to participate in the study were included. The exclusion criteria were as follows: >4 mm translation at maximum flexion and extension, unstable spondylolisthesis or spondylolytic spondylolisthesis with angulation of >10° at maximum flexion and extension, and degenerative scoliosis with a Cobb angle of >30°. Moreover, patients who were predicted to undergo spinal fusion preoperatively due to the presence of conditions such as foraminal stenosis, patients undergoing combined surgery of the cervical and lumbar spine, patients in whom the decompression area included the thoracic spine, and patients in whom LBP evaluation was difficult due to conditions such as dementia, were excluded from the study (Table 1). Preoperative standing plain radiographs were obtained to evaluate the presence of spondylolisthesis, Cobb angle, presence of lumbar compression fracture, and lordotic angle at Th12–S1. A Cobb angle of >10° indicated the presence of scoliosis.

2.2. Outcome measures

The follow-up of all 140 patients was prospectively performed, and clinical outcomes were evaluated by using a questionnaire. The severity of LBP was evaluated using Visual Analog Scale (VAS) scores (0–10) before surgery and 2 weeks, 3 months, and 6 months after surgery. Patients were asked to score their pain from 0 (no pain) to 10 (severe pain) to determine the VAS score. The patients were administered the VAS questionnaire (Japan Orthopaedic Association Back Pain Evaluation Questionnaire; JOBPEQ [14]) on the day before the surgery and at regular check-ups, and were asked to fill in the information in an isolated room to avoid the influence of medical staff. The results of the questionnaire were stored along with the clinical records. Moreover, subjective disability before surgery and 3 and 6 months after surgery was measured using the Oswestry disability index (ODI) [15], where 0% represents no disability and 100% represents extreme debilitating disability.

Table 1
Inclusion and exclusion criteria for the prospective cohort study.

Inclusion criteria
1. Lumbar spinal stenosis
2. Lumbar disc herniation
3. Candidate for surgical treatment
4. Magnetic resonance imaging/Myelogram confirmation of the pathology
5. Ability to fill in assessment form
Exclusion criteria
1. Unstable spondylolisthesis
2. Foraminal stenosis and lateral herniation
3. Simultaneous surgery for both cervical and lumbar spine
4. Simultaneous surgery for both thoracic and lumbar spine
5. Dementia
6. Absence of consent to participate in the study

2.3. Measurement of dural sac cross sectional area (DCSA) by magnetic resonance imaging (MRI)

DCSA was measured using T2 axial plane MRI [16]. The scans performed at the facet joint level for each intervertebral space were selected for measurement, as this site is most commonly associated with degenerative LSS [17]. In patients who underwent multilevel decompression, preoperative DCSA was measured at the smallest transverse area [18].

2.4. Surgical technique

The decompression procedures included microendoscopic discectomy [19], herniotomy (Love's procedure) [20], microendoscopic laminotomy [21], and conventional laminectomy. All the above-mentioned procedures were performed using standard techniques.

2.5. Postoperative treatment

A suction drain was placed after the surgery and was removed when the drainage fluid reduced to <100 mL/day. Pain medication was also administered for the treatment of preoperative pain. Walking was permitted on the day after the surgery, and a soft brace was applied for 3 months after the surgery. Patients were discharged 13.6 ± 7.9 days (range, 4–47 days) after surgery. The patients were allowed to return to work or begin physical exercise according to their specific condition.

2.6. Statistical analyses

Patients who could not be followed until 6 months after the surgery were excluded from the analysis. The change in the VAS score for LBP was compared between each time point using the paired *t*-test with the Bonferroni correction (5 comparisons: before surgery vs. 2 weeks after surgery, 2 weeks after surgery vs. 3 months after surgery, 3 months after surgery vs. 6 months after surgery, before surgery vs. 3 months after surgery, and before surgery vs. 6 months after surgery). The descriptive data are presented as mean ± SD. Moreover, patients with different diagnoses were compared using Welch's *t*-test.

