Lumbar disc degeneration below a long arthrodesis (performed for scoliosis in adults) to L4 or L5

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Abstract A retrospective analysis of adults treated with long instrumented fusion for scoliosis from the thoracic spine proximally to L4 or L5. To evaluate the long-term clinical outcomes as well as radiological changes in distal unfused mobile segments and to evaluate factors that may predispose to distal disc degeneration and/or poor outcome. A total of 151 mobile segments in 85 patients (65 female), mean age 43.2 (range 21–68), were studied. Curve type, number of fused levels and pelvic incidence were recorded. Clinical outcome was measured using the Whitecloud function scale and disc degeneration using the UCLA disc degeneration score. Spinal balance, local segmental angulations and lumbar lordosis were measured pre- and post-operatively as well as at the most recent follow up—mean 9.3 years (range 7–19). A total of 62% of patients had a good or excellent outcome. Eleven had a poor outcome of which ten underwent extension of fusion—five for pain alone, three for pain with stenosis and two for pseudarthroses. Pre-operative disc degeneration was often asymmetric and was slightly greater in older patients. Overall, there was a significant deterioration in disc degeneration ($P < 0.0001$) that did not correlate with clinical outcome. Disc degeneration correlated with the recent sagittal balance (Anova $F = 14.285$, $P < 0.001$) and the most recent lordosis (Anova $F = 4.057$, $P = 0.048$). The post-operative sagittal balance and local L5-S1 sagittal angulation correlated to L4 and L5 degeneration,

respectively. There was no correlation between degeneration and age, pre-operative degenerative score, pelvic incidence, sacral slope, number of fused levels or distal level of fusion. Disc degeneration does occur below an arthrodesis for scoliosis in adults which does not correlate with clinical outcome. The correlation of loss of sagittal balance with disc degeneration may be as a result of degeneration causing the loss of balance or vice versa, i.e. sagittal imbalance causing degeneration. Immediate post-operative imbalance correlates with degeneration of the L4/5 disc, which may imply the latter.

Keywords Disc degeneration · Scoliosis · Adults

Introduction

The prevalence of thoracolumbar spinal fusion has continued to increase since its first description nearly 100 years ago [1] and is now undertaken for many spinal conditions including scoliosis and degenerative lumbar disease [10]. Adjacent segment disease (ASD) was first noted as a relatively unusual finding after lumbar or lumbosacral fusion [2] but it is not as uncommon as first thought with the reported prevalence of ASD being 5–43% [4, 7, 18].

Many clinical and biomechanical studies have analyzed problems at the adjacent motion segments following fusion, predominantly focusing on segments cephalad to a short lumbar fusion. There are, however, relatively few studies that examine the motion segment distal to a short fusion. Miyakoshi et al. [16] have shown a reduction in disc height adjacent and distal to an L4/5 posterior lumbar interbody fusion that does not correlate to clinical outcome. In fusions performed for scoliosis, a study using Harrington

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instrumentation in adolescents noted radiological disc degeneration distal to the fusion to be uncommon and also stated that this had no correlation to clinical outcome [15]. In a further study using modern multi-segmental instrumentation, degeneration distal to a long fusion was independent of the distal level fused or age at time of surgery [21]. The same group describe 16% degeneration is discs distal to a long fusion for scoliosis in adults but, again, that this had no correlation to clinical outcome [5].

Adjacent segment degeneration has been explained by biomechanical studies, which have shown increased stress on facet joints adjacent to a fusion [13] and a change in the loading characteristics of a disc distal to a fusion [19]. In addition, hypermobility at adjacent segments has been shown using radiostereometric analysis [4]. Ghiselli et al. [9] hypothesized a multi-level fusion would therefore lead to more adjacent segment degeneration, but their study found this not to be the case: Patients having a single level fusion were at greater risk of developing ASD which was in itself hypothesized to be due to an increased number of levels at risk. Conversely, many other studies have shown an increased risk of ASD with a polysegmental fusion [6, 20, 26].

