Risk Factors for Adjacent Segment Disease Development after Lumbar Fusion

Sergei Masevnin, Dmitry Ptashnikov, Dmitry Michaylov, Hao Meng, Oleg Smekalenkov, Nikita Zaborovskii

Vreden Russian Research Institute of Traumatology and Orthopedics, Saint-Petersburg, Russia

Study Design: Retrospective cohort study.
Purpose: To identify factors which may be important in the occurrence of symptomatic adjacent segment disease (ASD) after lumbar fusion.
Overview of Literature: Many reports have been published about the risk factors for ASD after lumbar fusion. Despite on the great numbers of risk factors identified for ASD development, study results have been inconsistent and there is controversy regarding which are the most important.
Methods: This study evaluated 120 patients who underwent 360° fusion lumbar surgery from 2007 to 2012. We separated the population into two groups: the first group included 60 patients with long lumbar fusion (three or more levels) and the second group included 60 patients with short lumbar fusion (less than three levels).
Results: In the first group, symptomatic ASD was found in 19 cases during the one year follow-up. There were 14 cases with sagittal imbalance and 5 cases at the incipient stage of disc degeneration according to the preoperative magnetic resonance imaging. At the three year follow-up, symptomatic ASD was diagnosed in 31 cases, of which 17 patients had postoperative sagittal balance disturbance. In the second group, 10 patients had ASD at the one year follow-up. Among these cases, preoperative disc degenerative changes were identified in 8 patients. Sagittal imbalance was found only in 2 cases with symptomatic ASD at the one year follow-up. At the three year follow-up, the number of patients with symptomatic ASD increased to 14. Among them, 13 patients had initial preoperative adjacent disc degenerative changes.
Conclusions: Patients with postoperative sagittal imbalance have a statistically significant increased risk of developing symptomatic ASD due to an overloading the adjacent segments and limited compensatory capacities due to the large number of fixed mobile segments. In the case of a short fixation, preoperative degenerative changes are more important factors in the development of ASD.

Keywords: Fusion; Adjacent segment disease; Sagittal balance

Introduction

Spinal segment arthrodesis has become a widely accepted treatment for numerous pathologic conditions of the spine [1]. Currently, rigid internal fixation with 360° fusion has been viewed as the gold standard for spine stabilizing surgery. However, spinal fusion alters the normal biomechanics of the spine and eliminates mobile
segments, causing an overload of the adjacent segments. Thus, spinal fusion, according to some authors, can accelerate the degeneration of adjacent segments [2-5].

The main pathological changes of the adjacent segments include disc degeneration, segmental instability, hypertrophic facet arthritis, spinal stenosis, osteophyte formation, scoliosis, and vertebral compression fracture [6].

The number of risk factors for adjacent segment disease (ASD) development is constantly increasing and can be separated into two basic categories according to the classical studies: patient factors, which are usually do not depend on the surgeon, and surgical factors, which can directly be modified by the surgeon during the operation [5,7-11].

The most important patient factors include sex, age, body mass index, smoking, preexisting degeneration of adjacent discs, and menopause.

The surgical factors include the position and length of fixation, stiffness of the implants, technical fusion execution errors, and sagittal and coronal balance disturbance.

Despite the great numbers of risk factors for ASD development, there are inconsistent results and a determination of the most important risk factors has not been established [6,12-15].

To identify factors (both radiographic and on magnetic resonance imaging [MRI]) which may be important regarding the occurrence of symptomatic ASD after lumbar fusion.

