



# Impact of lumbar hypolordosis on the incidence of symptomatic postoperative spinal epidural hematoma after decompression surgery for lumbar spinal canal stenosis

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## Abstract

**Purpose** Potential associations between radiographic parameters and the incidence of symptomatic postoperative spinal epidural hematoma (PSEH) have not been identified. This study aimed to identify risk factors including radiographic parameters for symptomatic PSEH after posterior decompression surgery for lumbar spinal canal stenosis (LSS).

**Methods** We retrospectively reviewed 1007 consecutive patients who underwent lumbar decompression surgery for lumbar spinal canal stenosis (LSS) at our institution. We identified 35 cases of symptomatic PSEH, defined by clinical symptoms and MRI findings, and selected 3 or 4 age- and sex-matched control subjects for each PSEH subject (124 controls). We compared radiographic parameters and previously reported risk factors between PSEH and control subjects.

**Results** Compared to the control group, PSEH patients had significantly higher preoperative systolic ( $p = .020$ ) and diastolic ( $p = .048$ ) blood pressure, and more levels of decompression ( $p = .001$ ). PSEH and control subjects had significant differences in lumbar lordosis (PSEH  $24.8^\circ \pm 14.6^\circ$ , control  $34.8^\circ \pm 14.5^\circ$ ), pelvic tilt ( $25.1^\circ \pm 11.7^\circ$  vs.  $20.8^\circ \pm 8.4^\circ$ ), sacral slope ( $23.4^\circ \pm 9.4^\circ$  vs.  $27.6^\circ \pm 8.3^\circ$ ), and pelvic incidence minus lumbar lordosis ( $23.7^\circ \pm 15.0^\circ$  vs.  $13.7^\circ \pm 14.6^\circ$ ). Multivariate analysis revealed two significant risk factors for PSEH: decompression of two or more levels and lumbar lordosis  $< 25^\circ$ .

**Conclusions** Multilevel decompression and hypolordosis are significant risk factors for symptomatic PSEH after decompression surgery for LSS. LSS patients with lumbar hypolordosis or multilevel stenosis should be carefully observed for PSEH after decompression surgery.

**Graphical abstract** These slides can be retrieved under Electronic Supplementary Material.

**Key points**

1. Symptomatic postoperative spinal epidural hematoma
2. Decompression surgery
3. Lumbar spinal stenosis
4. Radiographic parameters
5. Lumbar hypolordosis

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Conditional logistic regression model				
	Cases/controls	Adjusted OR* (95% CI)	p value	
Hypertension	No	10/60	Reference	
	Yes	25/64	2.0 (0.8-4.9)	.12
Decompressed levels (n)	1	10/80	Reference	
	$\geq 2$	25/44	3.8 (1.5-9.6)	$< .01$
Lumbar lordosis	LL $> 40^\circ$	5/43	Reference	
	25-40°	14/55	1.7 (0.6-5.2)	.32
	LL $< 25^\circ$	16/26	3.4 (1.1-10.8)	.03

Abbreviations: CI, confidence interval; LL, lumbar lordosis; OR, odds ratio.  
\*OR is adjusted for hypertension, number of decompressed levels, and LL in addition to matching variables (age & sex).

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**Take Home Messages**

1. Lumbar lordosis  $< 25^\circ$  were significant risk factors for symptomatic postoperative spinal epidural hematoma (PSEH) after decompression surgery for lumbar spinal canal stenosis (LSS).
2. Multiple-level decompression was also associated with the incidence of symptomatic PSEH after lumbar posterior decompression surgery for LSS.
3. LSS patients with multilevel stenosis or lumbar hypolordosis should be carefully observed for the development of symptomatic PSEH after lumbar posterior decompression surgery.