Other potential risk factors include age, sagittal alignment and the presence of instrumentation. Most studies have shown age to be an important factor in the development of ASD [3, 6, 11, 21]. However, there is limited information on the role of sagittal or coronal malalignment in the development of ASD: In a cadaveric study hypolordosis was shown to adversely affect loading characteristics of the posterior column [24]. In vivo, a single study evaluating the role of sagittal balance and morphology in ASD following lumbar spine fusion showed that patients in good sagittal balance exhibited less ASD and that a normal sacral inclination was a risk factor in its development [11]. A further study in sheep showed that a local kyphotic deformity at L3-5 increased the risk of cranial disc degeneration [17]. Instrumented fusions appear to reduce the interval of occurrence of ASD compared to non-instrumented fusions. Aota et al. [3] describe radiographic instability 25 months after transpedicular fixation and Etebar and Cahill [6] describe an average of 26.8 months prior to symptomatic ASD after instrumented fusion. Conversely studies of non-instrumented fusions describe 8.5 and 13.1 years intervals before the development of ASD [12, 22]. The risk of ASD is further increased by PLIF [20] suggesting that increased rigidity of fusion may be a risk factor.

The aim of this study is to define the presence, progression and risk factors for disc degeneration distal to a long fusion for scoliosis in adults using multi-segmental instrumentation systems. Although we aim to evaluate some of the risk factors already discussed above, to our

knowledge, this is the first study that has attempted to evaluate the role of local segmental angulations as well as overall sagittal and coronal balance in the development of distal ASD below an arthrodesis.

Methods

This study is a retrospective review of 101 patients who underwent Cotrel-Dubousset or Colorado spinal instrumentation for scoliosis (using hybrid screws and hooks construct) between 1985 and 1994 at our institution. Inclusion criteria were age greater than 20 at the time of surgery, a follow up greater than 5 years, fusion to L4 or L5 (leaving 1 or 2 mobile segments) and idiopathic or degenerative curves. Patients with a history of previous surgery at the mobile segment or an incomplete set of radiographs were excluded.

Patient age, sex, curve type, number of fused levels fused and number of years follow up were recorded. Full length anteroposterior (AP) and lateral films as well as views centred on the lumbar mobile segments were used to evaluate radiographs pre-operatively, post-operatively and at the most recent follow up. Measurements (performed by a single observer) included pelvic incidence, sacral slope, lumbar lordosis (L1 superior endplate to S1 superior endplate), L4-S1 and L5-S1 angulations in both the sagittal and frontal planes, overall frontal/sagittal balance and disc degeneration. ASD was measured using the UCLA disc degeneration score [8] outlined in Table 1. In the case of asymmetric degeneration the worst score was taken. At the most recent follow up, the presence or absence of low back pain was noted and functional outcome was measured using the Whitecloud function scale [25] as outline in Table 2.

Statistical analysis was performed using SPSS software (SPSS, Chicago, IL, USA). A paired *t* test was used to evaluate the change in degeneration in individual discs. Analysis of variance between groups was used to analyse potential clinical and radiological correlations with disc degeneration whereas the Pearson Chi-square was used to measure any correlation between functional outcome and degeneration. When considering disc degeneration we

Table 1 University of California at LA grading scale for disc degeneration

Grade	Disc narrowing	Osteophytes	End plate sclerosis
1	—	—	—
2	+	—	—
3	+/-	+	—
4	+/-	+/-	+

Table 2 Modified function scale (Whitecloud)

Outcome	Pain	Medication	Activity	Work status
Excellent	None, occ back pain	None	Normal	Normal
Good	Markedly improved, occ back pain	Occ	Minimal limitation	Return, but different activity
Fair	Some improvement	Frequent	Restricted	Limited
Poor	No change or worse	Narcotics	Incapacitated	Disabled

considered those patients that did not have a change in their score to be in the “no degeneration” group. In all cases, a P value < 0.05 was considered as significant.

Results

Eighty-five patients (65 females) fulfilled our inclusion and exclusion criteria with a mean age of 43.2 years (range 21–68). The mean follow up period was 9.3 years (range 7–19). Nineteen patients underwent fusion to L5 whereas 66 underwent fusion to L4 leaving 151 mobile segments for analysis.