**Materials and Methods**

This retrospective study evaluated 120 patients who underwent 360° fusion lumbar surgery from 2007 to 2012 for the treatment of degenerative conditions of the lumbar spine. The follow-up rate was 90%. The subject population was 64% female. The average age was 56 years (range, 19–78 years), and the average follow-up period was 36.0 months (range, 24–84 months). The mean follow-up was 3 years (range, 2–7 years). We compared two groups: the first group included 60 patients with long lumbar fusion (three or and more levels). Among them, 17 patients had degenerative spondylolisthesis, 31 patients had multi level spinal stenosis, and 12 patients had herniated lumbar discs with segmental instability. The second group include 60 patients with short lumbar fusion (1–2 levels). This group included patients with single level degenerative spondylolisthesis (22), a herniated lumbar disc with segmental instability (14), and degenerative spinal stenosis (24). MRI evaluation of the adjacent segment’s condition and long cassette standing anteroposterior and lateral radiographs were performed during the preoperative, postoperative, and follow-up visits. All patients had no signs of preoperative radiographic instability on the adjacent levels as compared with the normal height of the adjacent discs. In all cases we studied the preoperative, postoperative, and follow-up sagittal plane alignments according to the Schwab Sagittal Modifiers (Fig. 1).

**Coronal curve types**

- **T:** thoracic only
  - with lumbar curve <30°

- **L:** thoracolumbar (TL)/lumbar (L) only
  - with thoracic curve <30°

- **D:** double curve
  - with T and TL/L curves >30°

- **N:** no major coronal deformity
  - all coronal curves <30°

**Sagittal modifiers**

**Pl minus LL**

- 0: within 10°
- +: moderate 10–20°
- ++: marked>20°

**Global alignment**

- 0: SVA<4 cm
- +: SVA 4 to 9.5 cm
- ++: SVA>9.5 cm

**Pelvic tilt**

- 0: PT<20°
- +: PT 20–30°
- ++: PT>20°

*Fig. 1. Curve type and 3 sagittal modifiers. PI, pelvic incidence; LL, lumbar lordosis; SVA, sagittal vertical axis; PT, pelvic tilt.*
The adjacent segment condition was evaluated by MRI and scored with a modified Pfirrmann classification (Table 1).

The findings from the measurements were analyzed statistically using simple regression analysis and Student's $t$-test. A $p$-value <0.05 was defined as statistically significant.

**Table 1. Modified Pfirrmann grading system of lumbar disc degeneration**

| Grade | Signal from nucleus and inner fibers of annulus | Distinction between inner and outer fibers of annulus at posterior aspect of disc | Height of disc |
|-------|-----------------------------------------------|--------------------------------------------------------------------------|----------------|
| 1     | Uniformly hyperintense (equal to CSF)         | Distinct                                                                 | Normal         |
| 2     | Hyperintense (>presacral fat and <CSF)       | Distinct                                                                 | Normal         |
| 3     | Hyperintense (>presacral fat)                | Distinct                                                                 | Normal         |
| 4     | Mildly hyperintense (slightly>outer fibers of annulus) | Indistinct                                                                 | Normal         |
| 5     | Hypointense (=outer fibers of annulus)       | Indistinct                                                                 | Normal         |
| 6     | Hypointense                                 | Indistinct                                                                 | <30% reduction of disc height |
| 7     | Hypointense                                 | Indistinct                                                                 | 30% to 60% reduction of disc height |
| 8     | Hypointense                                 | Indistinct                                                                 | >60% reduction of disc height |

CSF, cerebrospinal fluid.

The adjacent segment condition was evaluated by MRI and scored with a modified Pfirrmann classification (Table 1).

The findings from the measurements were analyzed statistically using simple regression analysis and Student’s $t$-test. A $p$-value <0.05 was defined as statistically significant.

**Results**

In the first group, symptomatic ASD was found in 19 cases (28%) during the one year follow-up. There were 14 cases (75%) with sagittal imbalance and 5 cases (25%) with pre-existing initial degenerative changes in adjacent discs according to the preoperative MRIs. The three year follow-up showed an increase in ASD to 31 cases (52%); however, at this period, the preoperative initial adjacent disc degenerative changes prevailed over the sagittal imbalance cases in a 3:1 ratio (9 cases with Pfirrmann 2–5 stage and only 3 cases with sagittal imbalance). In summary, after three years, the first group had 31 cases (52%) of symptomatic ASD, of which 17 patients (53%) had postoperative sagittal balance disturbance and 14 patients (47%) had preoperative incipient disc degeneration changes seen on the MRI. In all, 87% required revision surgery (Figs. 2, 3).

