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Surgical risk factors associated with the development of adjacent segment pathology in the lumbar spine

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- Adjacent segment pathology (ASP) is a major cause of disability, and the recognition of the surgical risk factors associated with the development of this condition is essential for its prevention.
- Different surgical approaches, from decompression without fusion to non-instrumented and instrumented fusion, have distinct contributions to the development of ASP.
- Although motion-preservation procedures could reduce the prevalence of ASP, these are also associated with a higher percentage of complications.
- Several risk factors associated with previous surgery, namely the chosen surgical approach and anatomical dissection, the choice of interbody fusion, the increment and length of the fusion, and the restoration of sagittal alignment, may influence the development of ASP.

Keywords: adjacent segment degeneration; adjacent segment disease; adjacent segment pathology; lumbar fusion; lumbar spine

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Introduction

Spinal fusion is the most widely accepted treatment for lumbar disc degenerative disease.[1–3] However, it has been associated with adjacent segment degeneration (ASDeg) as a potential long-term sequel,[4,5] especially in those with preoperative risk factors, which may cause aberrant stress forces in these segments and lead to adjacent level degeneration.[6–8] Adjacent segment pathology can include ASDeg and adjacent segment disease (ASD), although a clear and consensual definition of ASD is missing. In most studies, ASDeg is defined as radiographic changes in the intervertebral discs adjacent to the surgically treated levels, whereas ASD is defined as the pathologic process associated with disc degeneration leading to clinical symptoms, such as radiculopathy, stenosis and instability.[9,10] Nevertheless, there are a few reports considering reoperation rate as being the most reliable parameter to define ASD, despite clinical symptoms.[11]

The incidence of radiographic evidence of ASDeg varies widely (from 8% to 100%) and is greatly overstated when compared with reported incidences of symptomatic ASD in the literature (5.2–18.5%).[4] On the other hand, the surgical revision rate represents only a portion of the symptomatic ASD (2–15%). In summary, radiographic signs of adjacent level degeneration after lumbar fusion are relatively common, although they do not comprise a mandatory clinical correlation, with only a small proportion of these patients needing revision surgery for symptoms directly associated with ASD.[12]

It is well documented that lumbar fusion accelerates degeneration of the adjacent segments (Fig. 1).[13] This article proposes to perform a review of the definition, epidemiology, pathophysiology, and surgical risk factors influencing the probability of developing adjacent segment pathology (ASP).

The influence of fusion on ASD development

The spine has a high biomechanical complexity, and understanding these concepts is essential to the correct diagnosis and treatment of spinal disorders. Intervertebral disks are essential to stabilize the spine and grant its flexibility, since they provide a biological shock absorber that stabilizes the spine while transmitting forces between two rigid segments. The concept of motion segment is extremely important and consists of two adjacent vertebrae, the intervertebral disk, two posterior facet joints, capsules and the ligaments spanning the two segments. The symbiosis between these structures provides a safe

