

Review Article

Adjacent segment disease in the lumbar spine following different treatment interventions

Kristen E. Radcliff, MD^{a,*}, Christopher K. Kepler, MD^a, Andre Jakoi, MD^b,
Gursukhman S. Sidhu, MBBS^a, Jeffrey Rihn, MD^a, Alexander R. Vaccaro, MD, PhD^a,
Todd J. Albert, MD^a, Alan S. Hilibrand, MD^a

^aDepartment of Orthopedic Surgery, Rothman Institute, Thomas Jefferson University, Philadelphia, PA 19107, USA

^bDepartment of Orthopedic Surgery, Drexel University, Philadelphia, PA, USA

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Abstract

BACKGROUND CONTEXT: Adjacent segment disease (ASD) is symptomatic deterioration of spinal levels adjacent to the site of a previous fusion. A critical issue related to ASD is whether deterioration of spinal segments adjacent to a fusion is due to the spinal intervention or due to the natural history of spinal degenerative disease.

PURPOSE: The purpose of this review is to summarize the recent clinical literature on adjacent segment disease in light of the natural history, patient-modifiable risk factors, surgical risk factors, sagittal balance, and new technology.

STUDY DESIGN: This review will evaluate the recent literature on genetic and hereditary components of spinal degenerative disease and potential links to the development of ASD.

METHODS: After a meticulous search of Medline for relevant articles pertaining to our review, we summarized the recent literature on the rate of ASD and the effect of various interventions, including motion preservation, sagittal imbalance, arthroplasty, and minimally invasive surgery.

RESULTS: The reported rate of ASD after decompression and stabilization procedures is approximately 2% to 3% per year. The factors that are consistently associated with adjacent segment disease include laminectomy adjacent to a fusion and a sagittal imbalance.

FDA device/drug status: Not applicable.

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* Corresponding author. 2500 English Creek Ave., Egg Harbor Township, NJ 08422, USA. Tel.: (609) 573-3301; fax: (215) 955-4322.

E-mail address: radcliffk@gmail.com (K.E. Radcliff)

CONCLUSIONS: Spinal surgical interventions have been associated with ASD. However, whether such interventions may lead to an acceleration of the natural history of the disease remains questionable. © 2013 Elsevier Inc. All rights reserved.

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Introduction

The natural history of lumbar degenerative disease includes gradual desiccation of all lumbar disks although the rate of degeneration is not uniform across spinal segments. Degeneration can occur even in asymptomatic individuals and is more common at lower lumbar levels. The most commonly degenerated level is L5-S1, which constituted 50% of herniations in asymptomatic individuals, L4/5 in 35% of individuals, followed by L3/4 in 15% of individuals [1]. The concept of adjacent segment disease is based in the hypothesis that specific spine interventions increase the likelihood of spinal degeneration relative to the natural rate. Confounding studies of ASD is the observation in natural history studies that disk degeneration progresses even in 41% of asymptomatic individuals [2]. The interest of the spine community lies in identification of patient and surgical factors that have an effect on the rate of adjacent segment disease (ASD). The purpose of this review article is to evaluate current, recent literature on lumbar ASD in light of recent information on the natural history of spinal degeneration, patient-modifiable risk factors, surgical risk factors, sagittal balance, and new technology.

Adjacent segment disease: a disease entity?

ASD is a clinical phenomenon that has been defined, for the purposes of this review, as the presentation of new symptoms referable to an adjacent level after patients have undergone successful surgical treatment of a spinal problem at an index level [3]. To distinguish clinically significant disease from minor symptoms, the definition also suggests that patients should seek or undergo treatment for the new symptoms and that the new symptoms should be present on at least two occasions. ASD does not classically include axial pain, muscle spasms, or numbness that are sequelae of the index arthrodesis. Adjacent segment disease should also be distinguished from adjacent segment degeneration (ASDeg), which is defined for the purpose of this review as asymptomatic radiographic deterioration of segments adjacent to a lumbar arthrodesis. To the authors knowledge, there is no universally accepted, validated outcome instrument to diagnose or quantify ASD [4–8].

Possible etiological risk factors

There are several hypotheses to explain adjacent segment disease. The initial description of ASD attributed the phenomenon to increased biomechanical demands upon other spinal segments after an arthrodesis [9]. In addition to

a net loss of mobile segments, there may be an increased force lever arm transmitted by the fused segments to adjacent, nonfused spinal segments. Other mechanical explanations include the theory that some procedures may promote spinal instability by removing bone and ligamentous structures and therefore create accelerated degeneration at other spinal segments [10]. Recently, some have theorized that open surgical dissection may induce ASD due to increased damage to paraspinal muscles and ligaments relative to percutaneous approaches [11–14]. However, there has not been definitive proof of reduction in incidence of ASD with percutaneous versus open approaches and therefore new technologies continue to be developed to reduce iatrogenic ASD due to surgical approach.

The fundamental question is whether ASD represents the natural history of spinal degenerative disease progressing in individuals who are symptomatic at one level or is the result of a particular intervention due to altered mechanics. There is evidence of a genetic predisposition to spinal degenerative disease in some individuals. If spinal degenerative disease were genetically predetermined, that would imply that deterioration at adjacent spinal segments is the natural result of a biological cascade. Evidence in favor of a biological etiology of adjacent segment disease includes observations from several population studies. Twin studies comparing patients in dissimilar occupations have attributed 26% to 72% of the variability in the incidence of lumbar degeneration, particularly in upper lumbar segments, to genetic influences and not to physical exposures [14]. Other studies have confirmed excessive relatedness of affected individuals with degenerative disc disease [15]. As evidence of the systemic nature of spinal degenerative disease, patients with lumbar vertebral herniations also commonly have cervical degenerative changes [16].

Demographic risk factors

Although patient characteristics, including obesity, age, duration of symptoms, medical comorbidities, mental illness, and compensation status have been shown to affect outcome of lumbar spinal surgery, there are no similar accepted patient risk factors for ASD (Table 1) [17–19]. Smoking has been proposed as a risk factor for adjacent segment degeneration [19] in some studies and found to be nonsignificant in others [20]. Patient age may be a significant risk factor. Some studies have determined that older age is associated with an increased risk of ASD [21,22] after arthrodesis and after fusion [23]. A small series found no effect of older age, body mass index, gender, or

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