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REVIEW

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How to prevent preoperative adjacent segment degeneration L5/S1 segment occuring postoperative adjacent segment disease? A retrospective study of risk factor analysis

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Abstract

Objective L5/S1 segment is one of the most common lumbar degenerative segments with high clinical failure rate. When the clinically responsible segment consists of one or more segments including L4/L5 segment, whether to merge the severely degraded L5/S1 segment together is a common problem plaguing clinicians. Therefore, the purpose of this study was to explore the risk factors for preoperative adjacent segment degeneration L5/S1 segment occuring Postoperative adjacent segment disease(ASDis), analyze the correlation between the high risk factors and the occurrence of adjacent segment disease, clarify the preventive measures and direction, and provide references for clinical selection of personalized treatment.

Methods The data of 119 patients with L5/S1 segment degeneration who underwent fixed to L4/5 posterior lumbar fusion surgery and were followed up in the orthopedic ward of Shandong Hospital of Traditional Chinese Medicine from January 2016 to January 2018 were retrospectively analyzed. According to the occurrence of ASDis at the last follow-up, all patients were divided into ASDis group (17 cases) and asymptomatic group (102 cases). The age, gender, BMI, bone mineral density and underlying diseases of the two groups were analyzed and compared. Perioperative time, intraoperative blood loss, incision length, number of surgical fusion segments, postoperative time on the ground, and hospital stay were recorded and compared. The improvement of VAS score and ODI index before and after operation were recorded and compared. X-ray and CT measurements were used to compare preoperative L5/S1 intervertebral space height, endplate Modic changes, gas in articular process, disc herniation calcification, sacral vertebrae lumbalization of patients, intraoperative L4/5 immediately corrected intervertebral space height, and sagittal position parameters of L5/S1 segment Segmental lordosis (SL), Pelvic incidence (PI), sacral slope (SS),lumbar lordosis (LL), pelvic tilt (PT), PI-LL and so on. Pfirmann grade, paravertebral muscle CSA, fat infiltration FI, paravertebral muscle rFCSA, psoas major CSA, and vertebral body area were measured and compared by MRI before surgery. The relative paravertebral cross-sectional area (rCSA), relative psoas major cross-sectional area (rCSA) and

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relative functional paravertebral cross-sectional area (rFCSA) were calculated. logistic regression analysis was used to determine the risk factors for preoperative adjacent segment degeneration L5/S1 segment occuring Postoperative ASDis, and the receiver operating characteristic (ROC) curve was described and the area under the curve was calculated.

Results All patients successfully completed the operation. Proportion of patients with osteoporosis combined with ASDis [yes/no, (9/8) vs. (21/81), $P=0.004$], BMI [(27.55 ± 3.99) vs. (25.18 ± 3.83), $P=0.021$], the number of fusion segments [(1.76 ± 0.75) vs. (1.28 ± 0.52), $P=0.020$], the correction height of L4/5 intervertebral space [(2.71 ± 1.21) mm vs. (2.10 ± 1.10) mm, $P=0.037$] were significantly higher than those in asymptomatic group. Bone mineral density T value in ASDis group [(-1.54 ± 1.68) g/cm^2 vs. (-0.01 ± 2.02) g/cm^2, $P=0.004$] was significantly lower than that in asymptomatic group. There were no significant differences in operation time, incision length, intraoperative blood loss and walking time between the two groups ($P>0.05$). Preoperative imaging: In ASDis group, paravertebral muscle CSA [(4478.37.3 ± 727.54) mm vs. (4989.47 ± 915.98) mm, $P=0.031$], paravertebral muscle rCSA [(3.14 ± 0.82) vs. (3.87 ± 0.89), $P=0.002$], paravertebral muscle rFCSA [(2.37 ± 0.68) vs. (2.96 ± 0.77), $P=0.003$] were significantly lower than those in non-sedimentation group. Endplate Modic changes (I/II/III/ no, (3/5/4/7) vs (23/16/5/56), $P=0.048$) and vertebral canal morphological classification (0/1/2 grade, (7/5/5) vs (69/25/8), $P=0.019$) in ASDis group were significantly different from those in asymptomatic group. The proportion of patients with gas in L5/S1 segment in ASDis group [yes/no, (6/11) vs. (13/89), $P=0.019$] was significantly higher than that in asymptomatic group. ASDis group of preoperative LL Angle [(34.10 + 13.83)° vs. (41.75 + 13.38) °, $P=0.032$) and SL Angle [(15.83 + 5.07) vs. (22.77 + 4.68) °, $P=0.022$],2 days after surgery LL Angle [(38.11 + 11.73) vs. (43.70 + 10.02) °, $P=0.038$) and SL Angle [(15.75 + 3.92) vs. (19.82 + 5.46) °, $P=0.004$), at the time of the last follow-up LL Angle [(37.19 + 11.99) vs. (43.70 + 11.34) °, $P=0.032$) and SL Angle [(13.50 + 3.27) vs. (16.00 + 4.78) °, $P=0.041$) were significantly less than the asymptomatic group. Postoperative imaging: There were no significant differences in the time of intervertebral bone fusion and the number of patients with failed internal fixation between the two groups ($P>0.05$). At the last follow-up, VAS score [(3.24 ± 1.39) vs. (1.63 ± 0.84), $P<0.001$] and ODI score [(21.00 ± 9.90) vs. (15.79 ± 4.44), $P=0.048$] in ASDis group were significantly higher than those in asymptomatic group. Bivariate logistic regression showed that BMI value (OR = 1.715, $P=0.001$) and number of surgically fused segments (OR = 4.245, $P=0.030$) were risk factors for preoperative adjacent segment degeneration L5/S1 segment occuring Postoperative ASDis. The degree of spinal stenosis grade 0 (OR = 0.028, $P=0.003$), the paraverteal muscle rFCSA (OR = 0.346, $P=0.036$), and the Angle of Postoperative L5/S1 segment SL (OR = 0.746, $P=0.007$) were protective factors for preoperative adjacent segment degeneration L5/S1 segment occuring Postoperative ASDis. Under ROC curve, the area of Postoperative L5/S1 segment SL Angle was 0.703, the area of paravertebral muscle rFCSA was 0.716, the area of BMI was 0.721, and the area of number of fusion segments was 0.518.

Conclusion Excessive number of surgical fusion segments, spinal canal stenosis greater than grade 0, excessive BMI, too small Postoperative L5/S1 segment SL Angle, and too small paravertal muscle rFCSA are risk factors for preoperative adjacent segment degeneration L5/S1 segment occuring Postoperative ASDis. Prevention should be focused on the above aspects to reduce the incidence of L5/S1 segment ASDis.

Keywords ASDis, L5/S1 segment, Logistic regression analysis

Introduction

Adjacent segment degeneration (ASD) is a common complication following spinal fusion surgery that is most common in the lumbar spine. It includes radioactive ASD (ASDeg) and adjacent segment disease (ASDis). ASDeg refers to imaging changes in the adjacent segment of the patient and is manifested as decreased disc signal, spinal canal stenosis, decreased vertebral space height, new osteophyte formation, or increase of existing osteophyte in MRI of adjacent segments. ASDis refers to a series of symptoms and signs that are caused by the degeneration of adjacent segments, including lower limb radiation pain, intermittent claudication and other neurological symptoms, or significant aggravation of lower back pain. In a study by Abraham et al. [1] on 217 patients, the incidence of ASDeg and ASDis was reported to be 29% and 18%,respectively. They reported the incidence of reoperation to be 9%. Xia et al. [2] reported the incidence of ASD following interbody fusion to be 4.8–92.9%. In-depth studies have been conducted on the degeneration of adjacent segments of the lower lumbar spine and lumbosacral vertebrae based on existing biomechanical and clinical studies. The degenerative changes of unfused adjacent segments can be accelerated and the motion amplitude of adjacent segments and intervertebral stress can be increased by spinal fusion [3]. Finite element model analysis confirmed the increase of intervertebral disc pressure to lead to intervertebral disc degeneration. Yan et al.