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Extended author information available on the last page of the article

**Keywords** Symptomatic postoperative spinal epidural hematoma · Decompression surgery · Lumbar spinal stenosis · Radiographic parameters · Lumbar hypolordosis

### Abbreviations

LSS	Lumbar spinal canal stenosis
PSEH	Postoperative spinal epidural hematoma
MRI	Magnetic resonance imaging
BP	Blood pressure
NSAID	Nonsteroidal anti-inflammatory drug
JOA	Japanese Orthopaedic Association
VAS	Visual analog scale
BMI	Body mass index
FBSS	Failed back surgery syndrome
ASA	American Society of Anesthesiologists physical status
HTN	Hypertension
PT/INR	Platelet count and prothrombin time–international normalized ratio
SVA	Sagittal vertical axis
TK	Thoracic kyphosis
LL	Lumbar lordosis
SS	Sacral slope
PT	Pelvic tilt
PI	Pelvic incidence
OR	Odds ratios
CI	Confidence interval

### Introduction

Posterior decompression surgery for lumbar spinal canal stenosis (LSS) is a comparatively simple and well-established procedure with a relatively low risk of complications. However, serious complications requiring extended hospitalization or surgical intervention can arise. One such complication is symptomatic postoperative spinal epidural hematoma (PSEH), which causes intractable pain, sensory disturbance, motor paresis, and bladder and bowel dysfunction [1]. Magnetic resonance imaging (MRI) studies have shown that the incidence of asymptomatic PSEH is 15–89% [2–6], while symptomatic PSEH requiring surgical evacuation of the hematoma is much less frequent (.1–.5%) [7–13]. Clinical outcomes for PSEH are poor [2], and identifying the risk factors for symptomatic PSEH is critical for its prevention. Several risk factors have been reported, including advanced age, preoperative coagulopathy, alcohol consumption, multi-level surgery, blood type, high blood pressure (BP), previous spinal surgery, and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) [9, 10, 12–14]. To the best of our knowledge, there are no studies of potential associations between radiographic parameters and the incidence of symptomatic PSEH. Moreover, most studies have defined symptomatic

PSEH as cases involving evacuation surgery [7–13]. However, cases of symptomatic PSEH that involve intractable pain but no motor paralysis or sensory disturbance are sometimes treated conservatively. Therefore, in the present study, the diagnosis of symptomatic PSEH was based on clinical findings, including neurological deficit or intractable pain, and subsequent MRI scanning provided a definitive diagnosis. We aimed to identify factors associated with symptomatic PSEH, including radiographic parameters and previously reported risk factors, using datasets for patients with symptomatic PSEH.

