JNS SPINE

CLINICAL ARTICLE

J Neurosurg Spine 37:222–231, 2022

Role of obesity in less radiographic correction and worse health-related quality-of-life outcomes following minimally invasive deformity surgery

Khoi D. Than, MD,[1] Vikram A. Mehta, MD, MPH,[1] Vivian Le, MPH,[2] Jonah R. Moss,[12] Paul Park, MD,[3] Juan S. Uribe, MD,[4] Robert K. Eastlack, MD,[5] Dean Chou, MD,[2] Kai-Ming Fu, MD,[6] Michael Y. Wang, MD,[7] Neel Anand, MD,[8] Peter G. Passias, MD,[9] Christopher I. Shaffrey, MD,[1] David O. Okonkwo, MD, PhD,[10] Adam S. Kanter, MD,[10] Pierce Nunley, MD,[11] Gregory M. Mundis Jr., MD,[5] Richard G. Fessler, MD, PhD,[12] Praveen V. Mummaneni, MD, MBA,[2] and the International Spine Study Group

[1]Departments of Neurosurgery and Orthopedics, Division of Spine, Duke University Medical Center, Durham, North Carolina; [2]Department of Neurosurgery, University of California, San Francisco, California; [3]Department of Neurosurgery, University of Michigan, Ann Arbor, Michigan; [4]Barrow Neurological Institute, Phoenix, Arizona; [5]Department of Orthopedic Surgery, Scripps Clinic, La Jolla, California; [6]Department of Neurosurgery, Cornell Medical Center, New York, New York; [7]Department of Neurosurgery, University of Miami, Florida; [8]Department of Orthopaedics, Cedars-Sinai Medical Center, Los Angeles, California; [9]Department of Orthopedics, New York University Langone Health, New York, New York; [10]Department of Neurosurgery, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania; [11]Spine Institute of Louisiana, Shreveport, Louisiana; and [12]Department of Neurological Surgery, Rush University Medical Center, Chicago, Illinois

OBJECTIVE Minimally invasive surgery (MIS) for adult spinal deformity (ASD) can offer deformity correction with less tissue manipulation and damage. However, the impact of obesity on clinical outcomes and radiographic correction following MIS for ASD is poorly understood. The goal of this study was to determine the role, if any, that obesity has on radiographic correction and health-related quality-of-life measures in MIS for ASD.

METHODS Data were collected from a multicenter database of MIS for ASD. This was a retrospective review of a prospectively collected database. Patient inclusion criteria were age ≥ 18 years and coronal Cobb angle ≥ 20°, pelvic incidence–lumbar lordosis mismatch ≥ 10°, or sagittal vertical axis (SVA) > 5 cm. A group of patients with body mass index (BMI) < 30 kg/m^2 was the control cohort; BMI ≥ 30 kg/m^2 was used to define obesity. Obesity cohorts were categorized into BMI 30–34.99 and BMI ≥ 35. All patients had at least 1 year of follow-up. Preoperative and postoperative health-related quality-of-life measures and radiographic parameters, as well as complications, were compared via statistical analysis.

RESULTS A total of 106 patients were available for analysis (69 control, 17 in the BMI 30–34.99 group, and 20 in the BMI ≥ 35 group). The average BMI was 25.24 kg/m^2 for the control group versus 32.46 kg/m^2 (p < 0.001) and 39.5 kg/m^2 (p < 0.001) for the obese groups. Preoperatively, the BMI 30–34.99 group had significantly more prior spine surgery (70.6% vs 42%, p = 0.04) and worse preoperative numeric rating scale leg scores (7.71 vs 5.08, p = 0.001). Postoperatively, the BMI 30–34.99 cohort had worse Oswestry Disability Index scores (33.86 vs 23.55, p = 0.028), greater improvement in numeric rating scale leg scores (−4.88 vs −2.71, p = 0.012), and worse SVA (51.34 vs 26.98, p = 0.042) at 1 year postoperatively. Preoperatively, the BMI ≥ 35 cohort had significantly worse frailty (4.5 vs 3.27, p = 0.001), Oswestry Disability Index scores (52.9 vs 44.83, p = 0.017), and T1 pelvic angle (26.82 vs 20.71, p = 0.038). Postoperatively, after controlling for differences in frailty, the BMI ≥ 35 cohort had significantly less improvement in their Scoliosis Research Society–22 outcomes questionnaire scores (0.603 vs 1.05, p = 0.025), higher SVA (64.71 vs 25.33, p = 0.015) and T1 pelvic angle (22.76 vs 15.48, p = 0.029), and less change in maximum Cobb angle (−3.93 vs −10.71, p = 0.034) at

ABBREVIATIONS ALIF = anterior lumbar interbody fusion; AP = anteroposterior; ASD = adult spinal deformity; BMI = body mass index; EBL = estimated blood loss; EQ-5D = EuroQol–5 Dimensions; EQ-5D VAS = EQ-5D visual analog scale; LLIF = lateral lumbar interbody fusion; LOS = length of stay; MIS = minimally invasive surgery; MISDEF = minimally invasive spine deformity; NRS = numeric rating scale; ODI = Oswestry Disability Index; PI-LL = pelvic incidence–lumbar lordosis; PT = pelvic tilt; SRS-22 = Scoliosis Research Society–22 outcomes questionnaire; SVA = sagittal vertical axis; TLIF = transforaminal lumbar interbody fusion; TPA = T1 pelvic angle.

