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Adjacent Segment Pathology in Spinal Fusion Surgery

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Study Design: Literature review
Objectives: To present up-to-date evidence on adjacent segment pathology (ASP) in spinal fusion surgery
Summary of Literature Review: Several prior studies have been conducted on the definition, pathology, etiology, risk factors, and treatment of ASP in spinal fusion surgery.
Materials and Methods: Review of the associated literature and latest research.
Results: ASP shows various pathologies, including disc degeneration, spondylolisthesis, and instability. Important risk factors are patient-related factors such as high body mass index and a pre-existing degenerated disc at the adjacent level, and surgical-related factors such as facet joint violation due to pedicle screws and changes in sagittal alignment before and after surgery. ASP often cannot be prevented because it is part of the natural history of degeneration. However, to reduce the occurrence after initial surgery, the surgeon should try to reconstruct the spine in a way that maintains balance and avoid injuring adjacent disc, facet joints.
Conclusions: ASP should be accurately defined and its pathology and etiology should be accurately identified. Risk factors should also be recognized and avoided during spinal fusion surgery.

Key words: Adjacent segment pathology, Adjacent segment degeneration, Adjacent segment disease, Proximal junctional kyphosis, Proximal junctional failure

Introduction and Definition

Adjacent segment disease and adjacent segment degeneration are terms generally used for degenerative changes at an immediate adjacent segment after spinal fusion surgery. Adjacent segment degeneration is just radiologic term, and accompanying symptoms do not necessitate. On the other hand, adjacent segment disease is always a symptomatic term. To prevent confusion between the two terms, adjacent segment pathology (ASP) is generally used nowadays. ASP can be divided into radiologic ASP (RASP) and clinical ASP (CASP). RASP refers to adjacent segment degeneration and CASP refers to adjacent segment disease.[1] In addition, it is also important how far the adjacent segment means in the definition of ASP. In general, it refers only to the immediate adjacent segment of the fusion site. However, some studies included adjacent two segments.[2-4] Although there is a report that degeneration is more severe in the first adjacent segment than in the second adjacent segment, some researchers claim that the second segment shows a similar disc height reduction.[5-7] Therefore,

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these considerations must be considered when defining adjacent segments around the fusion site.

Pathology

Various pathologies seen in the ASP are mainly based on radiologic components. Typically, nine pathologies are shown: disc degeneration, spondylolisthesis, instability, herniated nucleus pulposus (HNP), stenosis, hypertrophic facet arthritis, osteophyte formation, scoliosis, vertebral compression fracture. The most common finding is disc degeneration of an immediate adjacent segment.[6,8–10] Also, spondylolisthesis, instability, herniated nucleus pulposus, stenosis, and hypertrophic facet arthritis are commonly reported.[3,5,11–13] Less commonly reported findings are scoliosis and vertebral compression fractures.[12] Most pathologies can be confirmed by plain radiographs, Magnetic Resonance Imaging (MRI), Computed Tomography (CT), and myelogram. Among these, the criterion of instability has been varied in many studies. The most common criterion is dynamic sagittal translation >3 to 4 mm and/or angle change greater than 10 to 15° between adjacent vertebral bodies.[3,14] By integrating these pathologies, various classifications such as the University of California Los Angeles (UCLA) grading system or Weiner classification, which have been widely used in the past, are used to define ASP (Table 1).[15]

Incidence

Although the incidence of ASP has been reported in many studies, there are differences in patient group, methodology, and follow-up time. Thus, incidence rates are hard to define in the relevant studies. Radiologic ASP reports from 5.6% to 100% depending on the literature, clinical ASP from 2.7 to 21.4%, and for ASP-related reoperation, it is reported from 4.0 to 27.8%. According to two meta-analyses, incidence rates for RASP were 37.5%, 26.5% each, 14.4%, 8.5 for CASP, 7.7% for reoperation.[16,17] This wide range is because many studies on incidence rates that have been conducted so far use a retrospective and differing methodology, show differences in patient groups, and have different definitions of ASP. In many studies, when defining ASP, it was defined as a radiographic finding rather than the patient's symptoms. Radiographic disc degeneration, stenosis, HNP, spondylolisthesis, or instability at the adjacent level was considered ASP. In this case rates ranged from 8 to 100%.[5–11,13,18–21] On the other hand, when only the symptomatic ASP, that is, the CASP, was 5.2 to 18.5%.[19,21,22]