### Materials and methods

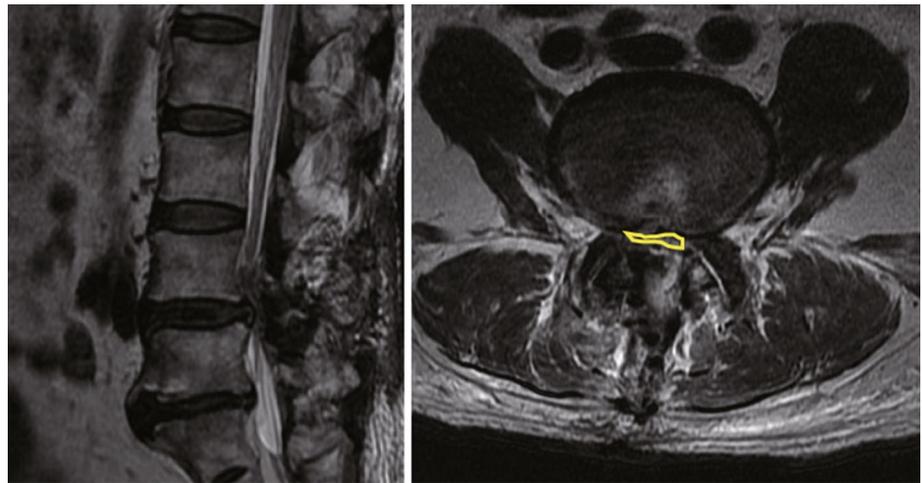
#### Subjects

We retrospectively reviewed 1007 consecutive patients who underwent posterior lumbar decompression surgery for LSS at our institution between September 2008 and December 2017. Clinical data were collected for each patient using patient charts and operative records according to our institution's ethics guidelines (approval number 20110222). All cases of lumbar decompression surgery used spinous process-splitting laminectomy without spinal instrumentation [15]. Preoperative anticoagulation therapy was discontinued in accordance with the manufacturer's instructions. Postoperative epidural suction drains were removed on the second postoperative day. The diagnostic criteria for symptomatic PSEH were as follows: 1) neurological deficit or intractable pain developing within 1 week after surgery, 2) confirmation of an epidural hematoma compressing the dura mater, either in re-operative findings or in postoperative MRI findings, showing a dural sac area < 75 mm<sup>2</sup> in an axial view of the compressed level (Fig. 1) [5].

#### Data collection

We collected data for possible risk factors for PSEH, including patient characteristics, coagulation status, preoperative radiographic parameters, Japanese Orthopaedic Association (JOA) score, visual analog scale (VAS), preoperative BP (at admission), postoperative BP (on returning to the hospital ward after surgery), and other perioperative factors. We reviewed the following patient characteristics: age, gender, body mass index (BMI), smoking status, failed back surgery syndrome (FBSS), American Society of Anesthesiologists physical status (ASA), and medical history, including type 2 diabetes

**Fig. 1** Postoperative MRI in a representative patient with symptomatic postoperative spinal epidural hematoma. Left panel, sagittal plane; right panel, axial plane. Yellow outline: measurement of dural sac area



mellitus, hypertension, cerebral infarction, angina pectoris, myocardial infarction, and cancer. ASA classes were condensed into three categories (I–III) in the analysis. Coagulation status was evaluated by laboratory analysis of the platelet count and prothrombin time–international normalized ratio (PT/INR) and by the oral administration of anticoagulant drugs and NSAIDs. Perioperative factors included operative time, operative blood loss, number of decompressed levels, intraoperative dural tear, and the volume of postoperative drainage output.

### Radiographic parameters

The following radiographic data were recorded from standing full-length spine radiographs: Cobb angle, anterior percentage slip, compression fracture, sagittal vertical axis (SVA), thoracic kyphosis (TK), lumbar lordosis (LL), sacral slope (SS), pelvic tilt (PT), and pelvic incidence (PI). We assessed spinopelvic alignment using PI–LL, SVA, TK, LL, PI, and PT, measured as previously described [16]. Lumbar scoliosis was diagnosed if the Cobb angle was  $\geq 10^\circ$ .

### Statistical analysis

Data are presented as mean  $\pm$  standard deviation. Differences between the PSEH and control groups were evaluated by Pearson's Chi-square test and Student's *t* test. We used a conditional logistic regression model to examine associations between symptomatic PSEH and individual risk factors, and estimated the odds ratios (ORs) and 95% confidence intervals (95% CI). Logistic regression was performed with STATA 14 software (Stata Corporation, College Station, TX). We considered  $p < .05$  to be statistically significant.

### Results

We assessed 54 cases of a neurological deficit or intractable pain developing within 1 week after surgery. Symptomatic PSEH was definitively diagnosed based on re-operative findings in one case and on MRI findings in 35 cases (Fig. 2). One case of symptomatic PSEH did not have preoperative whole-spine standing radiographs and was excluded. For each patient with symptomatic PSEH, three or four age- and sex-matched controls were selected from patients who did not have symptomatic PSEH. Our analyses in this study included 35 cases with symptomatic PSEH and 124 control cases (Fig. 2).