SUBMITTED May 18, 2021. **ACCEPTED** December 9, 2021.
INCLUDE WHEN CITING Published online February 18, 2022; DOI: 10.3171/2021.12.SPINE21703.

1 year. The BMI 30–34.99 cohort had significantly more infections (11.8% vs 0%, p = 0.004). The BMI ≥ 35 cohort had significantly more implant complications (30% vs 11.8%, p = 0.014) and revision surgery within 90 days (5% vs 1.4%, p = 0.034).

CONCLUSIONS Obese patients who undergo MIS for ASD have less correction of their deformity, worse quality-of-life outcomes, more implant complications and infections, and an increased rate of revision surgery compared with their nonobese counterparts, although both groups benefit from surgery. Appropriate counseling should be provided to obese patients.

https://thejns.org/doi/abs/10.3171/2021.12.SPINE21703

KEYWORDS minimally invasive surgery; obesity; spine surgery; adult spinal deformity; revision surgery; implant complications

The number of patients discharged from the hospital with a diagnosis of adult spinal deformity (ASD) has increased by 157% over the previous 10 years.[1] Sixty million adult patients will need spinal deformity correction surgery by the year 2050.[1] One risk factor in having a poor outcome after spine surgery is a high body mass index (BMI).[2] The prevalence of obesity has tripled between 1975 and 2016.[3] Should the current rates of obesity continue, it is expected that 38% of the world's population will be obese by the year 2030.[4] This has led to an increasing focus on the impact of obesity on surgical outcomes, such as deep and superficial surgical site infections, wound breakdown, and venous thromboembolism.

Traditional deformity surgery often requires a large incision with significant tissue dissection and manipulation in order to provide sufficient exposure of the bony landmarks of the spine. In patients with high BMI, this dissection leads to increased exposure of the adipose tissue, which is a common site for infection given its minimal vasculature.[5] Newer techniques, including minimally invasive surgery (MIS), have been developed to minimize incision length, tissue dissection, and tissue manipulation. Studies have shown that MIS techniques are an effective surgical option for spinal deformity.[6,7] However, there is minimal literature regarding the impact of BMI on postoperative outcomes regarding radiographic and health-related quality-of-life measures.

The goal of this study was to determine if patients with a BMI ≥ 30 kg/m^2 have worse outcomes according to radiographic and health-related quality-of-life measures 12 months after MIS for spinal deformity correction. We hypothesized that patients with a BMI ≥ 30 kg/m^2 will have less deformity correction and worse health-related quality-of-life measures at 12 months when compared with patients with a BMI < 30 kg/m^2.

Methods

Study Design

This was a retrospective review of prospectively collected data from a multicenter database of patients who are status post-MIS for the indication of ASD. Ten institutions collected data with institutional review board approval. All patients provided informed consent to be a part of this observational study. Each surgeon has had training and experience with MIS techniques for ASD. The surgical approach was selected at the discretion of the operating surgeon. Inclusion criteria included age ≥ 18 years and coronal Cobb angle ≥ 20°, pelvic incidence–lumbar lordosis (PI-LL) mismatch ≥ 10°, or sagittal vertical axis (SVA) > 5 cm. The obesity BMI groups were categorized in accordance with the Centers for Disease Control and Prevention classification system, with one obese cohort being 30.0–34.99 kg/m^2, and the second obese cohort being ≥ 35 kg/m^2.[8] All patients with a BMI < 30.0 kg/m^2 were considered not obese and constituted the control cohort. All patients had a minimum of 1 year of follow-up.

Health-Related Quality-of-Life Measures

Demographics, operative data, and health-related quality-of-life measures were recorded in centralized, secured servers. Demographic data included age, sex, BMI, Charlson Comorbidity Index, frailty, and smoking status. Outcomes were assessed 1 year after index MIS for ASD. Operative data included estimated blood loss (EBL), operative time, length of stay (LOS), and number of levels fused. Health-related quality-of-life measures included the Oswestry Disability Index (ODI), numeric rating scale (NRS), EuroQol–5 Dimensions (EQ-5D), EQ-5D visual analog scale (EQ-5D VAS), and Scoliosis Research Society–22 outcomes questionnaire (SRS-22).

Radiographic Data

Thirty-six-inch standing radiographs were used to determine spinopelvic parameters. LL was measured by the angle created by a line extending from the superior endplate of L1 and the superior endplate of S1. PI was measured as the angle from a line drawn perpendicular to the superior endplate of S1 and a line drawn from the center of the femoral heads to the midpoint of S1. Pelvic tilt (PT) was measured as the angle between a line drawn from the midpoint of the femoral heads to the midpoint of the S1 superior endplate and a perpendicular line to the ground. PI-LL was used to calculate mismatch. The SVA was measured as the distance from the posterosuperior aspect of the superior endplate of S1 to the vertical plumb line drawn from the midpoint of C7. The T1 pelvic angle (TPA) was measured as the angle formed from a plumb line drawn from the midpoint of the superior endplate of T1 to the center of the femoral heads and a line drawn from the center of the femoral heads to the midpoint of the superior endplate of S1. Coronal balance was measured as the distance between the inferiorly projecting vertical C7 midpoint plumb line and the superiorly projecting vertical S1 midpoint plumb line.