[4] discovered that the stress increase in the endplate of adjacent segments following lumbar fusion and fixation exceeded the physiological range, which also caused the degeneration of adjacent segments. Preoperative signs of adjacent segment degeneration are important factors that influence postoperative adjacent segment degeneration. This mainly includes the preoperative degeneration of adjacent intervertebral discs, lumbar stenosis, and the degeneration of adjacent facet joints [5]. Anandjiwala et al. [6] conducted a controlled study and found a significantly increased incidence of postoperative adjacent-level degeneration in patients with signs of intervertebral disc degeneration at adjacent levels before lumbar fusion and fixation. Similarly, lumbar spinal stenosis makes it more difficult for the lumbar spine with degeneration to withstand the increased adjacent level pressure due to fusion fixation, and patients undergoing surgical treatment due to spinal stenosis have a relatively high incidence of adjacent level degeneration [5]. Taking reasonable treatment measures is the key to preventing disease transmission for patients with adjacent degenerative segments before operation.

Extensive clinical studies on postoperative ASD have been conducted, and different preventive measures have been provided for the high risk factors. However, there are relatively few studies on the occurrence of ASDis following pre-operative degeneration. Clinicians often perform "one-size-fits-all" preventive surgery for segments with severe adjacent degeneration before surgery, but most do not require preventive surgery, thereby increasing surgical trauma and surgical cost. However, existing degeneration is not treated in some patients and performing a second operation for postoperative ASDis is often difficult and the operation effect is poor. Therefore, the high-risk factors for postoperative ASDis should be identified for patients with preoperative adjacent degeneration and intervention should be conducted in advance in order to reduce the incidence of ASDis. At the same time, surgical treatment can be performed in advance to avoid the trauma of a second operation in patients with more high-risk factors. L5/S1 is one of the most common degenerative segments in the clinic and it is also the last mobile unit of the spine, which connects the spine and important joints of the pelvis. L5/S1 fusion affects the squatting, sitting, and other postures of patients, and can reduce their quality of life. However, as there are more patients with L5/S1 degeneration before surgery and the degree of degeneration is severe, preventive surgery is performed by many clinicians to prevent the occurrence of ASDis, affecting patient quality of life. Therefore, identifying the risk factors for L5/S1 adjacent segment degeneration before operation is of great guiding significance for clinical treatment, prevention, and surgical plan design. In addition, analyzing the correlation of risk factors and identifying independent risk factors are also important.

Information and methods

General information

Data of 102 patients who underwent fusion fixation to L4/5 posterior lumbar surgery and preoperative L5/S1 level degeneration (the surgery was performed by experienced and senior physicians) who were admitted to the orthopedic ward of Shandong Hospital of Traditional Chinese Medicine from January 2016 to January 2019 and were followed up. A total of 119 patients were included and divided into ASDis group (17 cases) and asymptomatic group (102 cases) according to whether ASDis occurred at the last follow-up.

Diagnostic Criteria: The X-ray criteria for evaluating the degeneration of adjacent segments were: (1) anterior-posterior glide of adjacent segment in extension and flexion position was more than 3 mm, or the movement Angle of the vertebral body was more than 10°; (2) endplate sclerosis, degenerative scoliosis; (3) Vertebral space height loss of more than 10%; (4) The formation of new osteophytes or the increase of original osteophytes by more than 3 mm. At the last follow-up, ASD was diagnosed as long as there were adjacent level spinal stenosis, increased disc degeneration, or X-ray changes meeting the criteria for adjacent level degeneration on CT or MRI. At the same time, if the lower extremity nerve symptoms and lower back pain become significantly worse again, combined with the degeneration on the image, ASDis will be diagnosed [7].

Inclusion criteria: ① All surgical segments were fused to L4/5 segments, with the number of fused segments ≤ 4; (2) L5/S1 had different degrees of degeneration (e.g., MRI showed disc Pfirmann grade ≥ 3 or spinal canal stenosis grade ≥ 1, CT showed air accumulation in the disc and decreased spinal canal sagittal diameter, X-ray showed decreased vertebral space height and osteophytic hyperplasia); ③ PLIF surgery was performed; The clinical data and follow-up data were complete and the follow-up time was at least 3 years. ⑤ The patient agrees and signs the informed consent for surgery.

Exclusion criteria: ① not fused to L4/5 segments; (2) L5/S1 has unstable factors (slip, rotation, scoliosis, etc.); ③ Postoperative infection, broken nails and rods; ④ Patients with insufficient clinical or imaging data; (5) Patients with tuberculosis, tumor, compulsory spondylitis and other special diseases; Follow-up data are incomplete.

Surgical methods

After successful general anesthesia, the patient was placed in a prone position and the patient's abdomen was suspended. C-arm fluoroscopy was used to locate

the pedicle body surface projection of the vertebral body during the operation, and the median incision was made at the back of the waist. The electroknife was removed layer by layer to the lamina, and the facet joints and transverse process were expanded to both sides. The insertion point of the pedicle screw was located, the positioning needle was placed, the accurate position was confirmed by C-arm fluoroscopy, and the direction of the positioning needle was twisted into the pedicle nail, and spinal canal decompression was performed. Directly, the nucleus pulposus forceps removed the prolapsed nucleus pulposus tissue. In addition, the lateral recess and nerve root canal were enlarged, and the nerve root lysis was complete. After the nucleus pulposum tissue was removed, the cartilaginous endplate was alternately scraped with a tooth scraper, and the interdisk tissue was completely removed. The soft tissue of the bitten lamina was removed and then trimmed into granular bone pieces and implanted into the vertebral space. An interbody fusion device of appropriate size was inserted into the space, and the bone fragments were filled into the fusion device. Place the pre-bent titanium rod at the nail tail and tighten the nut to cut off the tail. Check instruments, built-in drainage tube, layer by layer suture.

Observation indicators

(1) The Visual Analogue Scale (VAS) is currently the main method for clinical pain scoring. This method uses a 10 cm scale to divide pain into 0–10 values, and patients can select the corresponding values on the scale according to their own feelings. The pain degree is evaluated according to different values, and the higher the value, the more severe the pain.

(2) The Oswestry Disability Index (ODI) scoring system was used to score 10 items including living ability, social activities, pain intensity, walking, and extracts before and after treatment, with each item scoring 0–5 points. The higher the score, the more obvious the functional impairment.

(3) Individual factors of the patient, including age, gender, bone mineral density, preoperative BMI, postoperative follow-up time, etc. Surgical data, including operation time, amount of blood loss, perioperative complications (including cerebrospinal fluid leakage, wound infection, postoperative neurological dysfunction, perioperative second operation, etc.), time spent on the ground, length of hospital stay, etc.

(4) Imaging data X-ray measurements of preoperative and postoperative sagittal position parameters of patients. Including the pelvic incidence, PI), pelvic tilt (PT), sacral slope (SS), lumba lordosis (LL), Segmental lordosis SL, and pelving-lumbar matching degree (pelvic Opponent-lumbar lordosis, PI-LL, and so on, measure the postoperative L5/S1 intervertebral space height and intraoperative L4/5 intervertebral space correction height (Fig. 1). CT was used to record the degree of articular process degeneration (pneumatosis, hyperplasia, etc.) and the calcification of L5/S1 intervertebral disc before operation, as well as the time of postoperative fusion. The Pfirmann grade of L5-S1 intervertebral disc and the degree of lumbar spinal canal stenosis were recorded by MRI before surgery (grade 0: no significant spinal canal stenosis, obvious cerebrospinal fluid filling in front of cauda equina nerve; Grade 1: Mild spinal canal stenosis, no obvious cerebrospinal fluid filling in front of cauda equina, cauda equina terminalis aggregation, but mutually variable; Grade 2: moderate stenosis, cauda equina clustered into bundles; Grade 3: Severe stenosis, almost complete occlusion of the dural sac space). MRI was used to measure the horizontal psoas major area (CSA), vertebral body CSA, paravertebral muscle CSA, functional paravertebral muscle cross-sectional area (FCSA) and paravertebral fat area before surgery using Imge J software (National Institutes of Health, USA). Set the gray threshold as 120, calculate the percentage of pixels representing fat infiltration (FI) in the paravertebral muscle CSA [8], and calculate the relative cross-sectional area of the psoas major. rCSA), Paravertebral muscle rCSA, paravertebral muscle rFCSA and FI; Relative CSA (rCSA) was calculated, that is, the ratio of muscle CSA to disc CSA at the same level (changed to vertebral CSA due to disc degeneration and deformation), which was used to control the influence of body type, weight and height on muscle CSA [9] (Fig. 2).

