



# Adjacent segment degeneration after posterior lumbar fusion: An analysis of possible risk factors



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## ABSTRACT

**Objective:** Many surveys have been distributed about the risk factors for adjacent segment degeneration (ASD) after lumbar fusion. Despite myriad of risk factors recognized for ASD evolution, study results have been inconsistent and there is not an agreement regarding which are the most important. Our study was done to identify factors which may be important in the development of symptomatic ASD after lumbar fusion.

**Patients and methods:** This retrospective study evaluated 1250 consecutive patients who underwent posterior lumbar fusion and pedicular fixation between February 2006 and February 2009. A total of 13 patients with symptomatic ASD (clinical ASD) who underwent secondary surgery were identified. Another group of 22 patients without symptomatic ASD (subclinical ASD) after spinal fusion were marked as the control group. These two groups were compared for demographic data and clinical and radiographic features to investigate the possible predictive factors of symptomatic ASD.

**Results:** The overall incidence rate of symptomatic ASD was 1.04%. Radiographic risk factors for the development of a symptomatic ASD were increased sagittal balance, loss of lordosis, and adjacent disc space collapse. In the clinical ASD group, by multivariate logistic regression analysis, demonstrated that BMI, preoperative ADD on MRI and disc bulge maintained their significance in predicting likelihood of clinical ASD.

**Conclusion:** Patients with increased BMI, preoperative ADD and disc bulge on MRI have a statistically significant increased risk of developing symptomatic ASD.

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## 1. Introduction

Spinal fusion has been studied as a means to treat pathology related to the spine for more than a century. Spinal fusion was first described by Albee for the treatment of Pott's disease [1], and then by Hibbs [2], who performed spinal fusion as a treatment for spinal deformity. One complication of spinal fusion is adjacent segment disease (ASD) following cervical, lumbar, or lumbosacral fusion. This is a broad term that encompasses symptoms such as listhesis, instability, herniated nucleus pulposus, stenosis, hypertrophic facet arthritis, scoliosis, and vertebral compression fracture [3]. These poor outcomes are known to follow the accelerated degenerative changes at the adjacent segments after fusion [4]. Although the development of adjacent segment deterioration is a part of the normal aging process, this process may be accelerated by the altered mechanics that occur with lumbar fusion. Several studies

have examined the risk factors that put a portion of patients at risk for ASD. Nevertheless, these are still controversial. The aim of this study was to assess the real incidence of adjacent segment disease after posterior lumbar spinal fusion, and to identify the predictors for the pathology.

## 2. Materials and methods

The study population in this retrospective study (an observational study using analytic retrospective study cohort design) consisted of 1250 consecutive patients of degenerative lumbar disease who had undergone posterior lumbar fusion and pedicle screw fixation in our institute between February 2006 and February 2009. Those patients undergoing surgery for nondegenerative disease (trauma, infection, tumor, deformity, inflammation) and patients in whom hook or hybrid constructs and other fusion procedures (non-instrumented posterolateral fusion, transforaminal lumbar interbody fusion, posterior lumbar interbody fusion and anterior lumbar interbody fusion) were used were excluded. Criteria of degenerative change at adjacent segments: radiological degener-

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ative change at the adjacent segments was considered to exist when anterior or posterior displacement of >3 mm was found on the X-ray of the sagittal plane of the closest upper segment and the closest lower segment at the last follow-up, when the height of the intervertebral disc relative to that of the upper interbody had declined by 20% and when a segmental motion instability of more than 15° was observed on the X-ray of the sagittal plane with flexion and extension. Clinical ASD was defined as symptomatic spinal stenosis, mechanical back pain, and sagittal or coronal imbalance. The cohort identified was divided into patients undergoing surgery (clinical ASD) and those who did not (subclinical ASD). The clinical ASD group was consisted of patients who underwent secondary surgery because of clinical findings compatible with adjacent segment pathology. The subclinical ASD group made up of patients who had radiographic evidence of ASD but did not develop symptoms in agreement with adjacent segment disease during follow-up period. Only those patients develop symptoms who had a minimum of five-year follow-up were enrolled. Demographic data included age at the time of primary surgery, gender, smoking history, body mass index (BMI), and diagnosis. Follow-up was defined as the time from primary surgery to reoperation in the clinical ASD group and the period after surgery in the control group. Surgical data collected included levels of fusion, and bone graft material (autograft vs. allograft). Radiographs at the primary surgery and at the final follow-up or before an additional surgery were evaluated in each group to ascertain the lumbar lordotic angle, the distance between the L1 and the S1 sagittal plumb lines, the sagittal slope angle of the superior end plate of S1, adjacent disc angle, and adjacent disc height. In addition, magnetic resonance imaging (MRI) was used to discover whether there had been a degree of preoperative adjacent disc degeneration (ADD). Patients shown to have grade  $\geq$ III in the five-grade classification of Pfirrmann et al. [5] based on MRI were considered to have a degenerative change. The lumbar lordotic angle was measured by Cobb's method made by the superior endplate of the L1 and the superior endplate of the S1. The L1 sagittal plumb line was drawn with a lateral gravity plumb line from the center of L1. The center of L1 was noted by the intersection of crossing diagonals of vertebral body of L1 on the lateral radiograph. The S1 sagittal plumb line was drawn with a lateral gravity plumb line from the posterior end of S1 vertebrae. The distance between the plumb lines was measured as the shortest perpendicular distance between the two lines. The sagittal slope angle of the superior end plate of S1 was measured as the angle between a horizontal line and the superior end plate of S1. The adjacent disc angle was measured as the angle between the caudal and cranial end plates of the disc