TABLE 1. Demographic data in 106 obese and nonobese patients who underwent MIS for ASD

	BMI <30	BMI 30–34.99	p Value	BMI ≥35	p Value
No. of patients	69	17		20	
Age in yrs	68.97	68.44	0.844	69.2	0.914
Female sex	42 (61%)	11 (64.7%)	0.771	16 (80%)	0.092
BMI in kg/m^2	25.24	32.46	**<0.001**	39.5	**<0.001**
CCI	2.03	1.71	0.48	2.8	0.056
Smoker	3 (4.3%)	0 (0%)	0.46	0	0.338
Frailty score	3.27	3.78	0.162	4.5	**0.001**
Prior spine surgery	29 (42%)	12 (70.6%)	**0.04**	11 (55%)	0.335

CCI = Charlson Comorbidity Index.
Values are given as mean or number (%) unless otherwise indicated. Boldface type indicates statistical significance.

Complications

Complications were collected from each institution. The Glassman et al. categorization system was used to define major and minor complications.[9]

Statistical Analysis

Patients were categorized into groups of those with a BMI < 30.0 kg/m^2, 30.0–34.99 kg/m^2, or ≥ 35.0 kg/m^2. The cohort with a BMI < 30.0 kg/m^2 was considered the control group. Obese BMI cohorts were compared to the control group. Continuous variables are described as means, whereas categorical variables are described as frequencies. Student t-tests were used for parametric continuous data, and the Mann-Whitney U-test was used for nonparametric continuous data. Categorical data were analyzed with the chi-square test and ANOVA when appropriate. The Wilcoxon signed-rank test was used to analyze changes in spinopelvic parameters. A one-to-one propensity score matching system was used to account for any baseline differences observed. A p value ≤ 0.05 was statistically significant. IBM SPSS Statistics for Windows software (IBM Corp.) was used to perform the statistical analysis.

Results

Demographics

Demographic data are seen in Table 1. A total of 106 patients were included in the study: 69 patients with a BMI < 30 kg/m^2 (nonobese); obese BMI groups included 17 patients with a BMI of 30–34.99 kg/m^2 and 20 patients with a BMI ≥ 35 kg/m^2.

When compared to the control cohort, the BMI 30–34.99 cohort had significantly more prior spine surgery (70.6% vs 42.0%, p = 0.04). In addition, the BMI ≥ 35 cohort had significantly worse frailty scores (4.50 vs 3.27, p = 0.001). A propensity score matching system was used to account for the impact of these baseline frailty differences on health-related quality-of-life and radiographic measures. A cohort of 20 control patients and 20 BMI ≥ 35 patients were matched.

Operative Characteristics

Table 2 shows the operative data of the cohorts. No significant differences were seen in EBL, operative time, LOS, approach, total levels fused, distribution of levels fused, and interbody fusion technique (i.e., anterior lumbar interbody fusion [ALIF], lateral lumbar interbody fusion [LLIF], and transforaminal lumbar interbody fusion [TLIF]) among all cohorts. The BMI ≥ 35 cohort had significantly less anterior lumbar (25% vs 46.3%, p = 0.048) and lateral lumbar (60% vs 84.1%, p = 0.022) approaches compared to the control.

Preoperative Health-Related Quality of Life

The preoperative patient-reported data are seen in Table 3. When compared to the control, the BMI 30–34.99 cohort had significantly worse preoperative NRS leg questionnaire scores (7.71 vs 5.08, p = 0.001). When compared to the control, the BMI ≥ 35 cohort had significantly worse preoperative ODI (52.9 vs 44.83, p = 0.017). However, after propensity score matching to control for differences in baseline frailty, no differences were seen in preoperative health-related quality-of-life data, as shown in Table 3.

Postoperative Health-Related Quality of Life

The postoperative health-related quality-of-life outcomes are seen in Table 3. At 1 year postoperatively, the BMI 30–34.99 cohort had significantly worse ODI scores when compared to the control (33.86 vs 23.55, p = 0.028). Conversely, the BMI 30–34.99 cohort had significantly improved change in NRS leg score when compared to the control (−4.88 vs −2.71, p = 0.012). When compared to the control, the BMI ≥ 35 cohort had significantly worse scores in ODI (34.0 vs 23.55, p = 0.018), NRS back (4.11 vs 2.51, p = 0.011), EQ-5D (0.777 vs 0.828, p = 0.044), and SRS-22 (3.32 vs 3.72, p = 0.005) at 1 year postoperatively. However, after propensity score matching to control for differences in baseline frailty, only the change in SRS-22 was significantly less improved in the BMI ≥ 35 cohort (0.603 vs 1.05, p = 0.025).