Statistical analysis

SPSS20.0 software was used for statistical analysis. Measurement data were represented by ($x \pm s$). When data were normally distributed, independent sample t test was used for comparison between the two groups. When the data is not normally distributed, the rank sum test is used. Counting data were tested by 2 test or Fisher exact test. Mann-whitney U test was used to compare the grade data between the two groups. $P < 0.05$ was considered statistically significant. Regression analysis was performed on statistically significant indicators, and the dependent variables were divided into two categories. Therefore, binary multi-factor logistic regression analysis was selected, and the indicators selected by univariate analysis were included into the independent variables of the logistic regression model. The risk factors for postoperative ASDis after preoperative degeneration L5-S1 were determined through analysis. Describe the receiver operating characteristic (ROC curve) of risk factors, calculate the area under the curve and the critical point. intraclass correlation (ICC) was used to assess the agreement between the two observers.

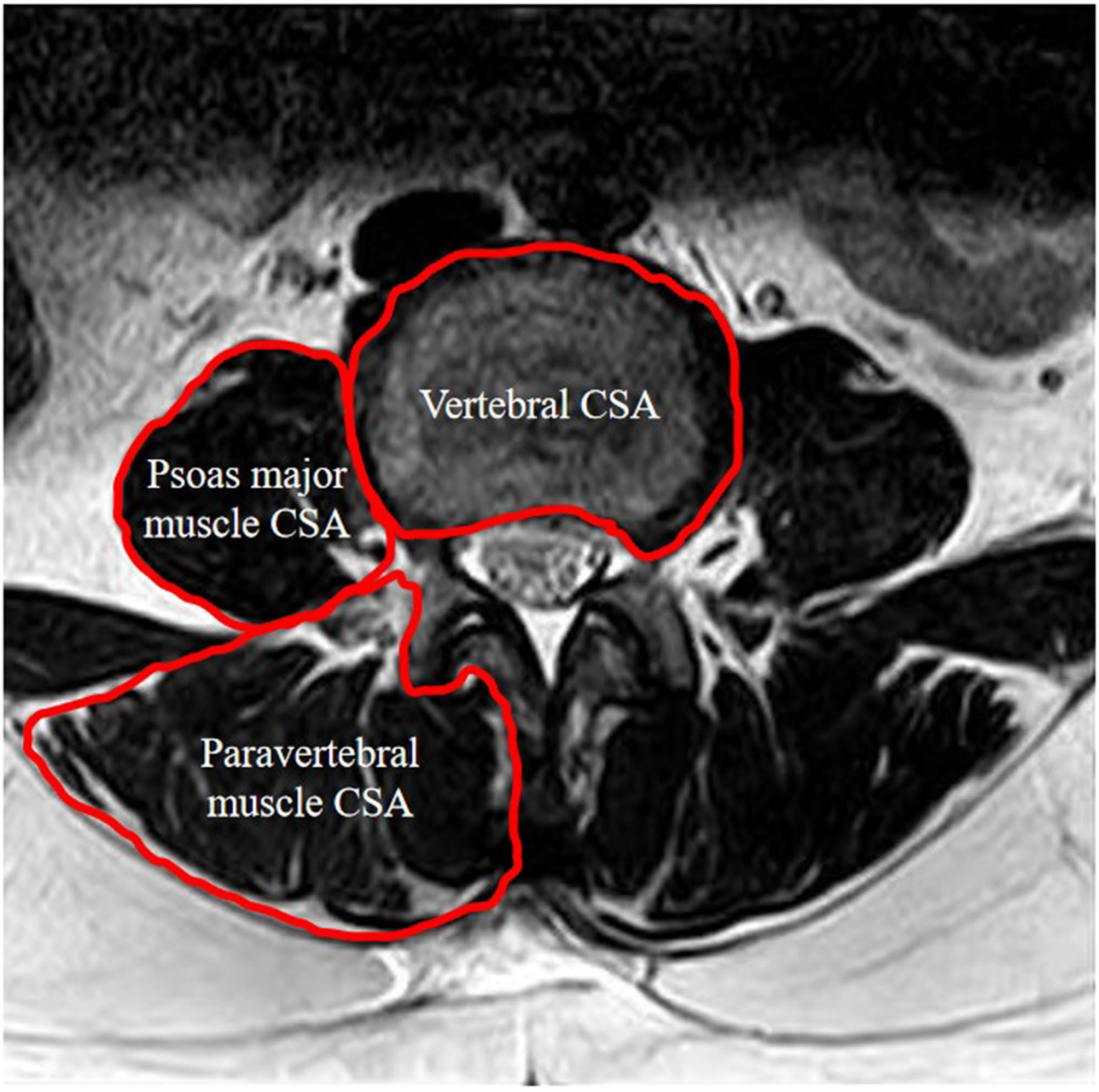

Fig. 1 Measurement of paravertebral muscle and psoas major muscle

Results

Consistent results

In order to evaluate the inter-observer and inter-observer consistency, ICC values were calculated. The intra-observer ICC values and inter-observer ICC values were 0.871 and 0.819, respectively, showing a good consistency.

General information

We collected 119 patients who met the criteria of this study, all of whom successfully completed PLIF surgery. Among them, 17 patients with ASDis were followed up after surgery, accounting for 14.3%. In the ASDis group, 6 males and 11 females were included, aged 56.18 ± 12.26 years, and the follow-up time was 36.34 ± 5.19 months. A total of 102 patients without ASDis were included in the asymptomatic group (accounting for 85.7%), including 14 males and 16 females, aged 53.59 ± 12.10 years and followed up for 34.98 ± 4.79 months. There were no statistically significant differences between the two groups in age, sex, drinking history, smoking history, diabetes history, hypertension history, course of disease, etiology and follow-up time, etc. There were significant differences in BMI between the two groups, and the BMI of