Table 1. University of California at Los Angeles (UCLA) grading system for intervertebral space degeneration

Grade	Disc-space narrowing	Osteophytes	End plate sclerosis
I	-	-	-
II	+	-	-
III	±	+	-
IV	±	±	+

Etiology

1) Biomechanical overstress of the adjacent cephalad segment after fusion surgery plays a major role in ASP development. In fusion surgery, the center of rotation is shifted cephalad to increase facet loading and intradiscal pressure of the adjacent mobile segment. An increase in facet loading and intradiscal pressure induce fact arthritis and disc degeneration, which are representative pathologies of ASP.[23] It is also known that in cellular level increased force on the intervertebral disc affects the biochemical milieu.[24] It modulates specific cytokine levels and consequently triggers the inflammatory cascade responsible for osteoarthritis. Interleukin-1b and tumor necrosis factor-a levels rise when disc compression increases, causing the release of proteoglycan and catabolic enzymes.[24] In addition, mechanical disc compression inhibits oxygen diffusion and accelerates degeneration.[25] These additional force on adjacent level, cytokine release, hypoxia, and disc degeneration accelerate ASP.[26] 2) Evidence has also been reported that postoperative sagittal alignment increases the incidence of ASP in the future. There are biomechanical data showing that the shear force at the adjacent level increases by 29% in the presence of hypolordosis after lumbar segment fusion. Also, a significantly lower sacral slope was observed in patients with ASP, which indirectly means sagittal malalignment after fusion.[27] 3) Relative postoperative kyphosis after cervical arthrodesis also increases the incidence of ASP.[28] In addition, 4) genetic predispositions such as the carbohydrate sulfotransferase 3 variant susceptible to degenerative disc disease can also identify

an at-risk population in the development of subsequent ASP as well as primary degenerative disc disease.[29] The above four etiologies are the direct causes of ASP, the last factor to be discussed is the pre-existing advanced degeneration at the adjacent segment at the time of fusion surgery. As most fusion surgeries are performed in severe degenerative conditions, these changes are not limited to fused segments. Several studies have reported that there is no significant difference in the occurrence of ASP between the fusion group and the non-fused surgery or conservative treatment group.[5] They argue that it is due to age-dependent degeneration rather than the fusion surgery itself that led to ASP.[10]

As such, ASP is a degenerative change by multifactorial etiology. Adjacent level biomechanical change after fusion surgery increases the pressure and strain of the segment, and in this regard, alteration of the biochemical milieu accelerates the inflammatory cascade. In addition, changes in sagittal alignment after surgery also cause biomechanical changes at the adjacent level. Genetic predisposition, which is vulnerable to disc degeneration, also causes disc degeneration at the adjacent level. ASP can be explained by these four directly related etiology, and in addition, degeneration itself, which is already in progress at the time of fusion surgery, is also related to the occurrence of ASP.

ASP can be according to the progressive disease course, or it can be a product of iatrogenic fusion. The controversy isn't so much whether this happens or not, but how much of it is just the natural history and how much of it is the surgery itself. The above 4 etiologies support a link between ASP and fusion surgery, but, preexisting degenerative disease, natural history of degeneration, and some biomechanical studies that suggest no increase of stress at adjacent segment under load control do not support it. The extent to which natural history of progressive disease or biomechanical alterations due to iatrogenic fusion contributes to ASP remains unclear, although it is reasonable to assume that both factors play a role.

Risk Factors

Although most studies regarding risk factors of ASP have been done retrospective, many studies have been conducted to identify risk factors to reduce the incidence of ASP. These risk factors can be divided into patient-related factors and surgical factors (Table 2).

Patient-related Factors

Several patient characteristics influence the occurrence of ASP. The most important risk factor is the patient's age at the time of primary surgery. Aota et al showed that the incidence of ASP was much higher in patients over 55 years of age.[18] This seems to be a result of the decrease in the ability of the spine to adapt to the biomechanical change caused by fusion as the age increases. Lee et al compared survivorship of adjacent segments 10 years after fusion surgery and showed a significant difference at 78% for those over 60 and 93% for those under 60.[2] Also, female, postmenopausal status, and osteoporosis are risk factors for ASP, which seem to be related to each other.[12,18] High BMI is also a demographic risk factor for ASP in several studies. For these adjustable risk factors, setting the BMI cutoff and making a weight management protocol can reduce the occurrence of ASP.[30] Pre-existing degenerated disc