Baseline characteristics of the study subjects are given in Table 1. The mean age, gender ratio, mean BMI, smoking habit, prevalence of FBSS, and ASA status were similar between the PSEH and control groups (Table 1). However, the prevalence of hypertension (HTN) was significantly higher in the PSEH group than in the control group ( $p = .030$ ). As to the preoperative coagulation status, the mean platelet count and the proportion of patients with PT/INR  $> 1.15$  were comparable in the two groups (Table 2). Although the proportion of patients taking preoperative NSAIDs was higher in the PSEH group than in the control group, this difference was not statistically significant ( $p = .052$ ) (Table 2). The BP status of two groups is also given in Table 2. The systolic and diastolic preoperative BP were both significantly higher in the PSEH group than in the control group ( $p = .020$  and  $.048$ , respectively), whereas there were no significant differences in postoperative BP between the two groups (Table 2). Perioperative factors, summarized in Table 2, showed that the PSEH group had a higher number of decompressed levels than the control group ( $p = .001$ ) and that the total volume of drainage output on the second postoperative day was

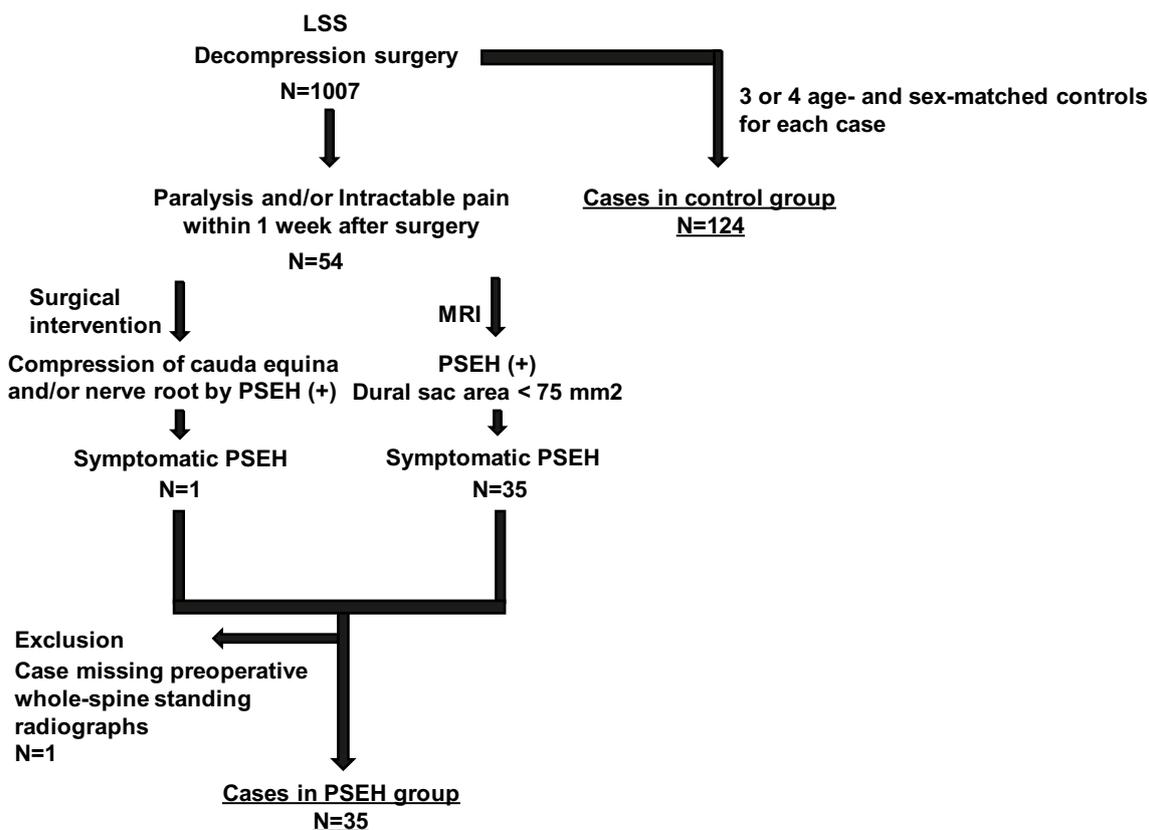


Fig. 2 Flowchart of study participant selection

significantly higher in the PSEH group than in the control group ( $p = .037$ ). As to clinical outcomes, the JOA score and VAS were similar in the two groups prior to surgery, but were significantly worse in the PSEH group than in the control group at discharge ( $p = .005$  and  $.022$ , respectively; Table 3). For the radiographic parameters, there were significant differences in LL (PSEH  $24.8^\circ \pm 14.6^\circ$  vs. control  $34.8^\circ \pm 14.5^\circ$ ;  $p < .001$ ), PT ( $25.1^\circ \pm 11.7^\circ$  vs.  $20.8^\circ \pm 8.4^\circ$ ;  $p = .017$ ), SS ( $23.4^\circ \pm 9.4^\circ$  vs.  $27.6^\circ \pm 8.3^\circ$ ;  $p = .012$ ), and PI–LL ( $23.7^\circ \pm 15.0^\circ$  vs.  $13.7^\circ \pm 14.6^\circ$ ;  $p < .001$ ) between the two groups (Table 4). Finally, to evaluate associations between symptomatic PSEH and the number of decompressed levels, HTN, or LL, we generated a conditional logistic regression model by adjusting each variable in addition to age and sex (Table 5). Multivariate analysis revealed that decompression of two or more levels (adjusted OR = 3.8, 95% CI = 1.5–9.6) was significantly associated with symptomatic PSEH (Table 5). In addition, the adjusted OR for symptomatic PSEH was 3.4 (95% CI = 1.5–9.6) in patients with LL  $< 25^\circ$  compared with those with LL  $> 40^\circ$ . In contrast, hypertension (adjusted OR = 2.0, 95% CI = .8–4.9) did not reach statistical significance.