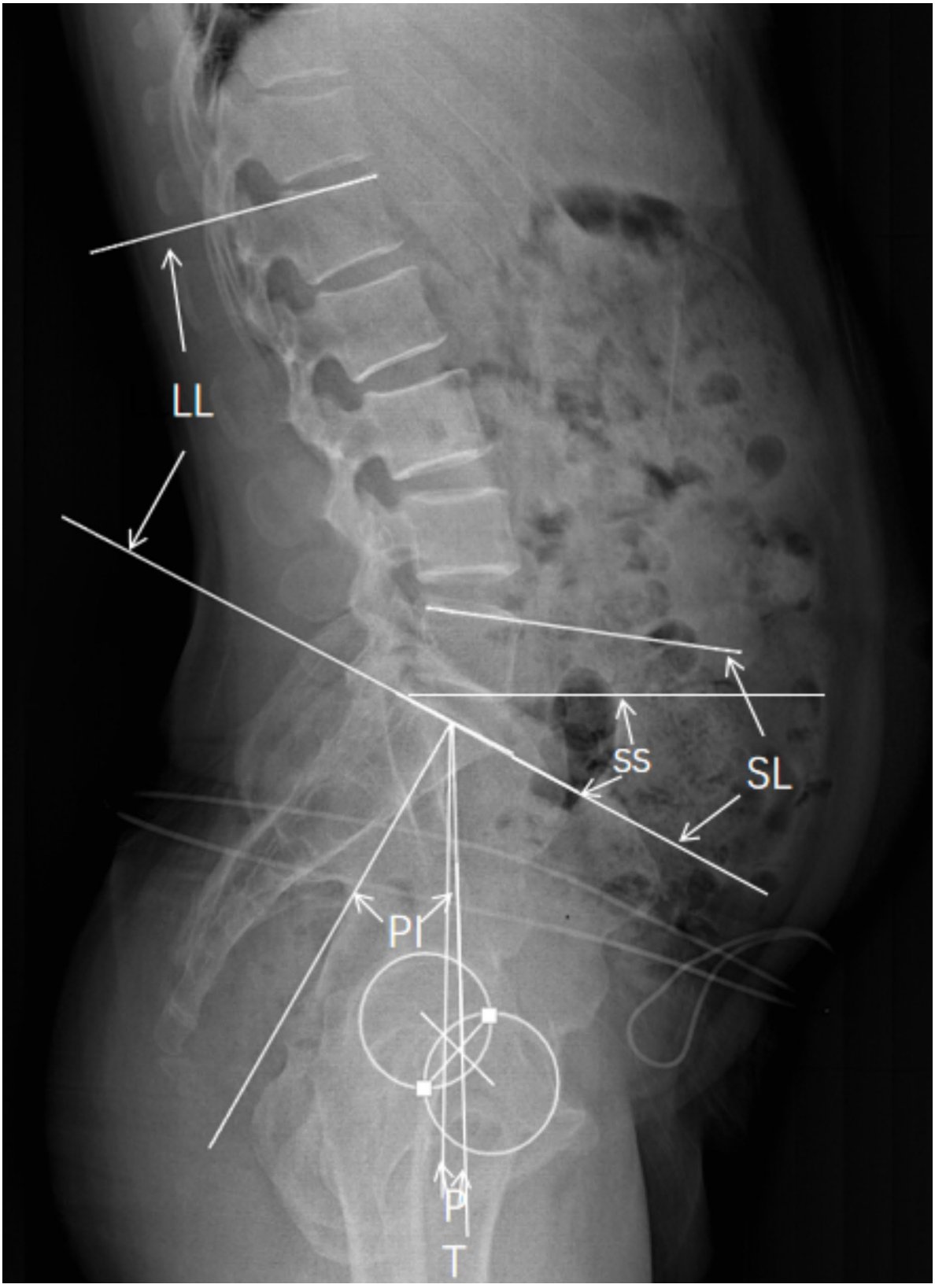

Fig. 2 Sagittal position parameter measurement method

Table 1 Comparison of basic information between the two groups

Indicated	ASDis group ($n=17$)	asymptomatic group ($n=102$)	*P* value
Age (years, $\bar{x}\pm s$)	56.18 ± 12.26	53.59 ± 12.10	0.417
Gender (e.g., male/female)	6/11	43/59	0.595
BMI (kg/m2, $\bar{x}\pm s$)	27.55 ± 3.99	25.18 ± 3.83	0.021
Drinking (e.g., yes/no)	4/13	27/75	0.798
Smoking (e.g., yes/no)	6/11	18/84	0.093
Diabetes (e.g., yes/no)	9/8	36/66	0.165
High blood pressure (e.g., yes/no)	11/10	23/47	0.105
Osteoporosis (e.g., yes/no)	9/8	21/81	0.004
Bone mineral density (T value, g/cm2, $\bar{x}\pm s$)	-1.54 ± 1.68	-0.01 ± 2.02	0.004
Duration of disease (months, $\bar{x}\pm s$)	5.82 ± 2.43	4.96 ± 2.22	0.146
Etiology (e.g., DH/SS/LS)	4/8/5	47/36/19	0.211
Follow-up time (months, $\bar{x}\pm s$)	36.34 ± 5.19	34.98 ± 4.79	0.286
Surgical fusion segment (segment, $\bar{x}\pm s$)	1.76 ± 0.75	1.28 ± 0.52	0.020

Table 2 Comparison of perioperative data between the two groups

Indicated	ASDis group ($n=17$)	asymptomatic group ($n=102$)	*P* value
Operation time (h, $\bar{x}\pm s$)	1.88 ± 0.29	1.96 ± 0.34	0.351
Incision length (cm, $\bar{x}\pm s$)	10.24 ± 2.05	10.56 ± 1.87	0.516
Intraoperative blood loss (ml, $\bar{x}\pm s$)	300.00 ± 86.60	269.12 ± 91.48	0.197
Surgical fusion segment (segment, $\bar{x}\pm s$)	1.76 ± 0.75	1.28 ± 0.52	0.020
Ground travel time (d, $\bar{x}\pm s$)	2.76 ± 0.66	2.81 ± 0.66	0.776
Incision healing (d, $\bar{x}\pm s$)	11.18 ± 1.78	11.58 ± 1.79	0.402
Length of hospitalization (d, $\bar{x}\pm s$)	10.41 ± 2.29	11.10 ± 2.31	0.687
L4/5 Corrected vertebral space height (mm, $\bar{x}\pm s$)	2.71 ± 1.21	2.10 ± 1.10	0.037

the ASDis group was significantly higher than that of the asymptomatic group ($P=0.021$). The number of osteoporosis patients and bone mineral density were significantly different between the two groups ($P=0.004$, $P=0.004$) (Table 1).

Perioperative situation

All the operations were successfully completed. One patient in the asymptomatic group had delayed incision healing, while the other patients had no abnormal incision, and all patients were healed. There was a significant difference in the number of fusion segments between the two groups. The number of fusion segments in the ASDis group was significantly higher than that in the asymptomatic group ($P=0.020$). There was also a significant difference in the correction height of the L4/5 intervertebral space between the two groups, and the correction height in the ASDis group was greater than that in the asymptomatic group ($P=0.037$). There were no significant differences in operation time, incision length, intraoperative blood loss, ground time, hospital stay and walking time between the two groups ($P>0.05$) (Table 2).

Preoperative degeneration of paravertebral muscle and psoas major muscle

The CSA of paravertebral muscle was (4478.37 ± 727.54) mm2 in ASDis group and (4987.47 ± 915.98) mm2 in asymptomatic group, and the difference was statistically significant ($P=0.031$). The rCSA of paravertebral muscle was (3.14 ± 0.82) in ASDis group and (3.87 ± 0.89) in asymptomatic group, and the difference was statistically significant ($P=0.002$). The rFCSA of the paravertebral muscle of ASDis was (2.37 ± 0.68) and that of the asymptomatic group was (2.96 ± 0.77), the difference was statistically significant ($P=0.003$). There were no significant differences in psoas major CSA, psoas major rCSA, vertebral body CSA, paravertebral FI, paravertebral fat CSA and paravertebral muscle FCSA between the two groups ($P>0.05$) (Table 3).

Imaging data

There were significant differences in LL and SL between the two groups before surgery, 2 days after surgery and the last follow-up, and the ASDis group was significantly smaller than the asymptomatic group, the difference was statistically significant ($P<0.05$). There were no significant differences in PT, SS, PI-LL and PI between the two groups before surgery, 2 days after surgery and at the last follow-up ($P>0.05$). There were 6 patients with L5/S1 gas in ASDis group and 13 patients in asymptomatic group, and there was a significant difference between the two groups ($P=0.019$). The degree of L5/S1 stenosis before surgery was significantly different between the ASDis group (grade 0/1/2, 7/5/5) and the asymptomatic group

Table 3 Comparison of preoperative degeneration of paravertebral muscle and psoas major muscle

Indicated	ASDis group ($n=17$)	asymptomatic group ($n=102$)	*P* value
Psoas major muscle CSA(mm2, $\bar{x}\pm s$)	2238.63 ± 710.70	2250.32 ± 671.50	0.948
Psoas major rCSA ($\bar{x}\pm s$)	1.54 ± 0.54	1.73 ± 0.55	0.175
Paravertebral muscle CSA(mm2, $\bar{x}\pm s$)	4478.37 ± 727.54	4987.47 ± 915.98	0.031
Paravertebral muscle rCSA ($\bar{x}\pm s$)	3.14 ± 0.82	3.87 ± 0.89	0.002
Vertebral body CSA(mm2, $\bar{x}\pm s$)	1092.86 ± 417.13	1163.90 ± 350.12	0.453
Paravertebral muscle FI(%, $\bar{x}\pm s$)	24.26 ± 8.31	23.84 ± 7.48	0.833
Paravertebral fat CSA(mm2, $\bar{x}\pm s$)	559.48 ± 197.30	630.39 ± 219.32	0.258
Paravertebral muscle FCSA(mm2, $\bar{x}\pm s$)	3385.51 ± 620.09	3823.57 ± 921.35	0.062
Paravertebral muscle rFCSA ($\bar{x}\pm s$)	2.37 ± 0.68	2.96 ± 0.77	0.003