just adjacent to the upper or lower fused levels. The adjacent disc height was measured on lateral radiograph from the middle of the superior border of the disc to the middle of the inferior border of the disc just adjacent to the upper or lower fused levels. The criteria used to prove solid fusion in patients were: no lucencies around the pedicle screws, no discernible movement on dynamic flexion and extension lateral X-rays.

Factors associated with clinical ASD were identified using univariate analysis. The data analysis was performed using SPSS version 19.0 (Chicago, IL, USA). Continuous variables were compared between the two groups using the student *t* test, whereas discrete variables were analyzed using the chi-squared test. Fisher's exact test was used for small data subsets ( $n < 5$ ). All significance tests were two-tailed, with  $p < 0.05$  representing statistical significance. In addition, a multivariate logistic regression analysis was performed to identify which factors helped predict the probability of a clinical ASD.

### 3. Results

A summary of the clinical data before spinal fusion for the clinical ASD and control groups is presented in Table 1. 25 cases were lost to follow-up (10 were moved to another province, 5 were died due to other reasons and 10 were refused to participate in follow-up study). The radiologic successful fusion rate in our study was 78%. Of the 13 patients identified as having clinical ASD, 1 had disc herniation, 1 had lumbar spondylolisthesis, and 11 had lumbar stenosis. The other group of 22 patients with subclinical ASD also were evaluated: 7 had disc herniation, 2 had lumbar spondylolisthesis, and 13 had lumbar stenosis. Mean age at index surgery was  $59.6 \pm 11.7$  years for the group of clinical ASD and  $60.7 \pm 10.5$  years for the other group. Clinical ASD was observed in 1.04% (13 of 1250) of patients. The average body mass index (BMI) for the clinical ASD and control group was  $27.4 \pm 1.9$  and  $25.8 \pm 2.3$ , respectively ( $p = 0.042$ ). No significant differences were observed between the two groups in age, gender, smoking, levels of fusion, type of adjacent segment abnormality and graft material.

The preoperative MRI was evaluated; of the 13 patients with clinical ASD, 9 patients (69.2%) were recorded to have a degenerative change. However, in the control group, only 7 patients (31.8%) were noted to have a degenerative change preoperatively ( $p = 0.031$ ). The disc bulge was also statistically associated with increased risk of clinical ASD: it was found in 12 of 13 in the group

**Table 1**  
Demographic data and surgery associated variables of the patients.

Variables	Clinical ASD (n = 13)	Subclinical ASD (n = 22)	p Value
Age	59.6 $\pm$ 11.7	60.7 $\pm$ 10.5	0.775
Gender:			
Male	4	6	0.825
Female	9	16	
Smoking:			
Yes	4	5	0.596
No	9	17	
BMI	27.4 $\pm$ 1.9	25.8 $\pm$ 2.3	0.042
Levels of fusion	2.6 $\pm$ 0.87	2.3 $\pm$ 0.66	0.256
Adjacent segment pathology:			
Lumbar stenosis	11	13	0.116
Lumbar spondylolisthesis	1	2	
Disc herniation	1	7	
Allograft:			
Yes	10	13	0.284
No	3	9	

**Table 2**  
Radiologic features of patients.

Variables	Clinical ASD (n = 13)	Subclinical ASD (n = 22)	p Value
Preoperative ADD:			
Yes	9	7	0.031
No	4	15	
Preoperative disc bulge:			
Yes	12	2	0.000
No	1	20	
Preoperative L1 to S1 sagittal plumb line (mm)	25.1 ± 12.9	14.2 ± 16.2	0.046
Preoperative lumbar lordosis	35.5 ± 13.8	45.5 ± 14.1	0.049
Preoperative adjacent disc angle	8.9 ± 5.1	9.7 ± 4.9	0.648
Preoperative adjacent disc height (mm)	9.1 ± 1.8	11 ± 2.3	0.015
Preoperative S1 sagittal slope angle	32.9 ± 9.8	33.6 ± 8.5	0.825
Postoperative L1 to S1 sagittal plumb line (mm)	18.2 ± 16.3	15.2 ± 13.8	0.565
Postoperative lumbar lordosis	31.3 ± 9.6	39.3 ± 10.6	0.032

with clinical ASD, as compared with 2 of 22 in the matched cohort ( $p=0.000$ ).