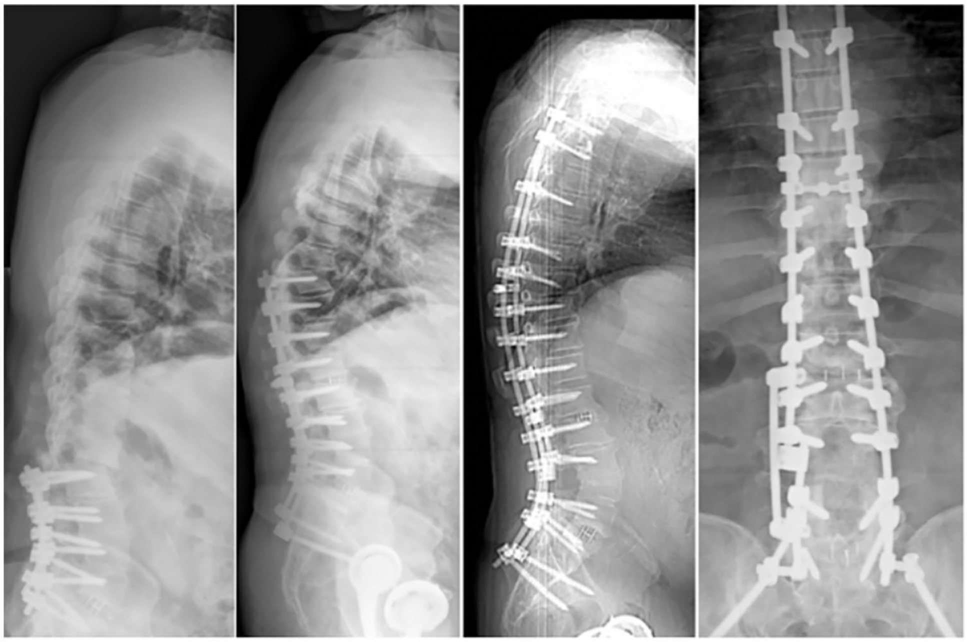

Fig. 1 (a) Case example of an adjacent segment disease (ASD) after L2-S1 fusion. (b) First revision was made with a pedicle subtraction osteotomy at L3-L4, TLIF L1-L2 and T10 to ilium posterior fusion extension. (c and d) After one year of follow-up the patient developed an ASD at the cephalic level. The second revision was made with a T2 to ilium posterior fusion extension.

physiological range of motion and, at the same time, a protection for the spinal cord and the nerve roots.[14] Motion segment instability is the primary indication for arthrodesis, which leads to increased stress on the adjacent cephalic levels, presumably from a larger lever arm and non-physiological centre of motion.[15] The incidence and prevalence of ASD has been extensively studied, although the factors that influence the potential for adjacent segment degeneration following spinal fusion remain controversial.[9] Different surgical approaches, from decompression without fusion to non-instrumented and instrumented fusion, have potentially distinct contributions for the development of ASD.

Decompression without fusion

Although ASD has traditionally been attributed to fusion, any intervention, including laminectomy alone, could result in ASD. The largest study in the literature regarding ASD after laminectomy in patients with spinal stenosis (N = 9644) reported a rate of 8% at five years.[16] Bydon et al,[17] in a study involving 500 patients undergoing initial 1 to 3 level laminectomies without fusions for degenerative lumbar disease, presented an overall reoperation rate of 14.4%, with ASD being responsible for 54.1% of these cases. Simultaneous decompression adjacent to the fusion was described as a prophylactic procedure in situations of mild degenerative stenosis of the adjacent level.[18] However, when performed without associated fusion, this approach sacrifices the integrity of the posterior complex between the fused and the adjacent segment, which may jeopardize adjacent segment stability and cause accelerated development of ASD.[18,19] Two prospective studies from Sears et al[20] and Ekman et al[13] have reported laminectomy adjacent to a fusion as a risk factor for the development of ASD. Therefore, once a simultaneous decompression surgery at the adjacent level is selected, fusion should also be recommended in order to stabilize the motion of this fragile segment.

Non-instrumented and instrumented fusion as risk factors for ASD

Currently, rigid internal fixation with 360° fusion is regarded as a common procedure for spine stabilization surgery. However, spinal fusion alters the normal spine biomechanics and eliminates mobile segments, causing an overload of the adjacent segments. Reoperation rates following non-instrumented lumbar fusions are typically lower than those for instrumented fusions.[21,22] Park et al[4] reported an increased risk of reoperations due to ASD after transpedicular fusions versus non-instrumented fusions. Although some studies have reported a lower rate of degeneration with anterior lumbar interbody fusion (ALIF) alone, the fusion method (circumferential vs. interbody vs. posterolateral) is not consistently associated with an increased rate of ASD.[23-26] Biomechanically, the increment of interbody fusion (IF) in the posterior fusion is associated with immediate postoperative biomechanical stability and a high fusion rate.[27–29] However, posterior lumbar interbody fusion (PLIF) has been reported to be more rigid than posterior lumbar fusion (PLF), raising concerns that the strong mechanical stability of PLIF may possibly increase mechanical stress and accelerate the degeneration of adjacent segments.[30] Lee et al[31] published a comparative study between PLIF and PLF. They found a 10-year prevalence of ASD requiring reoperation of 10.4% in the combined cohorts, with an increased risk of ASD requiring reoperation when PLIF was performed (by a factor of 3.4 compared with PLF).