We defined postoperative symptoms as follows: residual LBP, VAS >1; no improvement in LBP, VAS >1 and <3 improvement compared to preoperative values. Univariate logistic regression analyses were performed to identify the risk factors for residual LBP and no improvement in LBP 6 months after surgery. Multivariate analysis was also performed after adjusting for age, gender, and preoperative LBP. An AP value of <0.05 was considered significant. All analyses were performed using SPSS for Windows (version 21.0; SPSS, IBM, Chicago, IL, USA).

3. Results

A total of 140 patients (98 men and 42 women; mean age, 60.4 ± 17.6 years [range, 18–85 years]) were included in the final analysis after 2 patients were excluded due to death or the need for

additional surgery for another disease. Diagnosis was LDH in 50 patients (46.7 ± 19.3 years [range, 18–81 years]) and LSS in 90 patients (68.0 ± 10.8 years [range, 32–85 years]). The baseline characteristics of the patients, including their medical history, are presented in Table 2. Surgical techniques and complications of surgeries are presented in Table 3.

The percentages of subjects who used pain relief medication preoperatively and 6 months postoperatively were as follows: acetaminophen, 11.4% and 4.3%, respectively; non-steroidal anti-inflammatory drugs, 65.0% and 14.3%, respectively; tramadol hydrochloride, 8.6% and 3.6%, respectively; pregabalin, 20.0% and 14.3%, respectively. The use of pain relief medication was thus markedly reduced post operation.

3.1. Overall data

The mean VAS scores for LBP before surgery and 2 weeks, 3 months, and 6 months after surgery were 4.4 ± 3.0, 1.1 ± 1.5, 1.3 ± 1.8, and 1.9 ± 2.2, respectively. LBP significantly improved at 2 weeks after surgery ($P < 0.001$), stabilized between 2 weeks and 3 months after surgery, and significantly exacerbated between 3 and 6 months after surgery ($P < 0.001$) (Fig. 1). The ODI scores before surgery and 3 and 6 months after surgery were 44.9 ± 19.2, 14.7 ± 14.5, and 13.7 ± 12.8, respectively; a significant improvement was observed between the scores before surgery and those at 3 or 6 months after surgery ($P < 0.001$).

We noted that 117 (83.6%) patients had VAS scores for LBP of >1 before surgery, whereas 67 (47.9%) patients had VAS scores for LBP of >1 at 6 months after surgery, respectively. A total of 121 (86.4%) patients showed an improvement in the VAS scores for LBP at 6 months after surgery, whereas 19 (13.6%) patients showed deterioration in the VAS scores for LBP at 6 months after surgery. Fig. 3 shows the percentages of subjects with residual LBP and no improvement in LBP, 2 weeks, 3 months, and 6 months postoperatively.

Table 2
Characteristics of patients at baseline (N = 140).

Sociodemographic factors		
Age (years)		60.4 ± 17.6 (18–85)
Gender – no. (%)	Male	98 (70.0)
	Female	42 (30.0)
Diagnosis – no. (%)	Lumbar disc herniation	60 (42.9)
	Lumbar spinal stenosis	80 (57.1)
BMI (kg/m^2)		23.9 ± 3.7 (16.3–42.5)
Medical history – no. (%)		
Diabetes mellitus		22 (15.7)
Previous spinal surgery		15 (10.7)
Depression		4 (2.9)
Cerebral infarction		3 (2.1)
Cerebral hemorrhage		1 (0.7)
Arteriosclerosis obliterans		1 (0.7)
Symptoms		
Duration of symptoms (years)		1.7 ± 2.8 (0.1–20)
Sole numbness – no. (%)		77 (55.0)
Numbness of the perianal area – no. (%)		16 (11.4)
Radiography characteristics		
Lumbar lordosis (degree)		37.2 ± 13.9 (−3.5 to 68.9)
Scoliosis – no. (%) (>10°)		26 (18.6)
Cobb angle (degrees)		5.1 ± 6.8 (0 to 42.8)
Spondylolisthesis – no. (%)		18 (12.9)
Vertebral compression fracture (%)		12 (8.6)
Magnetic resonance imaging characteristics (80 patients with LSS were evaluated.)		
DCSA (mm^2)		64.9 ± 34.7 (4.0–133.0)
Severity of the clinical presentation		
Pre-operative visual analog scale score of low back pain		4.4 ± 3.0 (0–10)
Pre-operative visual analog scale score of leg pain		6.7 ± 2.5 (0–10)
Pre-operative Oswestry Disability Index		44.9 ± 19.2 (0–100)