Spinopelvic Parameters

Preoperative and postoperative spinopelvic parameter data are seen in Table 4. There were no differences in preoperative spinopelvic parameters between the control and the 30–34.99 cohort. The BMI ≥ 35 cohort had sig-

TABLE 2. Operative characteristics in 106 obese and nonobese patients who underwent MIS for ASD

	BMI <30	BMI 30–34.99	p Value	BMI ≥35	p Value
EBL	438.3	430.31	0.897	490.5	0.709
Op time	405.06	414.36	0.471	409.1	0.945
LOS	5.59	5.88	0.167	5.75	0.868
Approach			0.595		0.791
Ant	3 (4.3%)	0 (0%)		0 (0%)	
Pst	8 (11.6%)	2 (11.8%)		3 (15.0%)	
Ant/pst	54 (78.3%)	15 (88.2%)		15 (75.0%)	
MIS only	4 (5.8%)	0 (0%)		1 (5.0%)	
Same day	40 (58%)	10 (58.8%)		13 (65.0%)	
Staged	24 (34.8%)	7 (41.2%)		4 (20.0%)	
Total levels fused	5.11	6.18	0.251	4.71	0.854
No. of levels fused					0.142
1	12 (17.6%)			2 (11.1%)	
2	11 (16.2%)			7 (38.9%)	
3	13 (19.1%)			6 (33.3%)	
4	18 (26.5%)			1 (5.6%)	
5	12 (17.6%)			1 (5.6%)	
6	1 (1.5%)			1 (5.6%)	
7	1 (1.5%)			0 (0.0%)	
Interbody fusion	68 (98.6%)	16 (94.1%)	0.277	18 (90%)	0.125
No. of interbody devices	3.21	2.86	0.852	2.72	0.247
Approach					
ALIF	32 (46.3%)	10 (58.8%)	0.648	5 (25%)	**0.048**
TLIF	7 (10.1%)	2 (11.8%)	0.696	3 (15%)	0.941
LLIF	58 (84.1%)	12 (70.6%)	0.182	12 (60%)	**0.022**

Ant = anterior; pst = posterior.
Values are given as mean or number (%) unless otherwise indicated. Boldface type indicates statistical significance.

nificantly worse preoperative TPA when compared to the control group (26.82° vs 20.71°, p = 0.038). After propensity score matching to control for differences in baseline frailty, there were no differences in preoperative spinopelvic parameters between the control and the BMI ≥ 35 cohort. The BMI 30–34.99 cohort had significantly worse SVA 1 year after surgery (51.34 mm vs 26.98 mm, p = 0.042) when compared to the control. The BMI ≥ 35 cohort had significantly worse 1-year postoperative measures in SVA (64.71 mm vs 26.98 mm, p = 0.001), TPA (22.8° vs 16.43°, p = 0.007), and change in maximum Cobb angle (−3.93° vs −10.78°, p = 0.019). These significantly worse radiographic measures were maintained after propensity score matching.

Complications

The distribution and type of complications are shown in Table 5. The BMI 30–34.99 cohort had significantly more infections when compared to the control (11.8% vs 0%, p = 0.004). The BMI ≥ 35 cohort had significantly more implant complications (30% vs 7.2%, p = 0.014) and revision surgery (5% vs 0%, p = 0.034) when compared to the control. Specific implant failure differences included implant loosening (25% in BMI ≥ 35, 2.3% in BMI < 35; p < 0.001) and neural impingement by screws (5% in BMI ≥ 35, 0% in BMI < 35; p = 0.037).

A summary of significant results is shown in Table 6. There was no difference in fusion rates between groups.

Discussion

The findings of this study suggest that obese patients (defined in this paper as those with a BMI ≥ 30 kg/m^2) have significantly worse outcomes after MIS for ASD when compared to patients with a BMI < 30 kg/m^2. The cohort with BMI 30–34.99 had significantly less deformity correction as measured by the 1-year postoperative SVA. Also, the BMI 30–34.99 cohort had significantly worse ODI scores 1 year after MIS for ASD than did nonobese patients on multivariate analysis. Last, the BMI 30–34.99 cohort had significantly more infections than the control group.

The BMI ≥ 35 group had significantly worse measures of SVA, TPA, and change in maximum Cobb angle at 1 year postoperatively when compared to the control. In addition, the BMI ≥ 35 cohort had significantly less improvement in SRS-22 scores at 1 year postoperatively compared to the control. Furthermore, the BMI ≥ 35 cohort had sig-

TABLE 3. Health-related quality of life measures in obese and nonobese patients who underwent MIS for ASD

	BMI <30	BMI 30–34.99	p Value	BMI ≥35	p Value
Overall results					
Preop ODI	44.83	51.53	0.091	52.9	**0.017**
Preop NRS back	6.91	7.41	0.374	7.74	0.126
Preop NRS leg	5.08	7.71	**0.001**	6	0.233
Preop EQ-5D	0.754	0.732	0.215	0.726	0.083
Preop EQ-5D VAS	61.43	55.41	0.361	50.21	0.074
Preop SRS-22	2.89	2.63	0.077	2.65	0.071
1-yr ODI	23.55	33.86	**0.028**	34	**0.018**
1-yr NRS back	2.51	3.41	0.171	4.11	**0.011**
1-yr NRS leg	2.39	2.82	0.552	2.56	0.813
1-yr EQ-5D	0.828	0.816	0.643	0.777	**0.044**
1-yr EQ-5D VAS	93.1	67.87	0.43	69.75	0.45
1-yr SRS-22	3.72	3.37	0.07	3.32	**0.005**
Δ ODI	–21.71	–17.67	0.315	–18.9	0.478
Δ NRS back	–4.5	–4.0	0.493	–3.5	0.18
Δ NRS leg	–2.71	–4.88	**0.012**	–3.6	0.341
Δ EQ-5D	0.08	0.081	0.965	0.056	0.658
Δ EQ-5D VAS	34.78	13.07	0.527	20.13	0.164
Δ SRS-22	0.843	0.76	0.652	0.603	0.301
Propensity score matching results					
Preop ODI	53.2			52.9	0.936
Preop NRS back	7.05			7.74	0.361
Preop NRS leg	5.4			6	0.515
Preop EQ-5D	0.729			0.725	0.88
Preop EQ-5D VAS	58.19			50.21	0.35
Preop SRS-22	2.49			2.65	0.313
1-yr ODI	34.41			33.97	0.938
1-yr NRS back	3			4.11	0.184
1-yr NRS leg	3.3			2.56	0.439
1-yr EQ-5D	0.802			0.777	0.415
1-yr EQ-5D VAS	77.74			69.75	0.136
1-yr SRS-22	3.53			3.22	0.131
Δ ODI	–18.79			–18.93	0.975
Δ NRS back	–4.21			–3.5	0.47
Δ NRS leg	–2.1			–3.59	0.102
Δ EQ-5D	0.083			0.056	0.389
Δ EQ-5D VAS	20.06			20.13	0.993
Δ SRS-22	1.05			0.603	**0.025**