In surgical literature, the average reoperation rate after lumbar fusion is highly variable, presumably because of its dependency on intrinsic factors, the procedure of interest, the length of follow-up and several specific surgical aspects such as the inclusion of interbody fusion or instrumentation, number of laminectomy levels and length of fusion. However, none of these factors have been definitively associated with ASD in all studies.[32–36] Sears et al[20] reported a reoperation rate for ASD after lumbar spine fusion of 2.5% per year with a 10-year prevalence of 22.2%. Ghiselli et al[12] predicted a 10-year incidence of ASD of 36.1%, with 27.4% requiring adjacent level surgery. Pan et al[32] published a meta-analysis in 2016 demonstrating a prevalence of ASDeg, ASD and reoperation rate after lumbar fusion of 37.5%, 14.4% and 7.7%, respectively. Thus, despite being an asset in the treatment of multiple pathologies of the spine, surgical fusion is associated with an increased risk of developing ASD.

The importance of spine dissection

When considering anatomical preservation during the lumbar spine fusion, the facet joints have an important role as anatomical protective elements of the motion segment, mainly in relation to anterior shear forces, excessive rotation and flexion. Regarding transpedicular instrumentation technique and the potential for development of ASDeg, it is important not to damage the inferior facet of the cephalic adjacent segment.[37,38] Thus, anatomical dissection is also extremely relevant, and measures should be taken to preserve the interspinous and supraspinous ligaments, as the supra-adjacent facets and their capsules at the upper end of the instrumentation construct to help mitigate the risk of ASDeg.[38,39]

Decreasing the risk of developing ASD: motion-preserving procedures as an alternative to fusion

Motion-preservation procedures, including lumbar disc arthroplasty and dynamic posterior instrumentation, have been extensively studied with the rationale that they decrease the rigid fixation that leads to adjacent level intensification of stress forces.[40]

In multilevel lumbar degenerative cases, interspinous process devices were implemented as a surgical option that would allow the creation of a dynamic transition zone, proximal to the fusion, known as topping-off technique, to reduce the occurrence of ASDeg and ASD.[41–43] Putzier et al[42] reported a significant decrease in the incidence of ASD in the topping-off group versus the fusion group (9% vs. 24%, respectively). Lu et al,[41] in a study that analysed the role of proximal intervertebral assisted motion device implantation in reduction of ASDeg after multilevel posterior lumbar interbody fusion, revealed a 11.1% increase in ASDeg in the group undergoing fusion compared to the group undergoing the topping-off procedure. Many authors have tried to develop new materials suitable for dynamic lumbar fusions. The rigidity of these constructs is affected by the choice of material and design of the rods connecting pedicle screws. Semi-rigid fusions can be referred to as flexible fusions, dynamic fusions, or soft stabilization.[44] There are other devices that fall into this category, such as the Graf ligament (SEM Co., Mountrouge, France), the Isobar TTL (Scient'x Alphatec Spine, Bretonneux, France), the DYNESYS (Zimmer Spine, Minneapolis, USA), and many others. However, the optimal mechanical properties of a semi-rigid fixation system have not been determined.[45]

Total disc replacement (TDR) is also a therapeutic option to be taken into account, especially in patients with discogenic low back pain due to single-level degenerative disc disease. This technique allows a functional and clinical improvement while preserving segmental motion, restoring spinal stability and, in contrast to fusion, reducing the incidence of radiographic degeneration of adjacent segments.[46] In a systematic review, Harrop et al[10] compared lumbar arthrodesis to arthroplasty, reporting incidences of ASDeg of 34% vs. 9% and ASD of 14% vs. 1%, respectively.

