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Clinical Studies

Association between paraspinal muscle quality and surgery for adjacent segment disease

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ABSTRACT

Background: Adjacent segment disease (ASD) is one of the most common complications after spinal fusion. There are several risk factors for ASD, but recently the quality of the paraspinal musculature has been implicated as a potential risk factor. The purpose of this study is to examine the association between paraspinal muscle degeneration and risk of surgery for ASD.

Methods: We conducted a retrospective review of spinal fusion patients at our institution from 2009 to 2022 who underwent subsequent surgery for ASD. Inclusion criteria included patients aged 18 and older at time of index operation. Control cohort included patients who did not undergo subsequent surgery for adjacent segment disease with a minimum one year follow up. Patients were matched based on age, gender, and BMI. We measured paraspinal fat percentage and circumferential surface area (CSA) at L3 and at the proximal end of their future construct. Paraspinal fat percentage and CSA were measured using ImageJ (National Institutes of Health, Bethesda, Maryland, USA). Student T-test was used to evaluate for statistically significant differences with p-value $\leq .05$.

Results: A total of 154 patients were reviewed with 77 patients in each cohort. The average age and BMI in the control group was 61.3 and 30.0 versus 61.2 and 29.6 in patients who underwent subsequent surgery. Overall, patients who underwent surgery for adjacent segment disease had 24% higher paraspinal fat percentage at L3 ($13.8 \pm 7.7\%$ vs. $11.1 \pm 6.5\%$, p-value = .02) and 22% higher paraspinal fat percentage at the top end of their construct ($16.0 \pm 9.0\%$ vs. $13.1 \pm 7.1\%$, p-value = .03).

Conclusions: Our study found that patients who undergo surgery for adjacent segment disease have 24% higher fat percentage in their paraspinal musculature at L3 and 22% higher fat percentage at the proximal end of their fusion construct.

Introduction

Posterior spinal instrumented fusion is often used for treatment of a wide range of degenerative spine diseases, and there has been a general increase in the rate of posterior spinal instrumented fusions [1,2]. While often a successful procedure, spinal fusions are not without complications. One known complication is adjacent segment disease (ASD). It is thought that abnormal mechanical loading and increased motion through proximal or distal segments can accelerate degenerative changes of the unfused segments [3–6]. In general, there are two broad categories when discussing ASD, radiographic and symptomatic. Radiographic ASD is defined as changes on plain radiographs, computed tomographic imaging, or magnetic resonance imaging. The incidence of radiographic ASD ranges widely, with incidences ranging from 20% to 82% [7,8]. Symptomatic ASD is defined as radiographic ASD along with clinical symptoms such as persistent back pain, new onset radiculopathy or neurogenic claudication. One study found an overall annual incidence of 2.5% and a predicted 10-year prevalence of 22% of requiring additional surgery for ASD [9].

There are numerous factors associated with the development of ASD. Age greater than 60 years, BMI, a history of smoking, pre-existing facet degeneration, violation of adjacent facet joints, degenerative disc disease, multi-level fusions, and performing a laminectomy adjacent to a fusion have all been shown to be risk factors for ASD [10–13]. Another risk factor that has been the subject of recent studies is the quality of the patient's paraspinal musculature. It is thought that impairment of the paraspinal musculature could impact its ability to stabilize the vertebral segments [14]. There have been a few studies studying the relationship between paraspinal muscle quality and rate of ASD [15–18], however these studies all looked at populations in Asia. Therefore, the purpose of

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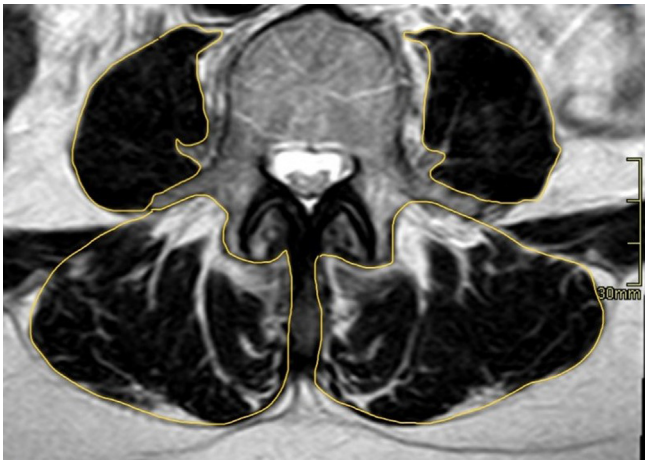

Fig. 1. Image demonstrating outlined boundaries for paraspinal and psoas musculature.