Table 3 shows mean disc degeneration scores for each disc level and overall, pre- and post-operatively. Disc degeneration (i.e. an increase of at least 1 in UCLA score) occurred in 98 discs but in 53 discs (17 at L4/5 and 36 at L5/S1) there was no change in the score. The majority of these discs were normal (score 1) at the time of fusion, but in eight cases the discs had reduced height (score 2) and did not progress. Interestingly, in two cases where fusion was performed to L5 with a markedly degenerate disc below (sclerosis/osteophytes), the patients did well with no low back pain and did not require extension of fusion. No fractures of olisthesis were seen distal to the fusion and there was no correlation between age, number of levels fused or pre-operative grade and subsequent degeneration.

When considering all discs together there was a correlation between the sagittal balance at the last follow up and disc degeneration (Anova, $F = 14.285$, $P < 0.001$). The mean sagittal balance in the non-degenerative group was 0.96 and 4.25 cm in the degenerative group. A similar correlation was observed with lumbar lordosis at the most recent follow up (Anova, $F = 4.057$, $P = 0.048$). The mean lumbar lordosis was 48° in the non-degenerative group and 42° in the degenerative group. There was no correlation between degeneration and any of the other radiological

parameters measured including the fixed parameter of pelvic incidence, although significance was almost reached with post-operative sagittal balance (Anova, $F = 3.45$, $P = 0.067$).

Considering the level L4/5 in isolation, the same pattern of correlation is noted, but with an additional correlation to post-operative sagittal balance (Anova, $F = 19.388$, $P < 0.001$, mean 0.94 cm non-degeneration, 2.00 cm degeneration). Furthermore, if one considers L5/S1 in isolation one sees a similar correlation to the most recent sagittal balance and lumbar lordosis plus an additional correlation to the local L5/S1 sagittal angulation post-operatively (Anova, $F = 5.270$, $P = 0.024$, mean 18° non-degeneration, 22° degeneration).

Fifty-three (62%) patients had a good or excellent outcome, whereas 21 (25%) had a fair outcome and 11 (13%) a poor one. Of these 11 patients, 10 underwent revision surgery with extended fusion—5 for pain alone, 3 for pain with stenosis and 2 for pseudarthroses. The extensions performed for pseudarthroses were to correct sagittal malalignment and provide a good biomechanical environment for fusion. No correlation was found between disc degeneration and the presence of back pain (Pearson Chi-square, $R = 0.330$, $P = 0.384$) or clinical outcome (Pearson Chi-square, $R = 0.497$, $P = 0.780$).

Discussion

This study describes the long-term clinical outcome of adult patients undergoing a long fusion for scoliosis down to L4 or L5. We have shown that ASD does occur distal to the fusion and that this correlates to sagittal balance and lumbar lordosis at mean 9.3 years follow up.

The magnitude of disc degeneration we have seen is similar to that seen by Ghiselli et al. [9] who used a similar method of quantitative analysis of disc degeneration in

Table 3 Mean disc degeneration scores pre- and post-operatively

	Mean pre-operative UCLA disc score	Mean post-operative UCLA disc score	P value (paired t test)
L4-L5, $n = 66$	1.25	2.15	<0.001
L5-S1, $n = 85$	1.23	1.91	<0.001
All discs, $n = 151$	1.24	2.01	<0.001

patients undergoing fusion of the lumbar spine with a mean follow up of 6.7 years. Rinella et al. [21] analyzed thoracolumbar fusions to a number of different levels down to L4 with a mean follow up of 7.8 years and we have observed a similar pattern of degeneration in our study although direct comparison is difficult as different scoring and methodology was used. Ghiselli et al. [9] hypothesized that a multi-level fusion would lead to more adjacent segment degeneration, but their study found this not to be the case. In agreement with this, we did not find any correlation between the length of fusion and subsequent degeneration. Furthermore, we agree with the findings of Rinella et al. [21] that age at time of surgery and distal level fused do not have a correlation with degeneration. Again, the studies are not directly comparable as we only consider L4 and L5 as opposed to T12 to L4, but a trend is evident.