In the second group, 10 patients (17%) had symptomatic ASD at the one year follow-up. Among those cases, preoperative disc degenerative changes were identified in 8 patients (Pfirrmann 2–5 grade). Sagittal imbalance was found in only 2 cases with ASD at the on year follow-up. Fig. 2. Female, 60-year-old: Th11–L5 fusion, postoperative positive sagittal imbalance.
After three years, the number of patients with symptomatic ASD increased to 14 (23%). Among them, 13 patients had preoperative adjacent disc degenerative changes (Pfirrmann 2–5 grade), and while only 5 patients had postoperative sagittal balance disturbance (Figs. 4, 5).

**Discussion**

The occurrence of degenerative changes proximal to the level of lumbar fusions has been reported in many studies. 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 [6]. Biomechanical research has shown a significant increase in mobility in the proximal motion segments following spinal fusion [16]. Changes in the adjacent motion segment biomechanics were studied by Weinhoffer et al. [1] in a spine model, who concluded that intradiscal pressures increased within adjacent levels as flexion motion increased. Additionally, it was shown that the increase in pressure directly correlated with the number of levels fused.

Penta et al. [17] conducted a radiologic study to determine the correlation between the incidence of ASD and fusion length. The investigators analyzed 52 patients with at least ten years’ worth of follow-up data who had no pre-existing degenerative changes above the fusion according to MRIs. The researchers showed that 32% of the 52 patients had degenerative changes and the degenerative changes were independent of the length of the fusion.

Despite the considerable amount of research, the causes of adjacent segment degeneration are not fully understood.

The importance of sagittal balance after lumbar fusion has been documented in several clinical studies. Oda et al. [18] showed that a kyphotic lumbar fusion may lead to degenerative changes in adjacent facet joints. Akamaru
et al. [16] described a human cadaver study, finding that hypolordotic fusion at L4/5 causes segment instability at L3/4.

Today, even the dynamic stabilization of the spine does not solve the problem of the degeneration of the adjacent segments. According to the study of Strube et al. [19], hyper-mobility of adjacent levels is a manifestation of ASD development, despite dynamic fixation of the lumbar spine.

Our study showed that most important factor in the development of ASD after long lumbar fusion is sagittal balance. Patients with postoperative sagittal imbalance have a statistically significant increased risk of developing ASD due to an overloading of the adjacent segments and limited compensatory capacities due to the large number of fixed mobile segments. Theoretically, at the three-year follow-up, the decreasing role of sagittal imbalance in the ASD development can be explained by the rapid decompensation in adverse biomechanical conditions from the first preoperative year. On the other hand, in the short instrumentation we did not find a statistically significant dependency between ASD and postoperative sagittal imbalance. This may be due to a considerable number of the unfused mobile segments, which have a better ability to compensate the sagittal balance disturbance. In the case of a short fixation, pre-existing initial degenerative changes are more important in development of ASD. Undoubtedly, its occurrence rate depends on the degree of these preoperative degenerative changes and the sagittal imbalance grade, so further investigation is needed to clarify the development of symptomatic ASD.