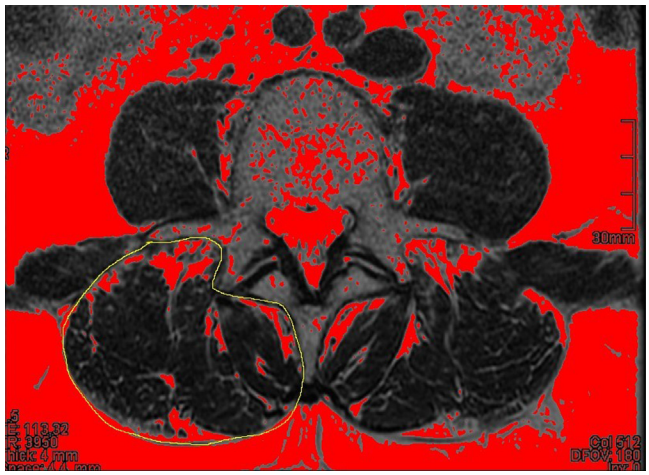

Fig. 2. Image demonstrating thresholding for calculating fat percentage. The right paraspinal musculature is outline in yellow in this figure.

this study was to examine the relationship between paraspinal muscle quality and the risk of ASD in a distinctly different population.

Methods

Patients characteristics

Institutional review board approval was obtained from our institution. We performed a retrospective review of spinal fusion patients at our institution from 2009 to 2022 who underwent subsequent surgery for symptomatic ASD. Inclusion criteria for the included patients aged 18 and older at time of index operation, underwent instrumented posterior lumbar or lumbosacral fusions, and had a presurgical MRI that included full paraspinal musculature visualization. The control cohort included patients who met the prior inclusion criteria who did not undergo subsequent surgery for symptomatic adjacent segment disease with a minimum one year follow up. Patients in the control cohort were matched 1:1 with patients from the experimental cohort based on age, gender, and BMI. We also recorded whether each patient is a current smoker, their albumin level, and hemoglobin A1c (HgA1c) level.

Measurements

For each patient we measured paraspinal fat percentage, paraspinal circumferential surface area (CSA), and psoas CSA at L3 and at the proximal end of their future surgical fusion construct. CSA was measured by drawing their outlines using ImageJ (Fig. 1). Axial turbo spine echo (TSE) T2 MRI sequences were used for measurements. Paraspinal fat percentage and muscle CSA were measured using ImageJ (National Institutes of Health, Bethesda, Maryland, USA). Fat percentage was measured by finding the average of the lowest pixel intensity values of the visceral fat immediately ventral and dorsal to the paraspinal musculature. This value was used as the threshold to differentiate fat from other soft tissues in the paraspinal musculature (Fig. 2). This measurement method allowed us to calculate the threshold for fat for each individual MRI image to account for varying MRI acquisition differences and quality of MRI images. Using a preset threshold like prior studies would result in inaccurate measurements due to differences in each MRI image. Each MRI image contained a scale that was used to correlate a set number of pixels to a known distance for the purposes of measuring CSA.

Statistics

The student T-test was used to evaluate for statistically significant differences with p-value $\leq .05$. A power analysis was performed based upon the preliminary data from the first 20 patients and for a sufficiently powered study ($\beta = 0.8$), 55 patients would be required per cohort (110 total).

Intra/inter-rater reliability

We selected 10 patients at random and 2 co-authors performed re-measurements of L3 paraspinal fat percentage, L3 paraspinal CSA, and L3 psoas CSA. We computed the intraclass and interclass correlation coefficient (ICC) using RStudio (RStudio, Boston, MA, USA) using both a 2-way mixed effects and 2-way random effects model [19].

Results

A total of 77 patients who underwent surgery for symptomatic ASD met the inclusion criteria, and 77 patients were matched for the control cohort (Table 1). The average age and BMI for the control cohort was 61.3 and 30.0 versus 61.2 and 29.6 for patients who underwent surgery for symptomatic ASD. There were no significant differences in patient age, BMI, smoking status, albumin level, or HgA1c level.

L3

The average paraspinal fat percentage for patients who underwent revision surgery for ASD and the control cohort were 13.8% ± 7.7 (range: 2.3%–37.5%) and 11.1% ± 6.5 (range: 1.9%–32.4%) ($p = .02$). There were no significant differences in paraspinal CSA or in psoas CSA at the level of L3 (Table 2).