Boldface type indicates statistical significance.

nificantly more implant complications requiring reoperation. It is worth noting that although the final SVA for obese patients was worse than for nonobese patients, the former also started with worse SVA; the change in SVA was not statistically different between groups. Patients with BMI ≥ 35 kg/m^2 did have fewer interbody devices placed via a retroperitoneal approach (i.e., ALIF, LLIF), which could have affected radiographic parameters, implant failure, and reoperation. In terms of experience, all surgeons were experienced with MIS for deformity. The operative plans were developed according to each surgeon's individualized decision-making. Similar surgical plans were made in both cohorts, as can be seen by the similar number of levels fused and interbody devices placed in both cohorts, as illustrated in Table 2.

Obesity is a risk factor for thoracic deformity.[10–12] As ventral downward forces increase, there are increased distraction forces on the posterior column of the thoracic spine and compression of the anterior column, thus causing thoracic kyphosis. There are few data concerning the

TABLE 4. Spinopelvic parameters in obese and nonobese patients who underwent MIS for ASD

	BMI <30	BMI 30–34.99	p Value	BMI ≥35	p Value
Preop coronal balance (mm)	30.4	41.26	0.231	23.01	0.317
Preop thoracolumbar Cobb (°)	31.89	20.1	0.719	19.56	0.636
Preop lumbar Cobb (°)	32.43	20.6	0.433	24.9	0.679
Preop max Cobb (°)	38.34	25.95	0.553	27.25	0.778
Preop PI (°)	55.75	53.57	0.584	56.79	0.78
Preop PT (°)	22.3	21.13	0.494	26.44	0.119
Preop PI-LL (°)	13.91	21.18	0.109	17.47	0.411
Preop SVA (mm)	55.7	63.85	0.62	85.04	0.056
Preop TPA (°)	20.71	23.26	0.379	26.82	**0.038**
1-yr coronal balance (mm)	26.87	30.8	0.483	28.77	0.711
1-yr thoracolumbar Cobb (°)	14.67	11.51	0.39	17.34	0.502
1-yr lumbar Cobb (°)	14.01	12.54	0.641	20.42	0.065
1-yr max Cobb (°)	17.55	15.77	0.59	23.32	0.104
1-yr PI (°)	55.76	52.81	0.456	56.27	0.889
1-yr PT (°)	20.28	21.05	0.757	24.29	0.09
1-yr PI-LL (°)	4.85	9.98	0.105	8.67	0.256
1-yr SVA (mm)	26.98	51.34	**0.042**	64.71	**0.001**
1-yr TPA (°)	16.43	19.57	0.179	22.76	**0.007**
Δ coronal balance (mm)	–3.53	–10.46	0.444	5.75	0.246
Δ thoracolumbar Cobb (°)	–7.21	–8.59	0.699	–2.22	0.154
Δ lumbar Cobb (°)	–9.41	–8.06	0.644	–4.48	0.056
Δ max Cobb (°)	–10.78	–10.18	0.855	–3.93	**0.019**
Δ PI (°)	0.003	–0.762	0.093	–0.518	0.198
Δ PT (°)	–2.02	–3.08	0.5	–2.15	0.924
Δ PI-LL (°)	–9.05	–11.2	0.566	–8.8	0.939
Δ SVA (mm)	–28.72	–16.73	0.362	–20.33	0.511
Δ TPA (°)	–4.27	–4.25	0.991	–4.06	0.91

Max = maximum.
Boldface type indicates statistical significance.

TABLE 5. Complications in obese and nonobese patients who underwent MIS for ASD

	BMI <30	BMI 30–34.99	p Value	BMI ≥35	p Value
Complication	29 (42%)	6 (35.3%)	0.613	11 (55%)	0.22
Reop	10 (14.5%)	2 (11.8%)	0.771	6 (30%)	0.107
Major	9 (13%)	1 (5.9%)	0.409	3 (15%)	0.537
Minor	14 (20.3%)	6 (35.3%)	0.19	4 (20%)	0.625
Cardiac	2 (2.9%)	0	0.478	1 (5%)	0.539
Gastrological	1 (1.4%)	0	0.618	1 (5%)	0.401
Implant	5 (7.2%)	2 (11.8%)	0.542	6 (30%)	**0.014**
Infection	0	2 (11.8%)	**0.004**	0	
Neurological	12 (17.4%)	1 (5.9%)	0.235	3 (15%)	0.552
Operative	5 (7.2%)	1 (5.9%)	0.843	2 (10%)	0.495
Radiographic	9 (13%)	4 (23.5%)	0.28	5 (25%)	0.171
Renal	0	0	0	0	
Wound	4 (5.8%)	0	0.309	0	0.354
Revision >30 days	1 (1.4%)	0	NA	1 (5%)	0.401
Revision >90 days	0	0	NA	1 (5%)	**0.034**

NA = not applicable.
Values are given as number (%) unless otherwise indicated. Boldface type indicates statistical significance.