Table 4 Comparison of imaging data between ASDis group and asymptomatic group

Indicated	ASDis group ($n=17$)	asymptomatic group ($n=102$)	*P* value
Preoperative image			
LL (°, $\bar{x}\pm s$)	34.10 ± 13.83	41.75 ± 13.38	0.032
PT (°, $\bar{x}\pm s$)	16.06 ± 8.48	19.94 ± 10.23	0.142
SS (°, $\bar{x}\pm s$)	28.60 ± 10.41	30.90 ± 8.39	0.316
PI-LL (°, $\bar{x}\pm s$)	10.48 ± 11.73	8.47 ± 14.02	0.578
SL (°, $\bar{x}\pm s$)	15.83 ± 5.07	22.77 ± 4.68	0.022
PI (°, $\bar{x}\pm s$)	44.58 ± 10.56	50.22 ± 11.61	0.063
L5/S1 intervertebral space height (mm, $\bar{x}\pm s$)	8.91 ± 1.44	9.40 ± 1.75	0.281
Gas in the L5/S1 process (e.g., yes/no)	6/11	13/89	0.019
L5/S1 Degree of spinal canal stenosis (e.g., grade 0/1/2)	7/5/5	69/25/8	0.019
L5/S1 Disc Pfirrman grading ($\bar{x}\pm s$)	3.41 ± 0.94	3.11 ± 0.72	0.125
L5/S1 disc herniation calcification (e.g., yes/no)	3/14	22/80	0.713
Endplate Modic change (e.g., I/II/III/ no)	3/5/4/7	23/16/5/56	0.048
2 days after surgery			
LL (°, $\bar{x}\pm s$)	38.11 ± 11.73	43.70 ± 10.02	0.038
PT (°, $\bar{x}\pm s$)	15.41 ± 6.38	18.13 ± 9.18	0.243
SS (°, $\bar{x}\pm s$)	28.77 ± 7.28	32.63 ± 7.86	0.061
PI-LL (°, $\bar{x}\pm s$)	6.50 ± 6.79	7.41 ± 9.36	0.702
SL (°, $\bar{x}\pm s$)	15.75 ± 3.92	19.82 ± 5.46	0.004
Last follow-up			
LL (°, $\bar{x}\pm s$)	37.19 ± 11.99	43.70 ± 11.34	0.032
PT (°, $\bar{x}\pm s$)	14.26 ± 5.80	17.89 ± 9.47	0.129
SS (°, $\bar{x}\pm s$)	29.92 ± 8.17	33.08 ± 7.42	0.112
PI-LL (°, $\bar{x}\pm s$)	7.49 ± 8.12	7.40 ± 10.81	0.973
SL (°, $\bar{x}\pm s$)	13.50 ± 3.27	16.00 ± 4.78	0.041
Intervertebral fusion time (months, $\bar{x}\pm s$)	6.12 ± 1.65	5.51 ± 1.57	0.145
Failure of postoperative internal fixation	1	3	0.533
Sacral lumbalization (e.g., yes/no)	2/15	19/83	0.492

(grade 0/1/2, 69/25/8) ($P=0.035$). There were significant differences in preoperative endplate Modic between the ASDis group (I/II/III/ no, 3/5/4/7) and the asymptomatic group (I/II/III/ no, 23/16/5/56) with statistical significance ($P=0.048$). There were no significant differences in preoperative L5/S1 intervertebral space height, intervertebral fusion time, postoperative internal fixation failure, preoperative L5/S1 intervertebral disc Pfirrman grade and intervertebral disc herniation calcification between the two groups ($P>0.05$) (Table 4).

Follow-up data

VAS score and ODI score in ASDis group were significantly higher than those in asymptomatic group at the last follow-up, and the difference was statistically significant. VAS scores and ODI scores were not significantly different between the two groups before surgery, 2 days after surgery and 3 months after surgery ($P>0.05$) (Table 5).

Multi-factor logistic regression analysis

With the occurrence of ASDis as the causal variation and other factors as independent variables, binary

Table 5 VAS and ODI scores

Indicated	ASDis group (*n* = 17)	asymptomatic group (*n* = 102)	*P* value
VAS score for low back pain			
Before operation	7.43 ± 0.60	7.57 ± 0.63	0.354
2 days after surgery	5.29 ± 1.01	4.84 ± 0.64	0.066
3 months after surgery	2.05 ± 0.67	2.06 ± 0.64	0.945
Last follow-up	1.33 ± 0.80	1.35 ± 0.97	0.933
ODI Score			
Before operation	47.11 ± 5.87	48.28 ± 8.06	0.538
2 days after surgery	34.71 ± 4.11	35.61 ± 4.98	0.456
3 months after surgery	24.29 ± 4.68	24.24 ± 4.15	0.962
Last follow-up	15.95 ± 5.15	16.25 ± 4.85	0.809

multi-factor logistic regression analysis was performed, as shown in Table 2. The classification ability of the model was 92.4%, and the model was valid after Chi-square test (x2 = 47.624, $P < 0.001$). BMI (OR = 1.715, $P = 0.001$) and number of surgically fused segments (OR = 4.245, $P = 0.030$) were risk factors for ASDis after the preoperative degeneration of L5/S1. The degree of spinal canal stenosis grade 0 (OR = 0.028, $P = 0.003$), the paraverteal muscle rFCSA (OR = 0.346, $P = 0.036$), and the Angle of SL on the second day after surgery (OR = 0.746, $P = 0.007$) were protective factors for the occurrence of ASDis after the preoperative degeneration of L5/S1 (Table 6).

ROC curve

ROC curve showed that the area under SL curve 2 days after operation was 0.703. The area under the curve of paravertebral muscle rFCSA was 0.716. The area under the BMI curve was 0.721. The area under the number curve of fusion segments is 0.518 (Fig. 3).

Discuss

Research status of ASDis after PLIF

Posterior lumbar fusion surgery (PLIF) is an incredibly mature technology that is applied in clinical treatment by many spinal surgeons [10]. It is appropriate for patients with minimally invasive operations, difficult operations, and complicated conditions. Full exposure of the visual field can better ensure the safety and operability of surgery. PLIF and related fusion methods are now the gold standard of spinal fusion. The technique was first proposed by Briggs and Milligan [11] and the excised laminae were first chopped as intervertebral space plants for fusion. Due to technological development and the rise of various materials, the use of different materials and types of fusion apparatus and internal fixation in clinical treatment, such as surgical titanium and polyether ether ketone (PEEK), has served to reduce the failure of internal fixation. The effect PLIF surgery has is worthy of recognition, but the etiology and preventive measures of postoperative degeneration of adjacent fusion segments have long been misunderstood by many clinical orthopedic surgeons [12]. ASD is a complication that is associated with spinal fusion surgery. One study reported spinal fusion surgery to result in degenerative changes in adjacent segments of the fused spine earlier than would have naturally occurred [13]. Studies on the exact cause are still ongoing.

Factors including the age, gender, and BMI of patients are closely related to the occurrence of ASD after fusion. Radcliff et al. [14] believed smoking to increase the risk of adjacent segment degeneration, and the older the patients were, the higher their incidence of adjacent segment degeneration after fusion. Wang et al. [15] conducted a retrospective study on 237 patients, finding the incidence of adjacent segment degeneration to be significantly increased in patients with BMI > 25 kg·m2. Abnormal estrogen receptors in postmenopausal women can cause facet arthritis, which can result in the degeneration of adjacent segments. Relevant studies have also shown osteoporosis to be an important cause of adjacent segment degeneration. Anti-bone pine therapy can improve bone mass and vertebral microstructure quite significantly, in addition to maintaining the height of the intervertebral disc and reducing the calcified area of the end plate, thereby reducing degeneration occurrence [16].

Although the exact ASD mechanism is not fully understood, in biomechanical terms, increased range

Table 6 Results of multi-factor logistic regression analysis ofpreoperative adjacent segment degeneration L5/S1 segment occuring postoperative ASDis

Influencing factor	B value	S.E.	Wald value	OR value	95% CI	*P* value
Paravertebral muscle rFCSA	-1.061	0.505	4.412	0.036	0.346	0.129–0.931
BMI	0.540	0.168	10.342	0.001	1.715	1.235–2.384
L4/5 Immediate correction of vertebral space height	0.026	0.330	0.006	0.938	1.026	0.537–1.961
Bone mineral density	0.063	0.209	0.092	0.761	1.065	0.708–1.604
Number of surgically fused segments	1.446	0.666	4.712	0.030	4.245	1.151–15.662
Postoperative L5/S1 SL Angle	-0.293	0.110	7.166	0.007	0.746	0.602–0.924
Postoperative LL Angle	0.077	0.052	2.208	0.137	1.080	0.976–1.196
Combined with pneumococcal process	1.860	1.028	3.276	0.070	6.424	0.857–48.146
Spinal stenosis is grade 0	-3.587	1.222	8.624	0.003	0.028	0.003–0.303