The following radiographic findings did not differ significantly between the two groups: preoperative S1 sagittal slope angle, preoperative adjacent disc angle, postoperative distance from L1 to S1 sagittal plumb line, postoperative S1 sagittal slope angle, and postoperative adjacent disc angle (Table 2). Radiographic risk factors for developing symptomatic ASD were increased sagittal balance, loss of lordosis, and adjacent disc space collapse.

In the clinical ASD group, by multivariate logistic regression analysis, demonstrated that BMI, preoperative ADD on MRI and disc bulge maintained their significance in predicting likelihood of clinical ASD.

#### 4. Discussion

Adjacent segment deterioration after lumbar fusion is of great concern and it accounts for a substantial amount of revision spine surgery. Although some of the progression of degeneration may be attributed to natural processes of aging, this process is influenced by the abnormal stresses that occur following lumbar fusion [6,7]. Biomechanical research has shown a significant increase in mobility in the proximal motion segments following spinal fusion [8]. The incidence of ASD has been reported to range from 5.2% to 100%, with higher rates generally found using radiographic criteria rather than symptomatology [9]. Lower rate of ASD in our study (3%) may be due to more rigid radiographic inclusion criteria in relation to other studies and other fusion methods except for instrumented posterolateral fusion were excluded from the study, so many patients were omitted from the study. There is substantial reporting in the literature regarding the development of adjacent level disease following lumbar fusions. Many investigators have reported their clinical series and demonstrated conflicting results. Lehmann et al. did a retrospective study of 62 patients at a mean of 33 years following lumbar fusion, and they noted segmental instability above the fusion in 45% of patients in whom follow-up radiographs were available. Fifty-seven percent of patients reported episodes of back pain in the previous year and 53% required pain medication. However, radiographic deterioration of the adjacent segment did not correlate with symptoms [10]. Etebar and Cahill performed a retrospective analysis on 125 consecutive patients who had undergone lumbar fusion with rigid instrumentation for degenerative instability at an average of four-years follow-up. A total of 18 of 125 patients (14%) developed symptomatic adjacent segment degeneration at a previously asymptomatic level, 15 of whom were postmenopausal females (83%). The investigators concluded that the risk of adjacent segment degeneration is higher for patients with rigid instrumentation performed to treat degenerative instability, and

that this risk appears to be particularly high in postmenopausal females [11]. Nagata et al. [12], that involved humans and animals, reported that the mobile segments adjacent to the fusion segment showed an increased range of motion, and the increase in motion at adjacent segments was in proportion to the number of the fused vertebrae. However, Soh et al. [13] found that there was little relationship between ASD and number of fusion segments. Also, Hilibrand et al. [14] found that the risk of clinical adjacent-segment pathology after multilevel fusion was significantly less than after single-level fusion. These findings are contrary to the results expected if the biomechanical consequences of fusion were the only cause of adjacent-segment pathology. Therefore, it remains controversial whether this accelerated adjacent-level degeneration is caused by the natural progression of aging or instead by increased motion stress related to biomechanical factors secondary to surgical fusion itself.

Despite the considerable amount of research, the causes of adjacent segment degeneration are not fully understood and risk factors that induce or trigger changes in the adjacent mobile segments are plentiful and based of our knowledge the predictive factors of clinical ASD in patients after posterior lumbar fusion have not been identified.

In this study, we have tried to compare some aspects of patients with clinical ASD who underwent surgery and those who did not. Among demographic and surgery related factors only BMI was significantly different between two groups. Among radiographic features, preoperative adjacent disc degeneration, preoperative disc bulge, preoperative L1 to S1 sagittal plumb line, preoperative lumbar lordosis, preoperative adjacent disc height, postoperative lumbar lordosis and postoperative adjacent disc height were significantly different between two groups. After multivariate logistic regression analysis, we found that BMI, preoperative adjacent disc degeneration and disc bulge on MRI had highest odds ratio and they could be the best predictors of clinical ASD.

#### 5. Conclusion

Risk factors that induce or trigger changes in the adjacent mobile segments are plentiful. Our study suggested that BMI, preoperative adjacent disc degeneration and disc bulge on MRI could be reasonable predictors for clinical ASD after posterior lumbar fusion and pedicular fixation.

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