## Discussion

This study revealed that multiple decompressed levels and lumbar hypolordosis contribute to the occurrence of symptomatic PSEH after lumbar posterior decompression surgery for LSS. Although several studies have reported that multiple-level decompression is associated with the incidence of symptomatic PSEH [9, 10, 12], we believe this is the first to report a significant correlation between the incidence of symptomatic PSEH and lumbar hypolordosis.

The incidence rate of symptomatic PSEH obtained in our study (3.0%) was higher than that reported in previous studies (.1–.5%) [7–13]. This is probably due to the difference in the criteria used to define symptomatic PSEH: Most studies define symptomatic PSEH as cases involving evacuation surgery [7–13], whereas in our study, the diagnosis of symptomatic PSEH was based on clinical symptoms and included cases that were treated conservatively as well as those treated by evacuation surgery. The PSEH was definitively diagnosed by MRI. We specified the presence of a dural sac area  $< 75 \text{ mm}^2$  in the axial view of the compressed level on MRI as a diagnostic imaging criterion; this is in line with Leonardi et al., who reported that the threshold for clinical significance in PSEH was a dural sac

**Table 1** Baseline characteristics

	PSEH (n = 35)	Control (n = 124)	p value <sup>a</sup>
Age	73.8 ± 8.8	73.0 ± 8.5	.608
Gender			
Male/female	24:11	82:42	.722
% Male	64.9%	66.1%	.787
BMI (kg/m <sup>2</sup> )	24.7 ± 3.101	25.6 ± 14.07	.696
History			
Diabetes mellitus	9 (25.7%)	34 (27.4%)	.841
Hypertension	25 (71.4%)	63 (50.8%)	<b>.030</b>
Cerebral infarction	1 (2.9%)	1 (.8%)	.336
Angina pectoris	3 (8.6%)	10 (8.1%)	.923
Myocardial infarction	1 (2.9%)	6 (4.8%)	.614
Cancer	5 (14.3%)	19 (15.3%)	.880
Smoking	11 (31.4%)	30 (24.2%)	.388
ASA			
1	2 (6.3%)	12 (9.9%)	.634
2	28 (87.5%)	97 (80.2%)	
3	2 (6.3%)	12 (9.9%)	
FBSS	3 (8.6%)	5 (4.0%)	.278

Bold indicates *p* value < 0.05, which is considered as statistical significance

ASA American Society of Anesthesiologists physical status, *BMI* body mass index, *FBSS* failed back surgery syndrome, *PSEH* postoperative spinal epidural hematoma

<sup>a</sup>By *t* test or Chi-square test

area of < 75 mm<sup>2</sup> in early postoperative MRI [5]. However, Ikuta et al. reported that among 30 patients who underwent endoscopic decompression, 33% had a cross-sectional area of < 75 mm<sup>2</sup> for epidural hematoma and did not exhibit severe clinical symptoms [2]. These observations indicate that the definition of symptomatic PSEH, including imaging findings, remains controversial and that establishing a universal definition will be important for future studies aimed at analyzing the risk factors for this disease.