TABLE 6. Summary of outcomes in obese patients who underwent MIS for ASD, compared with controls

BMI 30–34.99	BMI ≥35
Worse 1-yr ODI scores	Less improvement in SRS-22 scores
Greater improvement in NRS leg score	Worse 1-yr SVA
Worse 1-yr SVA	Worse 1-yr TPA
More infections	Less improvement in max Cobb angle
	More implant complications
	More revision surgeries w/in 90 days

effect of BMI on postoperative spinopelvic parameter correction. Fu et al. showed that increased BMI was a risk factor for increased thoracic kyphosis after deformity correction (42.3° ± 9.1° vs 23.3° ± 10.3°, p = 0.027).[13] In addition, obesity is a risk factor for developing proximal junctional kyphosis after deformity surgery.[14]

Park et al. conducted a similar study in determining the clinical and radiographic outcomes between patients with a BMI < 30 kg/m^2 versus ≥ 30 kg/m^2 who underwent MIS for ASD.[15] They found that the BMI ≥ 30 cohort had significantly less SVA correction (83.9 mm vs 20.4 mm, p = 0.002) and PI-LL mismatch correction (17.9° vs 9.9°, p = 0.028). However, they found no differences in clinical outcomes between the two cohorts.

The minimally invasive spine deformity (MISDEF) surgery algorithm, initially described in 2014, provided the first reproducible framework for determining if a patient with deformity might be a candidate for minimally invasive spine surgery.[16] A key branching point was the presence of a flexible curve. If the deformity was flexible, an MIS technique could be considered; however, if the deformity was rigid, then a traditional open surgery was needed. This branching point was based on the limitation of MIS techniques at that time to safely complete osteotomies, whereas this is a strength of open surgery.

As MIS techniques have advanced, spine surgeons are now able to safely and consistently perform mini–open pedicle subtraction osteotomies and lateral anterior column release with the insertion of expandable cages for deformity correction. An updated version of the MISDEF (MISDEF2) algorithm was created by Mummaneni et al. in 2019 to incorporate these MIS advances.[17] A key branching point in the MISDEF2 algorithm is the number of levels fused. MIS or hybrid open approaches are considered viable options if the fixed deformity is < 5 vertebral levels (if including L5–S1) or < 10 levels (not including L5–S1).[17] Along with the MISDEF2 algorithm, there is a growing number of studies that have shown that MIS can provide significant deformity correction.[7,18,19]

For example, Fig. 1A and B shows the preoperative upright anteroposterior (AP) and lateral scoliosis radiographs of a patient with a degenerative coronal curve. This patient is a 75-year-old White man with a BMI of 24.4 kg/m^2 and a past medical history of cancer, deep vein thrombosis, and alcoholism who reported having bladder incontinence, leg weakness, and loss of balance. Imaging revealed lumbar stenosis. The neurological examination showed intact function. Figure 1C and D shows postoperative upright AP and lateral scoliosis radiographs showing improvement and stabilization of the coronal curve after MIS L2–4 LLIF with L4–S1 ALIF and posterior percutaneous L1–S1 pedicle screw instrumentation. His preoperative coronal deformity was 28.05°, with a maximum lumbar Cobb angle of 25.8° and a PI-LL mismatch of 15.12°. His postoperative coronal deformity improved to 12.2°, with a

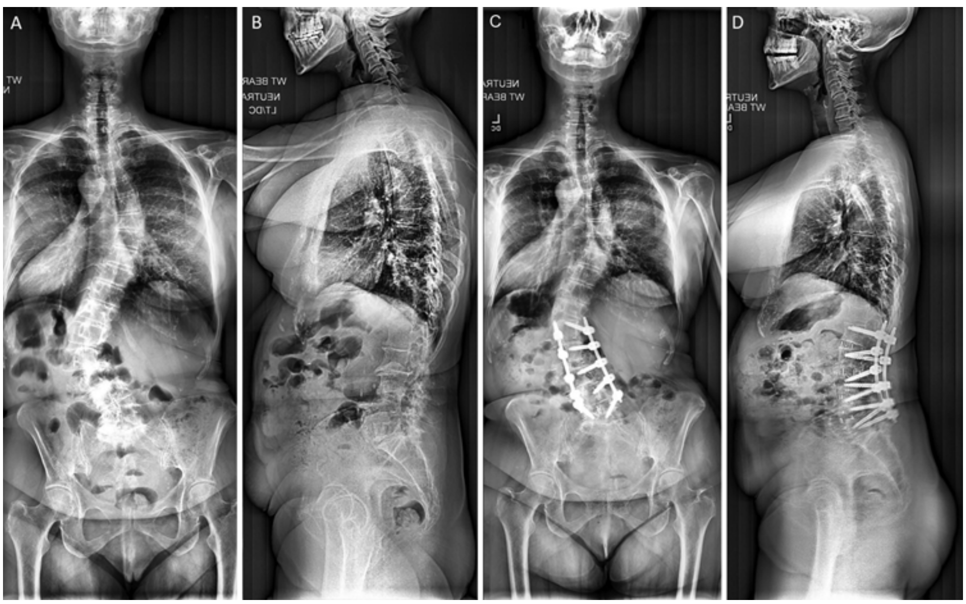

FIG. 1. A and B: Preoperative AP and lateral upright scoliosis radiographs. **C and D:** Postoperative AP and lateral upright scoliosis radiographs after L1–5 LLIF with posterior percutaneous pedicle screw instrumentation.

