Clinical Neurology and Neurosurgery 143 (2016) 15–18

Contents lists available at ScienceDirect

Clinical Neurology and Neurosurgery

journal homepage: www.elsevier.com/locate/clineuro

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

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ARTICLE INFO

Article history:
Received 1 December 2015
Received in revised form 30 January 2016
Accepted 4 February 2016
Available online 6 February 2016

Keywords:
Adjacent segment degeneration
Lumbar fusion
Pedicular screw
Risk factor

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.

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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http://dx.doi.org/10.1016/j.clineuro.2016.02.004
0303-8467/

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 ascretain 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 ≥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 ± 11.7 years for the group of clinical ASD and 60.7 ± 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 ± 1.9 and 25.8 ± 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 ± 11.7	60.7 ± 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 ± 1.9	25.8 ± 2.3	0.042
Levels of fusion	2.6 ± 0.87	2.3 ± 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	