Proximal level

The average paraspinal fat percentage for patients who underwent revision surgery for ASD and the control cohort were 16.0 ± 9.0 (range: 2.9%–43.1%) and 13.1 ± 7.1 (2.1%–33.9%) ($p = .03$). There were no significant differences in paraspinal CSA or psoas CSA at the proximal level of the construct.

Intra- and inter-rater reliability

Interpretation of ICC was based on prior published interpretations, with ICC < 0.5 indicative of poor reliability, 0.5 to 0.75 indicative of moderate reliability, 0.75 to 0.9 indicative of good reliability, and greater than 0.9 indicative of excellent reliability [19].

Table 1
Patient characteristics.

Characteristic	No adjacent segment failure (n = 77)	Adjacent segment failure (n = 77)
Age (years)		
Mean (SD)	61.3 (11.1)	61.2 (11.0)
Range	31–82	30–83
Gender		
Male	41	41
Female	36	36
BMI (Kg/m2)		
Mean (SD)	30 (5.1)	29.6 (5.6)
Range	21.8–46.6	20.0–55.0
Smoking status		
Yes	5	5
No	72	72
Albumin (g/dL)		
Mean (SD)		
HgA1c (%)		
Mean (SD)	5.6 (0.7)	5.6 (0.6)
Number of fused segments		
Mean (SD)	1.5 (0.8)	1.5 (0.7)
1	53	45
2	14	27
3	8	4
4	2	1
Proximal fused vertebrae		
L1	0	2
L2	7	6
L3	16	21
L4	39	41
L5	15	7
Clinic follow-up (days)		
Mean	841.7	
Last imaging (days)		
Mean	1576.3	

For the two-way random effects model (ICC2), all 3 intraclass correlation coefficients for paraspinal fat percentage, paraspinal CSA, and psoas CSA were found to have excellent reliability with ICC of 0.95, 1.00, and 1.00 respectively. Interclass correlation coefficient for paraspinal fat percentage was found to have good reliability (ICC = 0.76). ICC for paraspinal CSA and psoas CSA showed excellent reliability with ICC of 0.99 and 0.98 respectively (Table 3).

For the two-way fixed effects model (ICC3), all measurements showed excellent reliability (Table 3).

Table 3
Inter- and intrarater reliability of radiographic measurements.

	Intrarater reliability		Inter-rater reliability	
	ICC2	ICC3	ICC2	ICC3
L3 paraspinal fat percentage	0.95	0.95	0.76	0.91
L3 paraspinal CSA	1	1	0.99	0.99
L3 psoas CSA	1	1	0.98	0.99

CSA, circumferential surface area; ICC, intraclass/interclass correlation coefficient; ICC2, two-way random effects model; ICC3, two-way fixed effects model.

Discussion

Overall, our study found that patients with adjacent segment failure had on average higher paraspinal fat percentages at both the level of L3 and at the level of the proximal end of their construct.

Adjacent segment disease and failure is a known complication of any spinal fusion procedure. Whether it is related to changes from prior surgery or related to natural changes as we age is not clear. It is thought that abnormal mechanical loading and increased motion through proximal or distal segments can accelerate degenerative changes of the unfused segments [3–6]. In fact, one biomechanical study on cadaveric specimens found a 73% increase in the intradiscal pressure adjacent to a fused segment [20]. However, when examining the cervical spine of asymptomatic patients, it was found that 19% of patients had what were considered a major abnormality [21]. Fourteen percent of patients under the age of 40 had major abnormalities, and 28% of those over the age of 40 had major abnormalities. It is very likely that the adjacent segment changes we see are related both to biomechanical changes from surgery and to the natural aging process.

ASD had often been split into radiographic ASD and symptomatic ASD. Radiographic changes are diagnosed based upon certain radiographic criteria such as presence of listhesis > 4 mm, angular changes greater than 10° on flexion/extension radiographs, loss of disc height by greater than 10%, or deterioration of two or mare grades on the University of California, Los Angeles disc degeneration scale [8,16,17,22–24]. One study found an incidence of radiographic ASD of 29%, but the incidence of symptomatic ASD was only 18% [25]. Rates of radiographic ASD range from 20% to 82% in various studies [7,8]. A recent meta-analysis performed by Donnally et al. [26] found a pooled incidence for ASD of 36.4% after lumbar fusion. Meanwhile, symptomatic ASD has been found to occur at an overall annual incidence of 2.5% and

Table 2
Radiographic data for patients.