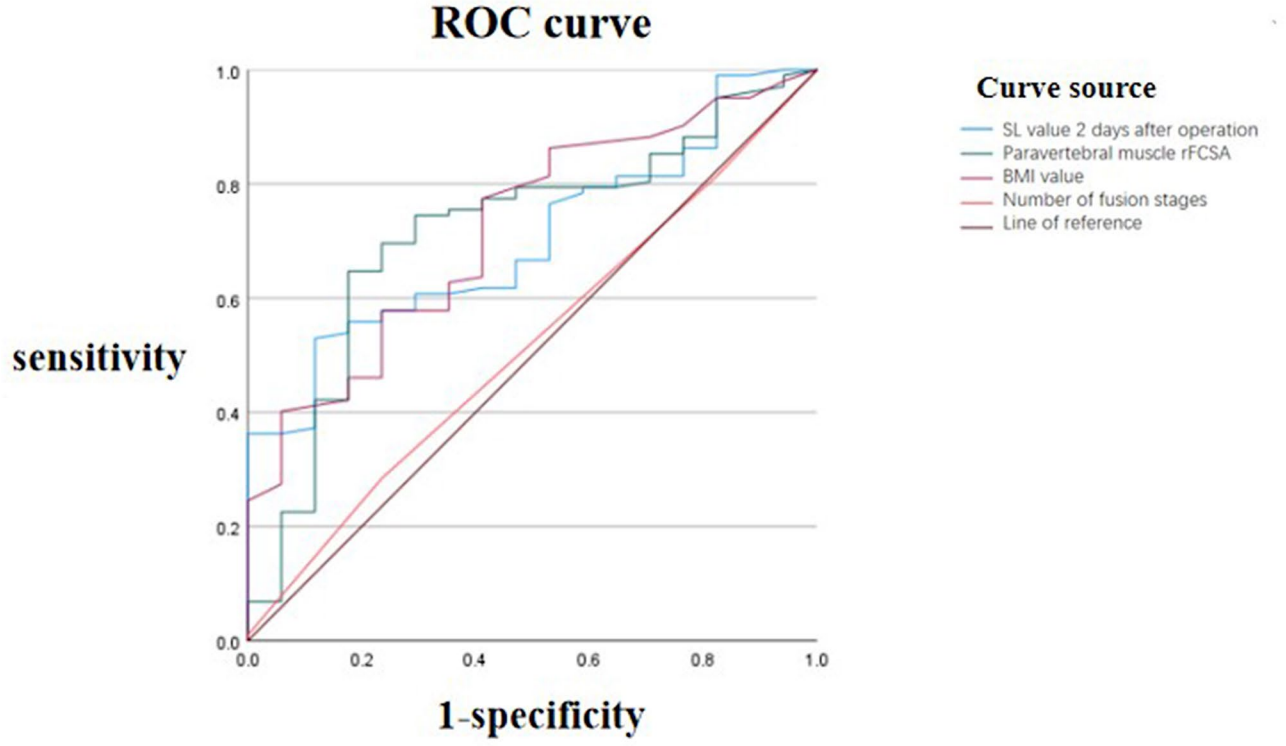

Fig. 3 ROC curve

of motion in adjacent segments and disc pressure are the causes that are most likely. In 1984, Lee and Langrana [17] conducted in vitro mechanical experiments and found that the range of motion and disc pressure of adjacent segments increased significantly following lumbar fusion and fixation. Jiang et al. [18] highlighted that after PLIF, the pressure of adjacent annulus fibrosus and nucleus pulposus increased most significantly during extension, inevitably leading to accelerated degeneration of the intervertebral disc over time. Excessive pressure caused by activity after fusion is an important reason for the degeneration of adjacent segments. In addition, existing adjacent segment degeneration prior to surgery is an influential factor for the occurrence of adjacent segment degeneration following surgery. Postoperative biomechanical changes will accelerate the degeneration and further worsen it. Existing degeneration is a response to human overload, and further aggravation of the degenerative segmental load following PLIF will result in a series of pathological changes and symptoms. Anandjiwala et al. [6] conducted a controlled study and found patients with signs of intervertebral disc degeneration at adjacent levels before lumbar fusion and fixation to have a significantly increased incidence of postoperative adjacent level degeneration.

Regarding surgery-related factors, the increase in the number of surgically fused segments caused the risk of adjacent segment degeneration to increase significantly. In the follow-up of patients after lumbar fusion and internal fixation, Cillet et al. [19] concluded that the longer the fusion segment, the higher the incidence of adjacent segment degeneration. They also found that the longer the fusion segment, the greater the compensatory motion of its adjacent segments will be, and the greater the pressure on the intervertebral disc and the process joints, thereby accelerating degeneration [20]. In addition, intraoperative correction of sagittal position parameters has important clinical significance for adjacent segment degeneration occurrence. Spinal-pelvic sagittal balance is considered one of the important factors that affect lumbar degeneration. Masevnin et al. [21] reported that PI-LL mismatch is a risk factor for adjacent segment instability – patients with high PI values and low LL values having a significantly increased risk of adjacent segment instability following short segment fusion. A study by Nakashima et al. [22] identified excessive PI as a high risk factor for early-onset ASDeg following lumbar fusion. Le Huec et al. [23] found postoperative PT increase to be significantly associated with postoperative back pain and the risk of adjacent segment degeneration. Min et al. [24] noted that correct lumbar lordosis can serve to prevent ASDs occurrence. The aforementioned studies all discuss the relationship between different sagittal position parameters and ASD. Although these studies have many limitations, they also reflect the important influence postoperative sagittal position parameters have

on ASD occurrence. Many factors affect the occurrence of ASDis, and clinical studies are not comprehensive, so ASDis prevention and treatment should be considered comprehensively.

Research significance for exploring the risk factors for ASDis after preoperative L5/S1 degeneration

The L5-S1 segment is the lumbosacral junction with unique segmental motor and biomechanical properties. It is also a susceptible transition point between the mobile lumbar spine and the rigid pelvis. L5-S1 is different from other lumbar vertebra segments and is closely related to the conversion of standing and sitting posture. The human body controls the forward and backward tilt of the pelvis through the mutual change of PT and SS as a means of achieving the mutual conversion of sit-standing. It can also adjust the compensatory spine and maintain the balance of the spine. However, PT and SS adjustment changes are closely related to the L5-S1 segment. SS and PT adjustment of the patient are severely limited after L5-S1 segmental fusion and the patient will have significant limitations in squatting. In addition, the sacroiliac joint will have compensatory stress loa, and in severe cases, sacroiliac joint torsion may result in sacroiliac joint pain, which will affect the quality of life of the patient. Clinical studies have demonstrated that the incidence of ASD in spinal fusion to S1 is significantly higher than that in fusion to L5, and the risk of proximal borderline kyphosis and fixation failure is greater due to "leverage" fusion to the L5-S1 segment [25]. Therefore, careful consideration should be given to the choice of L5-S1 fusion surgery. However, L5-S1 is one of the segments that has a high clinical failure rate and the most common occurrence of lumbar degenerative changes [26]. When the clinically responsible segment consists of a single segment that includes L4-L5 or multiple segments above it, a common problem that disturbs clinicians is whether L5-S1 with severe degeneration is fused together. Fusion segment selection has long been controversial. In a study by Anandjiwala et al. [6], the follow-up results of patients following lumbar fusion showed the risk of ASDis after lumbar fusion to increase significantly for adjacent segment degeneration that existed prior to surgery. Although ASDis after this segment was avoided after fusion, the incidence of adjacent segment degeneration at the head end was found to increase after fusion. Furthermore, the increase of fusion segment increases surgical trauma and impacts the quality of life of patients quite significantly. However, ASDis may occur after surgical fusion due to biomechanical changes for L5-S1 with relatively severe degeneration without treatment or intervention [27]. In severe cases, a second operation may be required, but due to the influence of the first operation, the difficulty, risk, and trauma of the second operation are all higher, and the effect is often worse than that of the first operation. Therefore, summarizing and analyzing all the factors of ASDis after preoperative L5/S1 degeneration, exploring the high risk factors of its occurrence, analyzing independent pathogenic factors with statistical analysis, and actively intervening and treating to prevent the occurrence of ASDis are all essential steps. If there are many high risk factors and the expected effect of non-surgical preventive treatment is not good, a surgical fusion treatment can be appropriate. In conclusion, analyzing the risk factors of ASDis following L5/S1 degeneration before surgery is essential in surgical protocol design and preventive treatment measure formulation.