Plus–minus values are presented as means ± standard deviation.
The mean duration of the symptoms is the time after the occurrence of low back pain, lower extremity pain, and lower extremity numbness until decompression surgery.
Abbreviations: BMI: body mass index; LSS: lumbar spinal stenosis; DCSA: dural sac cross-sectional area.

Table 3
Surgical technique and complications of surgery.

Surgical technique– no. (%)	
MED	50 (35.7)
MEL	15 (10.7)
Love procedure	10 (7.1)
Conventional laminectomy	65 (46.4)
No. of inter-vertebral levels of decompression	1.7 ± 1.0 (1–5)
Complications of surgery – no. (%)	
Dural tears	7 (5.0)
Epidural hematoma	3 (2.1)
Surgical site infection	1 (0.7)

Intervertebral levels of decompression are presented as mean ± standard deviation (range).
Abbreviations: MED: microendoscopic discectomy; MEL: microendoscopic laminotomy.

3.2. Predictors of postoperative residual LBP

The predictors of residual LBP were analyzed among 67 patients with a VAS score of >1 at 6 months after surgery by using logistic regression analysis. The results of the analysis are shown in Table 4. The risk factors of residual LBP included higher VAS score for preoperative LBP (+1; odds ratio [OR], 1.2; $P < 0.001$) and degenerative scoliosis (OR, 3.7; $P = 0.006$). Multivariate analysis indicated that degenerative scoliosis (OR, 4.6; $P = 0.005$) was a risk factor for residual LBP, independent of age, gender and VAS score for preoperative LBP. Moreover, a larger Cobb angle (>10°; OR, 3.2; $P = 0.002$) was associated with a higher risk of residual LBP. Patients with a larger decompression level (+1; OR, 1.4; $P = 0.05$) were more likely to have residual LBP; however, this relationship was not statistically significant.

3.3. Predictors of postoperative no improvement in LBP

The predictors of no improvement in LBP were analyzed using logistic regression analysis among 40 patients who showed no improvement in LBP 6 months after surgery. The results of the

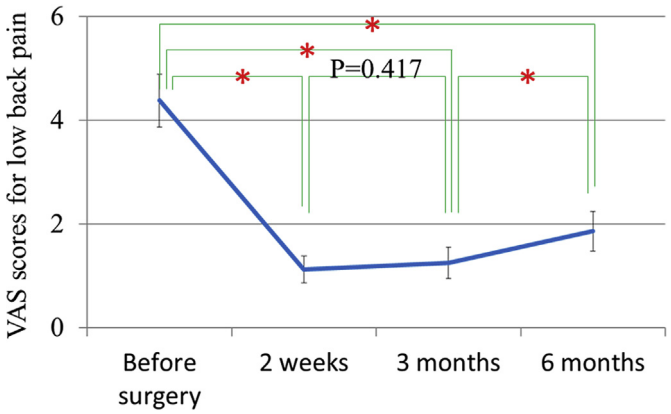

Fig. 1. Time course of the changes in low back pain before and after lumbar decompression surgery. Paired *t*-test with Bonferroni correction (a total of 5 comparisons), *$P < 0.01$. Error bars represent 95% confidence interval. VAS: Visual Analog Scale.