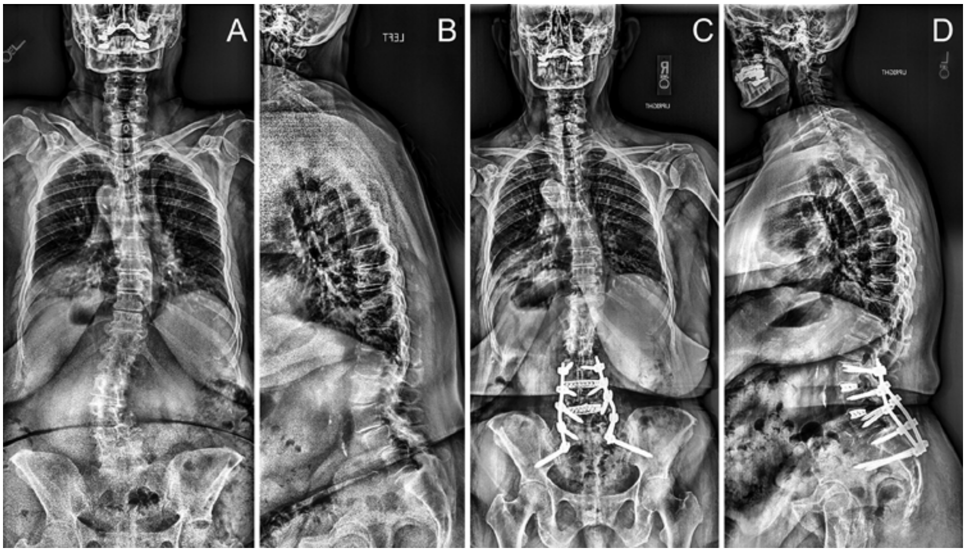

FIG. 2. A and B: Preoperative AP and lateral upright scoliosis radiographs. **C and D:** Postoperative AP and lateral upright scoliosis radiographs after L3–5 LLIF with L3–pelvis instrumentation.

maximum lumbar Cobb angle of 18.3° and a PI-LL mismatch of 2.59°.

In contrast, Fig. 2A and B shows preoperative upright AP and lateral scoliosis radiographs of an obese patient with a degenerative coronal curve, with a grade 2 spondylolisthesis of L4 on L5. This patient is a 74-year-old White woman with a BMI of 41.5 kg/m^2 and a past medical history of arthritis, diabetes, and hypertension. She presented with 6–12 months of back pain, numbness, and tingling in her legs. Her neurological examination showed intact function. Figure 2C and D shows postoperative upright AP and lateral scoliosis radiographs showing poor radiographic outcomes after L3–5 LLIF with L3–pelvis instrumentation. Her preoperative coronal deformity was 6.0°, with a maximum lumbar Cobb angle of 25.0° and a PI-LL mismatch of 27.0°. Her postoperative coronal deformity worsened to 42.0°, with a maximum lumbar Cobb angle of 12.0° and a PI-LL mismatch of 2.0°.

It is generally thought that after a deformity correction surgery, pain is reduced and mobility increases, which improves function, quality of life, and health-related quality-of-life measures.[20,21] Our data show that after MIS for deformity correction in those with a BMI of 30–34.99 kg/m^2, the patients had significantly worse ODI, and in patients with BMI ≥ 35 kg/m^2, the surgery resulted in significantly worse outcomes in depression, disability, and pain as recorded by the EQ-5D, SRS-22, ODI, and NRS scores at 1 year postoperatively. However, in the BMI ≥ 35 cohort after propensity score matching to account for frailty, only change in SRS-22 was found to be significant. There are no studies currently concerning the impact of MIS techniques on pain and depression scores after surgery for ASD. Several previous studies have documented that obesity is a risk factor for worse health-related quality-of-life measures after open ASD surgery. A study by Agarwal et al. found that an increasing BMI was associated with significantly worse outcomes as measured by the SRS-22 at 1 year postoperatively.[35] Similar findings were discovered by Smith et al., who found that BMI was associated with worse ODI and SRS-22 scores at 2 years postoperatively.[22] There is no known etiology that explains why obese patients may have worse pain and depression after MIS for deformity correction.

In the current study, the BMI ≥ 35 cohort had significantly worse frailty scores at baseline. In an aging Chinese population treated nonoperatively, frailty was a predictor of depression.[23] Frailty was also linked with chronic widespread musculoskeletal pain in a study of twins published by Livshits et al. in 2018.[24] Additionally, Tian et al. have posited that pain, depression, and frailty are interconnected. It is possible that the intervention of any surgery may have worsened the frailty in obese patients and consequently caused more pain and depression.[25] This may represent a relationship between a patient's frailty and postoperative depression, disability, and pain scores as measured by the EQ-5D, ODI, NRS, and SRS-22 surveys. Further studies are needed to determine the impact of frailty on postoperative health-related quality-of-life outcomes.