Result analysis

Through comparison and analysis of the general data, perioperative data, VAS score, ODI score, and imaging data (including paravertebral and psoas major muscle data, sagittal position parameter data, postoperative follow-up and occurrence data, etc.) of the ASDis and asymptomatic groups, the risk factors of postoperative ASDis occurrence were identified. These risk factors were included in the bivariate logistic regression analysis, and BMI, number of surgical fusion segments, grade 0 spinal stenosis, paraspinal muscle rFCSA, and SL Angle on the second day following surgery were all found to be independent related factors for ASDis occurrence following preoperative L5/S1 degeneration. The reliability and effectiveness of the above indexes were analyzed by ROC curve.

Influence of paravertebral muscle degeneration on the occurrence of ASDis following preoperative adjacent segment degeneration

Due to the issue of an aging population, the number of patients with lumbar diseases is increasing quite significantly, and the lower back muscles are receiving greater attention in the research of disease prevention and treatment. Paravertebral muscle degeneration occurs at the cellular level and changes including myolysis, endothelial cell degeneration, and decreased number of mitochondria are frequently observed. At the same time, at the tissue level, connective tissue increase and capillary density decrease occur. Morphological manifestations of paravertebral muscle degeneration are muscle atrophy and steatosis, which is muscle volume reduction that is characterized by reduced muscle fiber volume and decreased effective muscle proportion characterized by adipose tissue infiltration of muscle [28]. The CSA of paravertebral muscle measured by MRI can reflect the quantity of paravertebral muscle. FCSA or FI can reflect the mass of paravertebral muscle and can also be used as an indicator for the evaluation of the degree of paravertebral muscle degeneration. Following a comparison of healthy

people with non-specific lower back pain, Pan Fuwei et al. [29] found FI to have a correlation with gender, age, lumbar lordosis Angle, lumbar disc degeneration, and other imaging parameters. This demonstrated that the stability of the lumbar spine requires the stability of bone and joint structure and position, and the restriction and binding force provided by muscles and ligaments. Wang Sinian et al. [30] highlighted that paravertebral muscle degeneration has a close relationship with sagittal position parameters, and the paravertebral muscle affects the sagittal position force line as a means of maintaining spinal sagittal position balance through a compensatory mechanism. According to a retrospective study by Hyun [31], preoperative thoracolumbar muscle mass was lower and fat infiltration was higher among adult spinal malformation patients with proximal borderline kyphosis. Lumbar and back muscle degeneration and strength decline were identified as independent risk factors. In addition, multiple studies [32–34] have shown paravertebral muscle degeneration to have a close association with lower back pain, spinal disequilibrium, osteoporotic compression fractures, adjacent vertebral disease, and recovery following open surgery.

The inclusion of paravertebral muscle CSA, rCSA, FCSA, and FI was analyzed in this study, and paravertebral muscle rFCSA was innovatively proposed as an observational index. The study showed the CSA, rCSA, and rFCSA of paravertebral muscle in the ASDis group to all be smaller than those in the asymptomatic group ($P < 0.05$). Paravertebral muscle CSA, rCSA, and rFCSA all have important effects on ASDis occurrence after preoperative L5/S1 degeneration, and the reduction of these three indicators may result in ASDis occurrence. Regression analysis showed that paravertebral muscle rFCSA can be used as an independent factor that affects postoperative ASDis occurrence, which is in accordance with the findings of previous studies. The paravertebral muscle is an important muscular system that helps stabilize the spine during normal lumbar physiological lordosis and dorsal extension maintenance [35]. When the paravertebral muscle atrophy is severe, spinal stability is reduced and the compensatory stress of the process joints and intervertebral discs is increased, thereby accelerating degeneration. In severe cases, the compression nerve will cause ASDis. When spinal load increases in vertebral space degeneration, the paravertebral muscle compensates as a means of maintaining spinal stability. When the paravertebral muscle atrophy weakens compensatory ability and when the excess load is difficult to compensate, the further degeneration of the vertebral space and the joint process is decelerated. In addition, relevant studies have shown the paravertebral muscle to have a close relationship with osteoporosis [36], which is an important cause of adjacent segment degeneration. Bone mass and vertebral microstructure can be significantly improved and load capacity increased by Aatibone pine therapy, which maintains vertebral space height and delays the degeneration rate. In comparison to FCSA and rCSA, paravertebral muscle rFCSA reflects the strength degree of the paravertebral muscle of a person more individually, and is more specific in maintaining spinal stability. The importance of quality and quantity of paravertebral muscle in postoperative prevention of ASDis is demonstrated above. Paravertebral muscle degeneration and atrophy result in a decreased likelihood of ASDis occurrence. Therefore, paravertebral muscle functional exercise should be performed as soon as possible after surgery by patients with paravertebral atrophy, particularly those with preoperative adjacent segment degeneration At the same time, stability of the lumbar spine should be maintained to improve the mechanical load of paravertebral muscle and prevent postoperative ASDis occurrence.

Influence of sagittal position parameters on the occurrence of ASDis following preoperative adjacent segment degeneration

The load distribution of the spine is more dependent on its shape and curvature in the sagittal plane than on the coronal plane. Poor sagittal alignment following lumbar fusion can result in increased stress concentration in adjacent segments, which can cause adjacent segment diseases. Therefore, finding suitable sagittal balance after surgery is essential for preventing postoperative adjacent segment degeneration occurrence. This study showed LL and SL in the ASDis group to be significantly lower than those in the asymptomatic group before surgery, two days after surgery, and at the last follow-up ($P < 0.05$), which indicates that insufficient recovery of SL and LL angle following surgery causes ASDis occurrence. Umehara S et al. [23] found postoperative kyphosis in the internal fixation segment to lead to increased non-physiological load on adjacent segment structures, and the destruction of the posterior column structure was found to increase the stress imposed on the posterior column, leading to the proliferation and inflammation of facet joints and ultimately, ASDis occurrence. Zhou et al. [37] highlighted that when the human body changes from a standing position to a sitting position, lumbar lordosis and thoracic kyphosis decrease or disappear, the center of gravity shifting forward and the pelvis tilting backward. However, these changes were found to be relatively limited in patients following lumbar fusion, so adjacent unfused lumbar segments compensate for the pressure in a sitting position, which is potentially related to ASD occurrence. As L5/S1 is located in the lumbosacral junction, it has a greater burden to compensate for the sitting pressure following fusion, and the potential for ASDis after surgery

increases significantly if degeneration occurs before the merger. As Pinto et al. [38] and Rothenfluh et al. [39] reported, sagittal alignment of the spine, particularly maintaining a normal spine-pelvic relationship, is essential for ASD prevention following intervertebral fusion.

However, clinical studies on rational sagittal alignment after surgery remain unclear. Therefore, when exploring a reasonable sagittal alignment that can reduce the impact of fusion on the biomechanics of adjacent segments following surgery, particularly for those with adjacent segment degeneration before surgery, reasonably adjusting the sagittal alignment to reduce the impact on adjacent stages is of great importance. In a recent study results by zhao et al. [40] showed that as LL decreases, the stress of the adjacent disc increases. If the ideal lordosis angle is not restored following surgery, which will result in the loss of LL, and the continuous load of the adjacent segments exceeds the spine-pelvic compensatory capacity, this will cause flat back deformity, adjacent segment degeneration, and fracture. When the lumbar lordosis is small, the contact force acts mainly on the spine and the front of the disc, which increases both disc pressure and degeneration risk. In addition, the lumbar sagittal position arrangement of posterior internal fixation fusion has been analyzed in relevant studies and the lumbar lordosis was found to be reduced by approximately 10° on average in patients with degenerative degeneration at adjacent unfused segments following surgery [41]. Kumar et al. [38] described the biomechanical effects lumbar kyphosis has on the internal fixed segment and adjacent segments following lumbar fusion, and it was found to accelerate the deterioration of adjacent segments by loading the movement phase in a non-physiological manner. The loss of anterior convexity of the fixed segment affects the adjacent segment while also increasing the load of posterior spinal fixation. Clinical studies have also shown excessive PT to be a high risk factor for postoperative ASD. Excessive PT leads to reduced SS, which will cause reduced lower lumbar lordosis and increased intervertebral disc stress, thereby accelerating degeneration occurrence [42]. It can be seen that sagittal position parameters are of great significance in the prevention or slowing down of postoperative ASDis occurrence. This is similar to the results of this study where LL and SL are risk factors for postoperative ASDis, and SL can be used as an independent factor to cause ASDis occurrence. In comparison to LL, it is believed that SL may be more specific, and as a part of LL, SL can better reflect the mechanical characteristics of a segment. When a segment of SL is too small, the up and down pressure of the intervertebral disc increases, and when the pressure load of the intervertebral disc exceeds, this leads to the accelerated degeneration of the intervertebral space. Part of the pressure of the intervertebral disc is carried by the facet joints, the paraspinal muscles, and the peripheral ligaments, and the reduced pressure load of the intervertebral disc reduces postoperative ASDis possibility. Therefore, recovering a better SL angle following surgery is of positive significance for the prevention of postoperative ASDis occurrence for the L5/S1 segment that has been degraded before surgery. The sagittal position parameters are of great significance for the prevention of postoperative complications and should be included in the surgical design.