Conclusions

According to our results, patients with postoperative sagittal imbalance have a statistically significant increased risk of developing symptomatic ASD due to an overloading of the adjacent segments and limited compensatory capacities due to the large number of fixed mobile segments. In patients with a short fixation, pre-existing initial degenerative changes are determinative of ASD development.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

References

1. Weinhoffer SL, Guyer RD, Herbert M, Griffith SL. Intradiscal pressure measurements above an instrumented fusion. A cadaveric study. Spine (Phila Pa 1976) 1995;20:526-31.
2. Bastian L, Lange U, Knop C, Tusch G, Blauth M. Evaluation of the mobility of adjacent segments after posterior thoracolumbar fixation: a biomechanical study. Eur Spine J 2001;10:295-300.
3. Chow DH, Luk KD, Evans JH, Leong JC. Effects of short anterior lumbar interbody fusion on biomechanics of neighboring unfused segments. Spine (Phila Pa 1976) 1996;21:549-55.
4. Rahm MD, Hall BB. Adjacent-segment degeneration
after lumbar fusion with instrumentation: a retrospective study. J Spinal Disord 1996;9:392-400.
5. Schlegel JD, Smith JA, Schleusener RL. Lumbar motion segment pathology adjacent to thoracolumbar, lumbar, and lumbosacral fusions. Spine (Phila Pa 1976) 1996;21:970-81.
6. Park P, Garton HJ, Gala VC, Hoff JT, McGillicuddy JE. Adjacent segment disease after lumbar or lumbosacral fusion: review of the literature. Spine (Phila Pa 1976) 2004;29:1938-44.
7. Aota Y, Kumano K, Hirabayashi S. Postfusion instability at the adjacent segments after rigid pedicle screw fixation for degenerative lumbar spinal disorders. J Spinal Disord 1995;8:464-73.
8. Cheh G, Bridwell KH, Lenke LG, et al. Adjacent segment disease following lumbar/thoracolumbar fusion with pedicle screw instrumentation: a minimum 5-year follow-up. Spine (Phila Pa 1976) 2007;32:2253-7.
9. Etebar S, Cahill DW. Risk factors for adjacent segment failure following lumbar fixation with rigid instrumentation for degenerative instability. J Neurosurg 1999;90:163-9.
10. Kaito T, Hosono N, Mukai Y, Makino T, Fuji T, Yonenobu K. Induction of early degeneration of the adjacent segment after posterior lumbar interbody fusion by excessive distraction of lumbar disc space. J Neurosurg Spine 2010;12:671-9.
11. Okuda S, Iwasaki M, Miyachi A, Aono H, Morita M, Yamamoto T. Risk factors for adjacent segment degeneration after PLIF. Spine (Phila Pa 1976) 2004;29:1535-40.
12. Ghiselli G, Wang JC, Bhatia NN, Hsu WK, Dawson EG. Adjacent segment degeneration in the lumbar spine. J Bone Joint Surg Am 2004;86:1497-503.
13. Hilibrand AS, Robbins M. Adjacent segment degeneration and adjacent segment disease: the consequences of spinal fusion? Spine 2004;4:190S-194S.
14. Kumar MN, Balkanov A, Chopin D. Correlation between sagittal plane changes and adjacent segment degeneration following lumbar spine fusion. Eur Spine J 2001;10:314-9.
15. Lehmann TR, Spratt KF, Tozzi JE, et al. Long-term follow-up of lower lumbar fusion patients. Spine (Phila Pa 1976) 1987;12:97-104.
16. Akamaru T, Kawahara N, Tim Yoon S, et al. Adjacent segment motion after a simulated lumbar fusion in different sagittal alignments: a biomechanical analysis. Spine (Phila Pa 1976) 2003;28:1560-6.
17. Penta M, Sandhu A, Fraser RD. Magnetic resonance imaging assessment of disc degeneration 10 years after anterior lumbar interbody fusion. Spine (Phila Pa 1976) 1995;20:743-7.
18. Oda I, Cunningham BW, Buckley RA, et al. Does spinal kyphotic deformity influence the biomechanical characteristics of the adjacent motion segments? An in vivo animal model. Spine (Phila Pa 1976) 1999;24:2139-46.
19. Strube P, Tohtz S, Hoff E, Gross C, Perka C, Putzier M. Dynamic stabilization adjacent to single-level fusion: part I. Biomechanical effects on lumbar spinal motion. Eur Spine J 2010;19:2171-80.