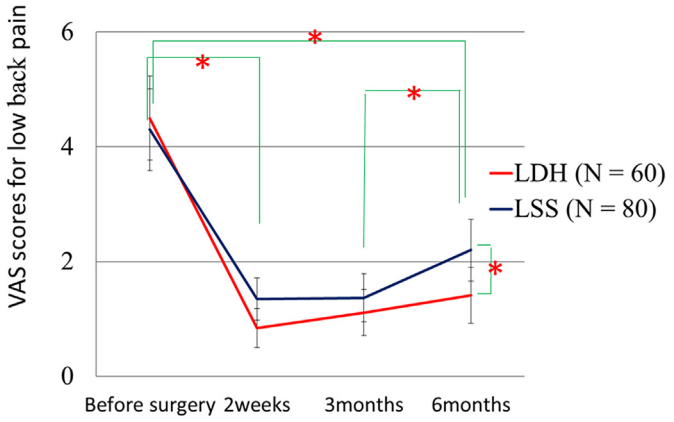

Fig. 2. Change of LBP after surgery according to diagnosis. Welch's *t*-test, *P < 0.05. Error bars represent 95% confidence interval. LDH: Lumbar disc herniation; LSS: Lumbar spine stenosis; VAS: Visual Analog Scale; LBP: Low back pain. Both groups showed a significant decrease in the VAS score for LBP 2 weeks after surgery (P < 0.001). LBP regressed significantly in the LSS group between 3 and 6 months (P < 0.001). LBP was significantly more severe in the LSS group 6 months after surgery (P = 0.032).

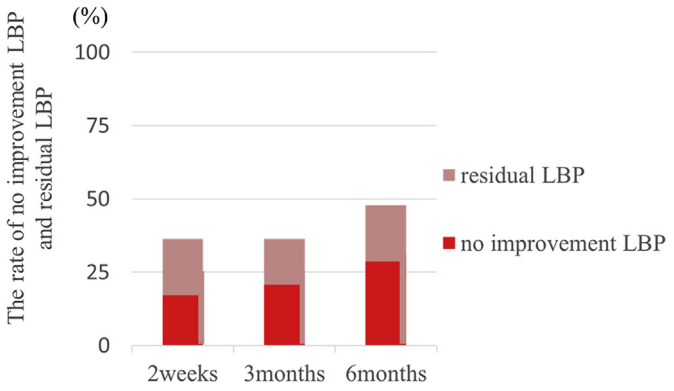

Fig. 3. Rates of no improvement in LBP and residual LBP. Residual LBP: LBP with VAS >1. No improvement in LBP: LBP with VAS >1 and <3 improvement compared to preoperative value. Two weeks, 3 months, and 6 months postoperatively, subjects showing residual LBP accounted for 36.4%, 36.3%, and 47.9%, respectively, and those who showed no improvement in LBP accounted for 17.1%, 20.7%, and 28.6%, respectively.

analysis are shown in Table 5. The risk factors of residual LBP included degenerative scoliosis (OR, 3.9; P = 0.002). Multivariate analysis indicated that degenerative scoliosis (OR, 4.2; P = 0.004) was a risk factor for residual LBP, independent of age, sex, and VAS score for preoperative LBP. Moreover, a higher Cobb angle (+10°; OR, 3.1; P = 0.001) was associated with a greater risk of residual LBP.

3.4. Comparison between LDH and LSS

The mean VAS scores for LBP before surgery and 2 weeks, 3 months, and 6 months after surgery in the LDH group (n = 50) were 4.5 ± 2.8, 0.8 ± 1.3, 1.1 ± 1.6, and 1.4 ± 1.9, respectively, and those in the LSS group (n = 90) were 4.3 ± 3.2, 1.3 ± 1.6, 1.4 ± 1.9, and 2.1 ± 2.4, respectively. In both the LDH and LSS groups, the LBP significantly decreased at 2 weeks after surgery (P < 0.001). The level of LBP 6 months after surgery tended to be higher in the LSS group. But there were not statistically significant (P < 0.081) (Fig. 2).

4. Discussion

This study found that: 1) LBP showed a significant improvement immediately after lumbar decompression surgery; and 2) Degenerative scoliosis and a larger amount of preoperative compression were significant predictors of residual LBP after lumbar decompression that were independent of age, gender, surgical technique, and preoperative LBP.