Radiographic measurement	No adjacent segment failure (n = 77)	Adjacent segment failure (n = 77)	p-value
L3 paraspinal fat (%)			.02
Mean (SD)	11.1 (6.5)	13.8 (7.7)	
Range	1.9–32.4	2.3–37.5	
L3 paraspinal CSA (mm^2)			.82
Mean (SD)	2729.8 (622.6)	2752.5 (597.2)	
Range	1578.6–4909.5	1738.3–4988.1	
L3 psoas CSA (mm^2)			.65
Mean (SD)	876.1 (367.0)	850.7 (327.9)	
Range	294.3–2188.9	323.7–1810.7	
Proximal paraspinal fat (%)			.03
Mean (SD)	13.1 (7.1)	16.0 (9.0)	
Range	2.1–33.9	2.9–43.1	
Proximal paraspinal CSA (mm^2)			.86
Mean (SD)	2809.0 (609.3)	2792.3 (602.3)	
Range	1889.4–4909.5	1703.4–4904.3	
Proximal psoas CSA (mm^2)			.19
Mean (SD)	1144.3 (468.4)	1046.1 (446.2)	
Range	344.1–2366.4	250.2–2420.2	

CSA = Circumferential surface area; Proximal refers to the uppermost end of the fusion construct.

a predicted 10-year prevalence of 22% of requiring additional surgery for ASD [9]. They also found that those who underwent fusion of 3 or 4 levels had a predicted 10-year prevalence of 40% of requiring additional surgery for ASD. Ghiselli et al. [22] found the 5 year and 10-year rate of requiring surgery for symptomatic ASD to be 16.5% and 36.1% respectively.

There are a multitude of risk factors for adjacent segment changes, such as increased age, BMI, smoking, HTN, preoperative adjacent disc degeneration, preoperative facet joint degeneration, and facet joint violation in surgery [10,11,16,27]. Paraspinal musculature is being investigated as a risk factor for ASD due to its role in maintaining lumbar segmental stability and lumbar lordosis. Kim et al. [16] compared patients with radiographic appearances of ASD to those without. They performed their measurements at the level of the L4 and L5 disc space. They found that those with radiographic appearances of ASD had on average 45% higher percentages of paraspinal fat (19.84 ± 9.13 vs. 13.62 ± 7.06). They also found that paraspinal CSA was larger for those without ASD (3438 mm^2 ± 820.88 vs. 3905 mm^2 ± 789.87). Their findings are generally in agreement with our study as well as we found that patients with adjacent segment failure had on average 24% higher paraspinal fat percentages. However, our study did not find any differences in paraspinal CSA. In addition, they looked at patients with radiographic signs of ASD while our study looked at patients who underwent surgery for adjacent segment failure.

Yun et al. [15] performed a similar study to ours where they compared patients who underwent subsequent surgery for ASD with a control group and found that on preoperative MRI there were no significant differences in fatty degeneration of the paraspinal musculature. However, they found that patients who underwent surgery for ASD trended towards higher percentages of fatty degeneration (15.63 ± 6.70 vs. 12.82 ± 4.37, p = .068) which were closely aligned with our results as well. One limitation of their study is the small sample size, and their study may have been underpowered. Chang et al. [17] looked at a similar population but focused on CSA of the paraspinal and psoas musculature. They found that while there was no difference in the total CSA of the paraspinal or psoas musculature, they found that patients with ASD had significantly smaller functional paraspinal CSA (2178.6 mm^2 ± 635.3 vs. 2594.0 mm^2 ± 776.4). In another study, Gong et al. [18] found no differences in erector spinae CSA at all levels but found that multifidus CSA was significantly smaller in patients who underwent surgery for ASD at the levels of L3-4, L4-5 and L5-S1. They also found no differences in the relative fat infiltration of the erector spinae muscles at all levels but found that patients who underwent surgery for ASD had higher percentages of relative fat infiltration in the multifidus muscles at levels L3-4, L4-5, and L5-S1.

In addition to preoperative paraspinal muscle quality, the effect of soft tissue damage during surgery can play a role in the development of ASD as well. Previous research has shown significant decreases in multifidus and erector spinae muscles after a posterior lumbar interbody fusion (PLIF) [28]. Many surgeons are changing from a traditional open approach that involves stripping the paraspinal muscles off of the spinous process and lamina, to a more minimally invasive approach. Kameyama et al. [29] looked at the change in paraspinal muscle changes after open PLIF versus lateral lumbar interbody fusion with posterior pedicle screw fixation (LLIF+PPS). The authors found that while the paraspinal muscle density significantly decreased in the open PLIF group, there was no change in the LLIF+PPS group. In contrast, a study by Moser et al. [30] looked at changes in paraspinal muscle morphology after a standalone LLIF without pedicle screw fixation and found that there was increased fatty infiltration postoperatively. However, the data would tend to suggest that minimally invasive surgery tends to preserve paraspinal muscle quality better than traditional open techniques.