Table 2. Risk factors for adjacent spinal pathology

Patient-related factors	Surgical factors
- Age	- Instrumentation
- Female gender	- PLIF
- High BMI	- Injury to the facet joint of the adjacent segment
- Pre-existing degenerated disc at the adjacent level	- Fusion length
- Osteoporosis	- Sagittal alignment
- Pre-existing lumbar stenosis at the adjacent segment	
- Rheumatoid arthritis	
- Post-menopausal state	
- Facet tropism and degeneration	
- Genetic influences: polymorphisms of the vitamin D receptor and collagen IX genes	

and lumbar stenosis in the adjacent segment are also important risk factors.[20] This is based on the assumption that an already degenerated disc is more likely to deteriorate.[20] Guigui et al found a significantly higher incidence in the pre-existing lumbar stenosis group in the retrospective study of 102 patients who underwent posterolateral fusion. Conversely, some studies suggest that these pre-existing conditions are not significantly associated with the development of ASP.[2,4,31,32]

At last, facet degeneration at the time of primary surgery and tropism of adjacent segment are the important risk factors. Lee et al reported that pre-existing facet degeneration was the only risk factor in 2.62% of 1069 ASP patients who required revision surgery.[2] Also, if patients have facet tropism, joints are faced different move with great stress. Okuda et al [32] reported that the occurrence of ASP was high when this facet tropism was at the adjacent level. On the other hand, there is also opposite contradicting report that facet tropism at the adjacent segment was not related to ASP.[31]

Surgical Factors

The addition of instrumentation in fusion surgery causes early development of ASP. The interval of occurrence of ASP is shortened upon instrumentation. Adding instrumentation induces more stress by giving immediate rigidity and accelerates degeneration of adjacent segments.[12] With the same logic, adding posterior lumbar interbody fusion (PLIF) to instrumentation also increases ASP by adding rigidity.[21] However, recently, instrumentation is almost always involved in fusion surgery to increase the fusion rate, so clinical significance for this issue has decreased.[33] Another risk factor is facet joint violation that may occur during insertion of the superior pedicle screw. This refers to damage to the inferior facet of the adjacent segment according to the entry site.[18] This contributes to the occurrence of ASP by affecting the load-bearing capability of the facet. In addition, the disruption of posterior elements during dissection or surgery also changes biomechanics and potentially predispose to ASP. Lai et al found that sacrifice of the posterior elements from spinal fusion to adjacent segments increased the risk of adjacent segment instability up to 3 times. The number of segments fused also affects the occurrence of ASP. The longer the fusion length, the longer the lever arm, increasing the stress on the remaining non-fused segment.[23] Postoperative abnormal sagittal alignment is also important in the development of ASP.[14] Kumar et al reported that the incidence of ASP increases when there is a postoperative change in the C7 plumb line and sacral inclination, and the lowest incidence of ASP in normal sagittal alignment.[22]

Proximal Junctional Kyphosis and Proximal Junctional Failure

Proximal junctional kyphosis (PJK) is a complication that may require reoperation after fusion surgery for adult spinal deformity especially after multilevel fusion. It presents development of kyphosis at the transition between fused and mobile motion segments. This is one specific form of ASP. It is defined by the presence of two criteria: (1) a proximal junction sagittal Cobb angle of ≥ 10° and (2) a postoperative proximal junction sagittal Cobb angle at least 10° greater than the measurement preoperatively. This is defined as the Cobb angle between the inferior end plate of the upper instrumented vertebra (UIV) and the superior end plate of the vertebra two levels above.[34] It is characteristic that these diagnostic criteria are defined only by serial angle change in plain radiograph compared to various radiologic components defining the ASP. PJK, like ASP, is multifactorial and is associated with several patient-related and surgical risk factors. The diagnosis of PJK is like that of ASP, and it is not symptomatic in all cases, and it presents a broad spectrum of disease from asymptomatic patients to symptomatic who show increased pain, functional deficit, and neurologic deficits.