Other studies[47,48] concluded that, although motion-preserving procedures were associated with a lower ASDeg incidence when compared to fusion, these results did not correlate with different clinical outcomes (Visual Analogue Scale and Oswestry Disability Index scores) or with the potential for developing ASD.

Even though the existing evidence proves that motion-preservation procedures could reduce the prevalence of ASDeg, ASD and reoperation on the adjacent segment, they are also associated with a higher percentage of complications, such as internal fixation loosening and spinous process fracture.[49] Thus, correct patient selection is extremely important when deciding to proceed with this type of technique. On the other hand, most comparative studies between fusion and arthroplasty compare groups with individual differences and with different surgical indications, making it difficult to draw serious conclusions from the current evidence.

The influence of fusion length in the development of ASDeg and ASD

When considering the amount of instrumented levels and the risk of developing ASDeg and ASD, study results are conflicting, with the majority considering the size of the fusion as a risk factor for the development of this complication. Aiki et al[50] and Gillet et al[51] regard fusion length as a significant risk factor for the potential development of ASD. In a retrospective series of 912 patients, Sears et al[20] reported an incremental risk of developing clinical ASD in patients with longer fusions compared with those with fewer than three levels fused. In a systematic review with meta-analysis published in 2012 concerning ASDeg and ADS after lumbar fusion for degenerative pathology, Zhang et al[52] reported that fusion length was the most common factor associated with the development of adjacent segment pathology. In contrast, Ghiselli et al,[12] reported an increased risk of developing ASD in patients undergoing a single-level fusion compared with those having multiple levels fused. Other studies have denied the existence of a clear association between fusion length and accelerated degeneration of the transitional level.[53] However, most publications on this topic support the idea that the length of the fusion is considered a risk factor for the development of ASDeg.[49–52]

Concerning the inclusion of L5-S1 in the fusion, traditionally, many surgeons believed it was a good strategy for pathology at L4-5. In comparison, fusion involving only L4-5 was called floating fusion. Effectively, there have been concerns that accelerated degeneration may occur at L5-S1 after L4-5 fusion, and this may lead to an increase in the incidence of revision surgery. Some studies report that when inclusion of S1 and lumbosacral junction is considered, fusions have a significantly lower risk for ASDeg.[54] This might in part be due to mechanical reasons based on the enhanced loads that act on this segment. However, contrary to these concerns, many studies have reported that L5-S1 is not required to be routinely included in fusion because it is not associated with a clinical benefit, mainly in short fusions.[55–57] Preservation of the L5–S1 motion segment may reduce the sensation of buttock stiffness and prevent the concentration of stress forces only at the cranial adjacent segment and sacroiliac joint.

Proper selection of upper instrumented vertebrae (UIV) is also an important factor as it influences the risk of developing ASD, primarily when planning procedures in cases of spinal deformity. Currently, there is no clear evidence to suggest an ideal UIV. When planning longer fusions to treat deformity pathology, proper UIV selection must be tailored to the individual patient's characteristics. In general, the UIV should be localized in a neutral and stable vertebra and avoid termination at apical segments of coronal or sagittal curves. Although some evidence suggests fusions to the thoracolumbar junction may lead to development of ASD at a greater rate than upper thoracic UIV, the additional cost, decreased flexibility, higher complication rate, and higher pseudarthrosis rate associated with fusions to the upper thoracic spine make it a poor routine preventive strategy.[58]

The influence of sagittal alignment

Spinopelvic sagittal alignment has been found to have a significant effect on clinical outcomes after fusion surgery.[59–61] Sagittal alignment describes the ideal and 'normal' alignment in the sagittal plane, resulting from the symbiosis between various organic factors, with pelvic morphology considered to be its foundation. Effectively, pelvic morphology, namely a significantly higher pelvic incidence (PI) and pelvic tilt (PT), lower sacral slop (SS) and lumbar lordosis (LL) and PI-LL mismatch prior and after fusion, are associated with an increased risk of developing ASD.[62]