Jones et al. [13] showed a significant improvement in LBP in patients with LSS at 6 weeks after surgery (VAS score [range, 0–10] changed from 5.14 to 3.07; P < 0.0001) [13], and our study showed a significant improvement during the early stage after surgery (2 weeks after surgery) (VAS score [range 0–10] from 4.4 ± 0.3 to 1.1 ± 0.1; P < 0.0001). In our facility, the patient remains in the hospital for about 2 weeks after the surgery, until suture removal and discharge; hence, the LBP improvement can be attributed to the period of rest during the hospital stay. Moreover, although the LBP improved significantly at 2 weeks after surgery, a minimal but significant worsening was observed between 3 and 6 months after surgery.

In the present study, LBP associated with LDH also showed a significant improvement at 2 weeks after surgery. Compared to patients with LSS, those with LDH tended to show a greater improvement in LBP from before surgery to 6 months after surgery. Toyone et al. [12] reported the findings of 40 consecutive patients with disc herniation who were treated by discectomy, and suggested that nerve root compression due to LDH may be a possible

Table 4
Univariate and multivariate analyses of risk factors for residual low back pain at 6 month after surgery.

	Crude			Adjusted		
	OR	(95% CI)	P value	OR	(95% CI)	P value
Gender (female)	1.4	(0.7–3.1)	0.29			
Age (+10 yrs.)	1.1	(0.9–1.3)	0.48			
Low back pain at baseline (+1)	1.2*	(1.1–1.4)	<0.001			
Diagnosis (LSS compared to LDH)	2.0	(1.0–3.9)	0.05			
Surgical techniques (Endoscopic surgery)	0.5*	(0.3–1.0)	0.04	0.5	(0.2–1.4)	0.22
DM (+)	1.4	(0.6–3.4)	0.50	1.3	(0.5–3.7)	0.58
No. of levels of decompression	1.4*	(1.0–2.0)	<0.05	1.4	(0.9–2.1)	0.16
Duration of symptoms (+1 yrs.)	1.1	(0.9–1.2)	0.076	1.0	(0.9–1.2)	0.87
BMI (+10 kg/m^2)	1.4	(0.6–3.5)	0.45	2.0	(0.7–5.4)	0.18
Degenerative scoliosis (+)	3.7*	(1.5–9.6)	0.006	4.2*	(1.4–12.0)	0.008
Cobb angle (+10°)	2.8*	(1.5–5.4)	0.002	3.0*	(1.5–6.4)	0.003

Data were calculated by logistic regression analysis after adjustments for age, gender, and LBP at baseline and diagnosis. *P < 0.05.
Scoliosis was defined as a Cobb angle of >10°.
Abbreviations: LDH: lumbar disc herniation; BMI: body mass index; LSS: lumbar spinal stenosis; CI: confidence interval; OR: odds ratio; DM: diabetes mellitus.

Table 5
Univariate and multivariate analyses of risk factors for no improvement low back pain at 6 months after surgery.

	Crude			Adjusted		
	OR	(95% CI)	P value	OR	(95% CI)	P value
Gender (female)	1.0	(0.5–2.2)	0.99			
Age (+10 yrs.)	1.1	(0.9–1.4)	0.43			
Low back pain at baseline (+1)	0.9	(0.8–1.1)	0.35			
Diagnosis (LSS compared to LDH)	1.4	(0.6–2.9)	0.42			
Surgical techniques (Endoscopic surgery)	0.6	(0.3–1.3)	0.18	0.6	(0.2–1.6)	0.3
DM (+)	2.4	(1.0–6.2)	0.06	2.5	(0.9–6.7)	0.07
No. of levels of decompression	1.3	(0.9–1.8)	0.19	1.2	(0.8–1.9)	0.3
Duration of symptoms (+1 yrs.)	1.0	(0.9–1.2)	0.62	1.0	(0.9–1.2)	0.6
BMI (+10 kg/m^2)	2.4	(0.9–6.5)	0.08	2.4	(0.9–6.7)	0.09
Degenerative scoliosis (+)	3.9*	(1.6–9.6)	0.002	4.2*	(1.6–11.1)	0.004
Cobb angle (+10°)	2.8*	(1.5–5.2)	<0.001	3.1*	(1.6–6.1)	<0.001