Proximal junctional failure (PJF) is the most severe form of this spectrum, showing progressive worsening and structural failure of the vertebral body and/or posterior discoligamentous complex. In other words, if the PJK satisfies at least one of the following conditions, we call this PJF: (1) fracture of the vertebral body of UIV or UIV+1, (2) pullout of instrumentation at the UIV, (3) adjacent vertebral subluxation, (4) neurological deficits related to the PJK, or (5) revision surgery requiring extension of the proximal fusion within the first 6 months of the index procedure.[34]

Treatment Options

Radiographic diagnosis of ASP does not always present

a poor prognosis.[8,20] Surgical treatment is not always necessary in patients with ASP, and it may be considered if there are symptoms that can be explained by the pathology corresponding to the adjacent segment after union in the patient for which conservative treatment has failed. In the study to date, there is no known superior surgical method for the ASP. However, the considerations to be taken for successful surgical treatment of ASP is as follows.

(1) Which anatomical structures require surgical decompression (2) Is the first fusion done properly, that is, whether there is a pseudoarthrosis (3) What is sagittal alignment like. Neural structures that are compressed due to the pathology of the ASP should be accurately reviewed radiologically and decompressed thoroughly. Representative pathologies include herniated nucleus pulposus, hypertrophic facet arthritis, and osteophyte formation. If fusion is not performed properly and pseudoarthrosis is suspected, the pre-operative CT scan should be performed to accurately evaluate the existing fusion state, and it is important to accurately check areas requiring revision fusion other than adjacent segments. However, Daniel et al preferred to treat most cases of ASP by decompression with fusion even in the absence of spondylolisthesis or instability.[35] On the other hand, based on numerous retrospective studies involving different patients and methodologies, the necessity of extending the fusion is uncertain.[3,14,36] In ASP patients with sagittal deformity like hypolordosis or lumbar kyphosis interbody fusion or corrective osteotomies may be useful techniques to improve sagittal plane alignment in addition to decompression. Considering decompression, fusion, and sagittal alignment, indirect decompression and restoration of disk height through lateral interbody fusion techniques has also been suggested as a treatment for ASP. There are still many controversies about the effectiveness of these surgical treatments. Nevertheless, it was particularly effective in alleviating leg symptoms in patients with symptomatic stenosis of adjacent segments.[36] In addition, persistent and severe postoperative back pain was the only significant predictor of subsequent poor outcome.[36] Although it is not a treatment for ASP itself that has already occurred, several methods have been proposed to avoid it by analyzing the pathology and etiology of ASP. Changing the biomechanical profile through a newly implanted device and minimizing adjacent level soft tissue disruption are those.[37,38] These can be achieved by the new technologies of arthroplasty, dynamic fixation, and percutaneous fixation. Still, the early results for these techniques have been controversial.[37,39]

Conclusions

Adjacent segment pathology is a common complication after spinal fusion surgery. It should be accurately defined and their pathology and etiology should be accurately identified. Also, risk factors should be recognized and avoided to avoid the progression of ASP during initial spinal fusion surgery.

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Review Article

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척추유합술에서의 인접분절병변

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연구계획: 문헌 고찰

목적: 척추유합술에서의 인접분절병변에 대한 최신 지견 소개

선행 연구문헌의 요약: 척추유합술에서의 인접분절병변에 대한 정의, 병리, 원인, 위험인자, 치료 등에 대한 여러 선행연구가 알려져 있다.

대상 및 방법: 문헌 고찰 및 최신 연구의 소개

결과: 인접분절병변을 추간판변성, 척추체전위, 불안정성을 포함한 다양한 병리를 보인다. 중요한 위험인자로, 높은 체질량지수, 수술 전 인접분절 추간판변성 등의 환자 관련 인자가 있으며, 척추경 나사로 인한 후관절 손상, 수술 전후 시상정렬의 변화 등의 수술관련 인자가 있다. 인접분절병변은 자연적인 퇴행성 변화의 일부로서 예방할 수 없는 경우가 많지만, 초기수술에서의 발생을 줄이기 위해 인접 추간판, 후관절의 손상에 유의하고 수술 후 시상면 균형을 회복시켜야 한다.

결론: 인접분절병변은 퇴행성변화의 일부로 여러 형태의 병리, 원인이 알려져 있다. 이에 미치는 여러 위험인자를 인식하고 피해야 한다. 특히 인접분절병변의 발생을 줄이기 위해 수술 중 인접분절 추간판과 후관절을 손상시키지 않도록 주의해야 하며, 수술 후 시상면 균형을 회복시켜야 한다.

색인 단어: 인접분절병변, 인접분절퇴행성변화, 인접분절질환, 상위이행부후만증, 상위이행부실패

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