Concerning LL, previous studies have reported that failure to restore LL after lumbar fusion is a risk factor for ASD.[63–65] Djurasovic et al reported that patients who developed ASD had significantly less lordosis both at the index fusion level and regionally compared to matched controls.[66] Kim et al[8], in a study evaluating ASD after fusion in patients with L4-L5 spondylolisthesis, concluded that maintaining L4-L5 lordosis angle greater than 20° was important for ASD prevention. Postoperative hypolordosis is common following fusion, and may increase biomechanical loads at adjacent segments. In a prospective study by authors of the International Spine Study Group, Schwab et al described an association between PI-LL mismatch and an increased disability and lower quality of life scores in spinal deformity patients. The authors concluded that PI-LL mismatch should be restored in adult spinal deformity patients, defining a PI-LL mismatch of $\geq$ 11° as being unbalanced.[66] Senteler et al, in a biomechanical study, found that a PI-LL mismatch greater than 15° was predictive of an increased joint load in the unfused and fused lumbar spine adjacent segment and resulted in an increased incidence of revision surgery for ASD after lumbar fusion.[67–69]

The restoration of the normal relationship between LL and PI seems to be essential for postoperative outcomes. Therefore, patients undergoing lumbar fusion with a higher PI may be more likely to develop ASD because of the increased PI-LL mismatch following failure to increase LL. As a compensatory mechanism, patients with fixed sagittal malalignment have an increased PT or pelvic retroversion during standing. After fusion, the inability to correct an increased PT is associated with a higher predisposition to develop ASD, suggesting that sagittal alignment was not optimally corrected.[70]

Given the association between spinal alignment and the development of ASD, spine surgeons should address spinal alignment in patients undergoing surgery for lumbar degenerative disease. Effectively, appropriate correction of sagittal alignment parameters during lumbar spine surgical procedures, particularly the maintenance of the normal spinopelvic relationship, is essential for the prevention of ASD after interbody fusion.

The natural history of the fused spine and individual factors related to ASD

The majority of studies analysing ASD focus mainly on the motion segments immediately above and below the fusion. Little or no attention has been paid to the effects of lumbosacral fusion on non-adjacent mobile segments and the natural degeneration of the remaining lumbar segments with time. Prospective magnetic resonance imaging (MRI) evaluation has shown that disc degeneration following lumbar fusion is not limited to the adjacent segment and could be better explained by individual characteristics than by the fusion itself.[71] Pellisé et al,[72] in a study evaluating the degenerative changes of all unfused lumbar segments 7.5 years after instrumented lumbar fusion, described the loss of disc height as being identical at all the unfused segments located above the fusion, not dependent on fusion parameters (lordosis, length, level) and with a weak correlation with age ($P = 0.045$) and length of follow-up ($P = 0.049$). Schlegel et al[73] and Hambly et al[74] also concluded that the segment next to the adjacent segment is almost as likely to degenerate as the adjacent segment.

Effectively, ASD is a problem with well-studied causal effect but with poorly understood risk factors. Results of exposure-discordant monozygotic and classic twin studies suggest that mechanical factors could play a very limited role and that heredity is a dominant part in disc degeneration, explaining 74% of the variance in adult populations.[78] Certain patient factors such as age, gender, obesity, pre-existing degeneration, osteoporosis, postmenopausal state, rheumatoid arthritis and facet tropism may also contribute to adjacent segment degeneration.[76] Genetic influences, such as polymorphisms of the vitamin D receptor and collagen IX genes, can also be a potential cause of disc degeneration with consequent deterioration of the motion segment.[77]

Authors' preferred treatment

The potential development of ASD should always be considered during surgical decisions to treat lumbar spine pathology. Several risk factors associated with surgery (e.g. the chosen surgical approach, anatomical dissection, the choice of interbody fusion, the increment of fusion, the fusion length and the restoration of sagittal alignment) may influence the development of ASD. All the variables previously described must be taken into account during the preoperative planning and surgical act in order to decrease the likelihood of developing ASD.

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