Influence of other risk factors on the occurrence of ASDis following preoperative adjacent segment degeneration

This study found BMI to be a risk factor for ASDis following L5/S1 degeneration. BMI in the ASDis group was found to be significantly higher than in the asymptomatic group ($P<0.05$), and excessive BMI can lead to ASDis occurrence. Following further statistical analysis, it can be used as an independent pathogenic factor for promoting ASDis occurrence. This is consistent with the findings of the study by Bagheri [43], which demonstrated patients with higher preoperative BMI to have a statistically increased risk of developing ASD. Liang et al. [44] also reached the conclusion that BMI can be used as an independent predictor of postoperative ASDis and the incidence of ASDis significantly increases if BMI is too large. BMI value is a standard that is commonly used for measuring the weight of the human body. The larger a BMI value is, the heavier the weight of a body is and the greater the human pressure that is carried by the intervertebral disc and facet joints will be. When the load of the intervertebral disc is exceeded, pathological changes will occur to the intervertebral disc and facet joints and degeneration will be accelerated. The pressure that is generated by the compensatory activity of adjacent segments following interbody fusion and the high BMI combined with the decrease of the preoperative degenerative pressure load will significantly increase the likelihood of ASDis. Therefore, both preoperative and postoperative weight management is essential for lumbar degenerative disease prevention. Health guidance should be provided for patients with excessive BMI after surgery to ensure normal BMI is maintained and the occurrence of postoperative ASDis is prevented.

It was also found that the number of fusion segments can be an independent risk factor for ASDis occurrence following degenerative segment surgery. In a study that was conducted by Zhang et al. [45], it was reported that active segments adjacent to fusion segments exhibited an increased range of motion, and the increase in motion of adjacent segments was proportional to the number of fusion segments. Ghiselli et al. [1] found the risk of ASD in multi-segment fusion to be three times that of single-segment fusion. Long segmental fusion causes decreased

elasticity and increased stiffness of the lumbar segment, making adapting to biomechanical changes in the adjacent moving segments difficult. This includes stress concentration and increased intradisc pressure, which serves to make the adjacent segments more susceptible to degeneration. When the adjacent segments have exhibited degeneration prior to surgery, carrying the compensatory movement after surgery becomes more difficult, further accelerating the aggravation of the degraded segments and potentially leading to ASDis. Therefore, clinicians should consider the fusion treatment of non-essential surgical segments carefully, and the operation should preserve the active segments to the greatest possible extent as a means of preventing ASD occurrence while achieving the surgical objective. The number of fusion segments should be reduced as much as possible to reduce the possibility of ASDis occurrence for patients with adjacent segment degeneration.

Previous studies prove that the degree of spinal canal stenosis in preoperative MRI is an independent risk factor for postoperative ASDis, and the higher the degree of preoperative stenosis, the higher the incidence of postoperative ASDis will be [46]. This study concluded that when the spinal canal morphology of preoperative MRI was 0 grade, it was a protective factor for postoperative ASDis, and when the spinal canal stenosis degree was 0 grade, the incidence of postoperative ASDis was lower in L5/S1 patients with preoperative degeneration. Spinal canal morphology classification includes the MRI imaging classification that Lee et al. [47] proposed in 2011, which classifies the degree of cerebrospinal fluid occlusion above the cauda equina nerve in the dural and reflects the degree of spinal canal stenosis, which is reliable and easy to evaluate. However, other clinical scholars have verified the relationship between morphological classification and clinical practice, proposing that spinal canal morphology grade 0 generally has no clinical symptoms. It is unclear whether spinal canal morphology grade 1 has clinical symptoms, and patients with spinal canal morphology grades 2 and 3 will have corresponding clinical symptoms [48]. However, in this clinical study, patients with grade 2 dysphoria did not have corresponding lower extremity symptoms, which could be related to personal neurological adaptation. Relatively few clinical studies have examined the relationship between preoperative MRI spinal canal morphology and postoperative ASDis. In a follow-up study, Cho et al. [49] concluded that preoperative degenerative spinal stenosis of adjacent segments is a risk factor for postoperative ASD and secondary repair surgery. In a retrospective study, Yugue et al. [50] found sagittal stenosis to be more than 47% of the important risk factors for postoperative ASD after lumbar fusion. Therefore, preoperative evaluation of the spinal canal morphology of the degraded L5/S1 is of great importance, and necessary preventive measures should be taken when the spinal canal morphology of patients is higher, including minimally invasive partial decompression surgery or interlaminar fenestration neurolysis.

The height of vertebral space reduction in the preoperative degenerative segment adjacent to the fusion segment is also a risk factor for postoperative ASDis. Kaito et al. [51] followed up 58 patients with posterior lumbar interbody fusion for (38.8 ± 17.1) months, dividing the patients into no adjacent vertebral degeneration, imaging only adjacent vertebral degeneration, and accompanying symptoms groups. The average respective span heights of the three groups were 3.1, 4.4, and 6.2 mm, which suggests a close relationship between span height and ASD. Previous biomechanical tests have demonstrated that the stress of the upper facet joint increases by 8% when the space height of the operative segment is increased by 2 mm [52], and the intervertebral motion of the adjacent segment and disc stress increase with the height of the fusion device. In a biomechanical study, Lu et al. [53] noted that in order to reduce the degeneration of adjacent segments, the height of the segmental fusion device should not exceed the preoperative vertebral space height by 2 mm for patients with mild to moderate vertebral space degeneration. In addition, the height of the fusion apparatus should be as close as possible to the preoperative height of the intervertebral space for patients with severe degeneration. Therefore, to prevent excessive spacing of the vertebral space and accelerate the degeneration of adjacent segments, surgeons should carefully consider the reduction height of the vertebral space when designing an operation.

Limitations

Firstly, this study is a retrospective study that has a small sample size, and this may be affected by unknown or unmeasured confounding variables, making it prone to publication bias. Secondly, it is a single-center study that may be affected by the surgical mode and thinking of regions or hospitals. Different surgical methods and concepts can impact surgery-related data. Thirdly, it explored the high risk factors, but there is still an insufficient amount of clinical studies on relevant preventive measures and a general lack of adequate discussion. Fourthly, the factors included in the study are insufficient. Factors including the angle of articular process, the space area of the foramen, and the sagittal diameter of the spinal canal should be included in the analysis to comprehensively study ASDis risk factors. Fifth, the study follow-up time was relatively short. Finally, the sagittal position parameters were measured manually, so there is the potential for errors to have occurred.

Conclusions

From the above discussion, it can be seen that there are many risk factors for ASDis occurrence following preoperative degeneration. BMI, the number of surgical fusion segments, and SL angle on the second day after surgery are independent risk factors for ASDis occurrence after surgery in this study. rFCSA of the paravertebral muscle could be an independent protective factor for postoperative ASDis. In addition, several other factors may affect postoperative ASDis occurrence, including bone mineral density, the reduction height of the vertebral space adjacent to the operation, LL, and gas in the articular process. Therefore, the prevention of ASDis after the operation of degraded L5/S1 should be comprehensively considered and multiple factors should also be considered so the ultimate goal of prevention and treatment can be achieved.

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Author contributions
Yan Liu: Methodology, Software, Investigation, Writing—original draft. Nianhu LI: Supervision, Project administration. Huapeng Guan: Conceptualization, Resources, Supervision, Project administration. Juan Yu: Conceptualization, Resources.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study was performed in line with the principles of the Declaration of Helsinki. The studies involving human participants were reviewed and approved by the Human Research Ethics Committee of Affiliated Hospital of Shandong University of Traditional Chinese Medicine. All study participants signed a written informed consent form.

Consent to publish
Not applicable.

Competing interests
The authors declare no competing interests.

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