Multilevel decompression can increase postoperative bleeding and is therefore considered a risk factor for symptomatic PSEH. Our study supports this hypothesis, since the PSEH group had a significantly higher volume of postoperative drainage output compared to the control group. In contrast, Kao et al. found no correlation between multiple decompressed levels and the incidence of PSEH [17]. However, Kao et al. included not only patients undergoing decompression surgery but also those undergoing instrumented fusion surgery, in which case the number of decompressed levels might not have directly influenced postoperative bleeding. Our study included only patients undergoing decompression, and surgical procedure was limited to spinous process-splitting laminectomy; thus, the influence of the number of decompressed levels on the incidence of PSEH could be evaluated with less bias. Our results indicate that LSS patients with multilevel stenosis should be monitored for symptomatic PSEH after decompression surgery.

**Table 2** Comparison of each variable between the PSEH and control group

	PSEH (n = 35)	Control (n = 124)	p value <sup>a</sup>	
Coagulation status				
Preoperative anticoagulants	5 (14.3%)	24 (19.4%)	.493	
Preoperative NSAIDs	5 (14.3%)	6 (4.8%)	.052	
PT-INR > 1.15	0 (0%)	9 (7.3%)	.101	
Plat (× 10 <sup>3</sup> )	209.51 ± 61.25	215.5 ± 50.87	.558	
Blood pressure (mmHg)				
Pre-operation	SBP	137.3 ± 17.2	129.5 ± 17.3	<b>.020</b>
(at admission)	DBP	78.2 ± 14.4	73.6 ± 11.3	<b>.048</b>
Post-operation	SBP	140.7 ± 21.3	137.6 ± 20.6	.440
(on returning to the hospital ward after surgery)	DBP	75.7 ± 12.0	72.4 ± 12.1	.160
Perioperative factors				
Number of decompression levels	2.0 ± .7	1.5 ± .8	<b>.001</b>	
Operating times (min)	69.2 ± 30.3	69.6 ± 31.7	.948	
Blood loss (ml)	43.2 ± 97.2	39.6 ± 87.3	.835	
Drainage output (ml)	326.1 ± 430.6	228.0 ± 153.7	<b>.037</b>	
Dural tear	2 (5.7%)	5 (4.0%)	.668	

Bold indicates *p* value < 0.05, which is considered as statistical significance

*PSEH* postoperative spinal epidural hematoma, *PT/INR* prothrombin time–international normalized ratio, *DBP* diastolic blood pressure, *SBP* systolic blood pressure

<sup>a</sup>By Chi-square test or *t* test

**Table 3** Preoperative and postoperative clinical outcomes

	PSEH (n = 35)	Control (n = 124)	p value <sup>a</sup>
JOA score			
Admission	14.6 ± 4.6	15.6 ± 4.6	.310
Discharge	20.6 ± 5.7	23.4 ± 4.4	<b>.005</b>
VAS			
Admission	61.4 ± 25.4	59.0 ± 25.6	.620
Discharge	25.8 ± 18.4	16.9 ± 18.1	<b>.022</b>

Bold indicates p value < 0.05, which is considered as statistical significance

JOA Japanese Orthopaedic Association, PSEH postoperative spinal epidural hematoma, VAS visual analog scale