There has also been recent interest in the association between sarcopenia and spine surgery outcomes. Sarcopenia has been defined as a syndrome characterized by progressive and generalized loss of skeletal muscle mass and strength [31]. However, the impact of sarcopenia on outcomes after spine surgery are not well known. The prevalence of sarcopenia in patients with lumbar degenerative spine disease has been reported as high as 24.8% [32,33]. A systematic review and meta-analysis performed in 2021 found no significant differences between patients with sarcopenia and those without in terms of clinical symptoms or postoperative complications [33]. However, they did find that patients with sarcopenia had worse postoperative quality of life when assessed with the EuroQol-5D. A recent retrospective review examined the association between sarcopenia index, defined as the total psoas area at the level of L3 divided by L3 vertebral body area, and 12-month patient reported outcomes (PROs) [34]. They found no association between sarcopenia index and 12-month PROs. Another systematic review and meta-analysis concluded that it was difficult to determine a relationship between sarcopenia and postoperative outcomes due to variability of definitions for and how sarcopenia is measured [35]. Prior studies have used the sarcopenia index [34] while other studies have used total psoas area [36,37], or a ratio of total psoas area to vertebral body area, or a ratio of total psoas area to height [38]. Therefore, it may be of future interest to investigate the association between sarcopenia and paraspinal fat percentage and its impact on outcomes in spinal surgery.

This study does have a few limitations. First, this is a retrospective review, and the patients were not randomized and controlled. Therefore, it is possible there are confounding factors that are unaccounted for. We attempted to control for some confounding by matching age, gender, and BMI. We also looked at each patient's smoking status, their albumin level, and their HgA1c level, which were all found to be not statistically different between cohorts. Second, there were no set criteria for re-operation for symptomatic ASD and the decision to proceed with surgery was dependent upon the surgeon and the patient. We were also unable to control for the amount of soft tissue damage that occurred during each surgery, which could affect the rates of ASD. Third, our average follow-up time for the control cohort was 841 days. It is possible that some patients in the control cohort eventually underwent surgery for ASD but were seen by a different hospital system. If that were the case, we would have no record of their subsequent surgery. Fourth, we chose to measure our fat percentages in a different manner compared to prior studies. Because our patients had MRIs performed between a large date range (2009–2022), we wanted to create a protocol that would account for the differences in MRI quality. This would result in different threshold values for fat when looking at MRIs from different years. Using a preset threshold like prior studies would lead to inaccurate fat percentage measurements. Therefore, instead of selecting a preset threshold, we chose to measure the threshold of the fat in each individual patient's MRI and used an average of the lowest values to determine our thresholds for fat. This allowed us to find the average fat density for each specific MRI, allowing us to be as accurate with our measurements as possible. Fifth, because there are many different methods for measuring paraspinal fat percentage, it is difficult for us to compare exact fat percentage values to other studies. Despite that, we believe that the trends shown in our study and prior studies are comparable and in agreement.

However, our study does have a few strengths. First, this study looked at patients based in North America. The average BMI in prior studies [15–18] ranged from 23.7 to 25.1 while the average BMI in our population was 29.8. Due to inherent differences in the two populations, their results may not have been applicable to our patient population. Second, our study included a total of 154 patients with 77 patients in each cohort, and is to our knowledge, the largest study to date. Third, our inter and intraclass correlations were good to excellent, showing good repeatability of measurements.

Conclusion

Our study found that patients who undergo surgery for adjacent segment disease have 24% higher fat percentage in their paraspinal musculature at L3 and 22% higher fat percentage at the proximal end of their fusion construct. While fatty degeneration of the paraspinal muscles may

not be a modifiable risk factor in the preoperative setting, its presence could serve as a predictive marker for increased risk of adjacent segment disease. Identifying patients with significant fatty infiltration preoperatively may allow for better risk stratification and individualized surgical planning. Future studies incorporating patient-reported outcome measures are necessary to determine whether fatty replacement of the paraspinal muscles influences functional recovery and symptom progression in patients undergoing revision surgery for adjacent segment disease.

Declaration of competing interest

One or more of the authors declare financial or professional relationships on ICMJE-NASSJ disclosure forms.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.xnsj.2025.100594.

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