Data were calculated by logistic regression analysis after adjustments for age, gender, and LBP at baseline and diagnosis. *P < 0.05.
Scoliosis was defined as a Cobb angle of >10°.
Abbreviations: LDH: lumbar disc herniation; BMI: body mass index; LSS: lumbar spinal stenosis; CI: confidence interval; OR: odds ratio; DM: diabetes mellitus.

cause of LBP [12]. Ohtori et al. [11] reported on 45 patients with LDH who were treated by discectomy; they indicated that the LBP in patients with disc herniation primarily originates from disc or nerve root compression, and that decompression surgery may decrease the pain in patients with a Modic type 1 signal change [11]. These reports support the presence of a relationship between LBP and nerve root compression. In the present study, LBP had improved at 2 weeks after herniotomy for LDH, and this early improvement can be explained simply by recognition of nerve root compression as a major cause of pain, because any improvement in pain caused by other conditions (e.g., trunk muscle power loss or facet joint or disc disorder) would have required more time.

The risk factors for residual LBP following lumbar decompression included a higher preoperative VAS score, scoliosis of 10–30°, LSS, and a higher level of preoperative compression. Moreover, a higher Cobb angle was associated with a higher risk of residual LBP. Jonsson and Stromqvist [22] reported that a greater degree of preoperative scoliosis was a predictive factor of more severe postoperative back pain; this result is consistent with that of the present study. Moreover, the present study indicated that a larger amount of preoperative compression is a risk factor of residual LBP, and a similar finding has been reported in other studies [23–25]. Ng et al. [26] evaluated lumbar decompression based on the functional outcome and reported that "the number of levels of decompression and the different types of decompression surgery did not influence the surgical results"; however, the results of the present study indicated that a larger amount of preoperative compression was associated with an increase in the likelihood of residual LBP. Our study demonstrated the existence of postoperative residual LBP, but evaluation of the surgery by the patients, including postoperative satisfaction, showed a relatively good improvement overall. This may be because the term residual LBP included milder symptoms that might not have affected patient evaluation.

Procedures without fusion such as herniotomy for LDH [12] and decompression for LSS [13] have been associated with a significant improvement in LBP; the current study similarly reported a significant improvement in LBP with both LDH and LSS, although LSS was found to be a predictor of residual LBP at 6 months after surgery. These results can be explained if LBP is partly attributable to symptoms of both cauda equina and nerve pain.

The causes of LBP due to LSS include a position involving greater bending of the lumbar spine [4], facet joint pain [5], and disk degeneration-related pain [6]. Hence, LSS should serve as a predictor of residual LBP, as it is associated with pain related to spinal position or facet joint pain, along with nerve compression pain. Kleinstuck et al. [27] stated that "greater back pain relative to preoperative LBP was associated with a significantly worse global outcome after decompression," and the evaluated outcome of that study was not limited to LBP; however, this finding indicates the relationship between more severe preoperative LBP and postoperative residual symptoms, and is hence consistent with the results of our study.

Ng et al. [26] reported that the ODI scores after decompression for LSS were inferior when the duration of the symptoms was more than 33 months; however, postoperative LBP was not evaluated in that study. In the present study, LBP also tended to show an inferior improvement when the duration of the symptoms was longer.

This study has a limitation. The main limitation of this study was that the postoperative follow-up period was only 6 months. We intentionally restricted this follow-up period in order to avoid the influence of LBP exacerbation as a result of factors unrelated to surgery. LBP is caused by complex reasons, and multiple factors may play a role in the long term. In addition, the validity of a comparison of LBP before and after surgery should be further discussed because the level of daily activity is reduced in majority of patients after surgery, and such change in activity level can, per se, affect pain perception. Hence, the LBP improvement observed in the present study may be transient, and long-term improvement has not been evaluated.

5. Conclusion

LBP was alleviated 2 weeks after surgery in patients with LSS and LDH. The predictors of residual LBP included higher VAS score for preoperative LBP and scoliosis. In particular, degenerative scoliosis was a risk factor of residual LBP that was independent of age, gender and VAS score for preoperative LBP, and a higher Cobb angle was associated with an increased risk.

Conflict of interest

The authors declare that they have no conflict of interest.

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