<sup>a</sup>By t test

**Table 4** Preoperative radiographic parameters

	PSEH (n = 35)	Control (n = 124)	p value <sup>b</sup>
Anterior slip (> 10%)	11 (31.4%)	41 (33.1%)	.855
Compression fracture	10 (28.6%)	22 (17.7%)	.134
Lumbar scoliosis <sup>a</sup>	8 (22.9%)	36 (29.0%)	.471
SVA	60.1 ± 49.2	51.4 ± 44.7	.317
TK (°)	28.0 ± 11.1	30.4 ± 11.9	.280
LL (°)	24.8 ± 14.6	34.8 ± 14.5	<b>&lt; .001</b>
PT (°)	25.1 ± 11.7	20.8 ± 8.4	<b>.017</b>
SS (°)	23.4 ± 9.4	27.6 ± 8.3	<b>.012</b>
PI (°)	48.6 ± 9.7	48.5 ± 10.0	.961
PI–LL (°)	23.7 ± 15.0	13.7 ± 14.6	<b>&lt; .001</b>

Bold indicates p value < 0.05, which is considered as statistical significance

LL lumbar lordosis, PI pelvic incidence, PSEH postoperative spinal epidural hematoma, PT pelvic tilt, SS sacral slope, SVA sagittal vertical axis, TK thoracic kyphosis

<sup>a</sup>Lumbar scoliosis was defined as a Cobb angle > 10°

<sup>b</sup>By Chi-square test or t test

In this study, we proposed that LL less than 25° was another possible cause of symptomatic PSEH. Aono et al. reported that postoperative PSEH occurs most frequently in the thoracic region after decompression surgery [8], possibly because of the narrower space between the paravertebral muscles and the spinal cord due to kyphotic alignment compared with the cervical and lumbar spine [8]. Similarly, it can be hypothesized that lumbar hypolordosis leads to a narrowing of the dead space wherein the postoperative hematoma occurs, which can raise the internal pressure of the hematoma and lead to the development of symptomatic PSEH. However, because lumbar hypolordosis is not a factor that can be controlled before or during

**Table 5** Conditional logistic regression model

	Cases/ controls (n)	Adjusted OR <sup>a</sup> (95% CI)	p value
Hypertension			
No	10/60	Reference	
Yes	25/64	2.0 (.8–4.9)	.12
Decompressed levels (n)			
1	10/80	Reference	
≥ 2	25/44	3.8 (1.5–9.6)	<b>&lt; .01</b>
Lumbar lordosis			
LL > 40°	5/43	Reference	
25–40°	14/55	1.7 (.6–5.2)	.32
LL < 25°	16/26	3.4 (1.1–10.8)	<b>.03</b>

Bold indicates p value < 0.05, which is considered as statistical significance

CI confidence interval, LL lumbar lordosis, OR odds ratio

<sup>a</sup>OR is adjusted for hypertension, number of decompressed levels, and LL in addition to matching variables (age and sex)

decompression surgery, patients with lumbar hypolordosis should be informed of the higher risk of symptomatic PSEH when obtaining their consent for surgery.

The present study showed that both the systolic and diastolic BPs at the time of admission were significantly higher in the PSEH group than the control group. Fujiwara et al. also concluded that a high preoperative BP value was the most critical risk factor for PSEH after single-level decompression surgery [13]. These observations indicate that the preoperative BP may contribute to the incidence of symptomatic PSEH and that managing BP prior to hospitalization may be important for preventing symptomatic PSEH.

This study has several limitations. First, there were no clear criteria for intractable pain after surgery, which is one of the clinical diagnostic criteria for symptomatic PSEH. Therefore, obtaining a subsequent MRI depended on the subjective judgment of the attending physician. Second, the total number of patients reviewed was relatively small compared with previous studies [6, 8–10, 12, 13, 17]. However, because the surgical site was limited to the lumbar region and the surgical procedures were consistent, we were able to identify some statistically significant risk factors for symptomatic PSEH.

In conclusion, our multivariate analysis identified multiple levels of decompression and lumbar hypolordosis as risk factors for symptomatic PSEH following the decompression surgery for patients with LSS. Accordingly, LSS patients with multilevel stenosis or lumbar hypolordosis should be carefully observed for the development of symptomatic PSEH after spinal decompression.

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## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

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