An association between poor sagittal and coronal balance of the spine with the development of ASD has been implicated previously [11, 17] and our study supports this. However, the correlation between the loss of sagittal balance and lordosis with disc degeneration at the last follow up does not tell us whether it is the loss of alignment that predisposes to degeneration or vice versa. However, the association between immediate post-operative sagittal alignment and degeneration, when one considers L4/5 and L5/S1 separately, may suggest that this is a contributing factor to degeneration. None of our patients had *gross* sagittal malalignment (greater than 5 cm) post-operatively and so, if sagittal alignment is an important factor then its influence here would be relatively small as we had no patients with significant loss of sagittal balance post-operatively. Indeed, the mean displacement from the C7-S1 plumb line for cases undergoing degeneration was only 2 cm post-operatively and 4 cm at follow up. To further evaluate this one could increase sample size, compare to age matched controls or analyse patients with greater sagittal malalignment post-operatively (which is obviously not a desirable outcome of surgery). A comparison to age matched controls (although difficult to achieve) would evaluate the role of disc degeneration that is expected with time and that which may be due to other factors. Time may in fact be the single most important factor in the development of ASD. Ghiselli et al. [9] using survival analysis suggest an intervention rate for ASD at 3.9% per year from the time of surgery, although ASD can also occur after a lengthy symptom free period [22].

Based on biomechanical studies [4, 13, 19, 24] it is probable that ASD does occur in the presence of unfavorable biomechanical conditions resulting from fusion. However, the mechanisms for this at a cellular level are poorly understood and it may be that other factors such as genotype [23] will prove to be the most important single

factor in disc degeneration and ASD. However, this should not discourage the surgeon from obtaining the ideal mechanical environment to minimize the stresses placed upon an unfused adjacent segment when fusing the spine, i.e. normal sagittal balance, coronal balance and lumbar lordosis. This applies equally to isolated lumbar fusions as well as the longer fusions for deformity.

The prevalence of radiological disc deterioration in our patients was 37.7% with only 9.4% of all patients undergoing revision surgery for ASD which is comparable to other studies [7, 14, 18] and less than that predicted by Ghiselli [9] using Kaplan-Meier survivorship analysis in isolated lumbar fusions. Similarly, 2 out of 26 (7.7%) of patients undergoing fusion to L4 in the Rinella et al. [21] study underwent extension of fusion to L5 for ASD. It could be hypothesized that the early loss of disc height does not cause problems and only when it becomes severe with osteophyte formation and sclerosis does it become a clinical concern. Our results, however, would not support this and did not show any correlation between the presence or absence of back pain and disc degeneration. Furthermore, we have not shown any correlation between clinical outcome and disc degeneration. Although we believe loss of disc height, osteophyte formation and sclerosis are part of the process contributing or caused by ASD, we realize that it is an oversimplification to state that disc degeneration equates to ASD which may be due to other factors such as facet pain and ligament hypertrophy. This may explain the poor correlation between clinical outcome and radiology, although this is a finding that has been documented by many authors previously [14–16, 21].

As already discussed this study has limitations in that it is retrospective with relatively small numbers and a lack of controls. However, we have attempted to quantify disc degeneration in a previously unstudied cohort of patients operated in a single centre by a single surgeon with uniform methods of assessment and measurement. The disc scoring system that we have used has previously been used in non-deformity studies where the endplates are often parallel and subject to symmetrical forces. However, in scoliosis, we have noted that the degeneration is often asymmetrical which often makes assessment difficult. It is our current practice to evaluate the potential distal mobile segments with MRI prior to arthrodesis and future studies will utilize this modality to provide more detailed information on asymmetric degeneration and the position of the nucleus, as well as ligament and facet hypertrophy.

In summary, we conclude from our study that disc degeneration does occur below an arthrodesis for scoliosis in adults and that revision surgery for ASD is relatively uncommon in this population. Whether degeneration occurs *de novo* with time or is related to the altered biomechanical environment is not clear, although this and

previous studies would suggest that imbalance and/or malalignment of the spine following fusion are factors that contribute to adjacent segment degeneration.

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