Last, we found that the obese cohort had a higher rate of implant complications and revision surgery than did the nonobese cohort. Obesity is a risk factor for readmission and revision surgery largely attributable to surgical site infection after an index spine operation.[2,26–28] Previous studies are mixed in finding BMI to be a risk factor for implant complications.[13,29–32] Soroceanu et al.[29] theorized that there might be a weight threshold rather than a BMI threshold that predisposes to implant failure. However, no additional studies have explored this. It should be noted that as BMI increases, the difficulty of performing MIS increases as well. This can be seen in our data—there was a significant increase in implant-related complications in the BMI ≥ 35 cohort.

The result of this study suggests that BMI < 30 kg/m^2 provides better outcomes after MIS for ASD. The role of preoperative bariatric surgery in spine surgery has been studied. Passias et al. showed that the preoperative bariatric surgery cohort had lower rates of device complications, hematomas, and overall complications than the cohort who did not have bariatric surgery.[33] An additional study by Jain et al. showed that having bariatric surgery prior to lumbar fusion resulted in significantly lower rates of respiratory failure, urinary tract infections, acute renal failure, medical complications, and surgical infections, and also shorter LOS when compared to a cohort of patients who did not have bariatric surgery.[34] These studies show that the role of bariatric surgery prior to elective spine surgery in the obese population is protective against perioperative complications.

Limitations

This is a retrospective review of prospectively collected data with a relatively small sample size; as such, it may be underpowered for broad generalization. There is certainly the possibility of selection bias when choosing an open surgery versus MIS for ASD. As a limitation of the retrospective nature, there are differences in baseline demographics that we attempted to control for through a propensity score matching analysis. However, given these identified baseline differences, there may be other baseline differences that we have not identified. An additional limitation is the lack of comparison to a group that underwent open surgery for deformity. The obese cohort may be likely to have worse outcomes in both MIS and open surgery for deformity. Further studies comparing outcomes for obese patients in MIS and open surgery for deformity need to be conducted. Last, some differences among the cohorts may take longer to see, and our follow-up was limited to 1 year.

Conclusions

As the rates of spinal deformity and obesity rise, there will be an increased pressure on spine surgeons to perform corrective interventions. MIS allows for correction of spinal deformity through smaller incisions and with little to no muscle disruption. This is the first study to describe the postoperative spinopelvic parameters and health-related quality-of-life measures in obese (defined as BMI $\geq$ 30 kg/m^2) patients who undergo MIS for ASD. Our results are novel in that they show that MIS for ASD results in less deformity correction, more implant complications, and worse health-related quality-of-life outcomes in obese patients compared to nonobese patients, although both groups appeared to benefit from surgery. Given the results of this study, we recommend weight loss to a BMI < 30 kg/m^2 prior to proceeding with elective major spine deformity surgery. However, within the constraints of clinical practice, a preoperative BMI < 35 kg/m^2 is recommended unless there is severe pain or neurological deficit that necessitates surgical intervention.

References

1. Ames CP, Scheer JK, Lafage V, et al. Adult spinal deformity: epidemiology, health impact, evaluation, and management. *Spine Deform*. 2016;4(4):310-322.
2. Sing DC, Yue JK, Metz LN, et al. Obesity is an independent risk factor of early complications after revision spine surgery. *Spine (Phila Pa 1976)*. 2016;41(10):E632-E640.
3. Obesity and overweight. World Health Organization. June 9, 2021. Accessed December 17, 2021. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight
4. Smith KB, Smith MS. Obesity statistics. *Prim Care*. 2016; 43(1):121-135, ix.
5. Lee JJ, Odeh KI, Holcombe SA, et al. Fat thickness as a risk factor for infection in lumbar spine surgery. *Orthopedics*. 2016;39(6):e1124-e1128.
6. Than KD, Park P, Fu KM, et al. Clinical and radiographic parameters associated with best versus worst clinical outcomes in minimally invasive spinal deformity surgery. *J Neurosurg Spine*. 2016;25(1):21-25.
7. Park P, Wang MY, Lafage V, et al. Comparison of two minimally invasive surgery strategies to treat adult spinal deformity. *J Neurosurg Spine*. 2015;22(4):374-380.
8. Luppino FS, de Wit LM, Bouvy PF, et al. Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. *Arch Gen Psychiatry*. 2010;67(3): 220-229.
9. Glassman SD, Hamill CL, Bridwell KH, Schwab FJ, Dimar JR, Lowe TG. The impact of perioperative complications on clinical outcome in adult deformity surgery. *Spine (Phila Pa 1976)*. 2007;32(24):2764-2770.
10. Valdovino AG, Bastrom TP, Reighard FG, et al. Obesity is associated with increased thoracic kyphosis in adolescent idiopathic scoliosis patients and nonscoliotic adolescents. *Spine Deform*. 2019;7(6):865-869.
11. González-Sánchez M, Luo J, Lee R, Cuesta-Vargas AI. Spine curvature analysis between participants with obesity and normal weight participants: a biplanar electromagnetic device measurement. *BioMed Res Int*. 2014;2014:935151.
12. Steele JR, Coltman CE, McGhee DE. Effects of obesity on breast size, thoracic spine structure and function, upper torso musculoskeletal pain and physical activity in women. *J Sport Health Sci*. 2020;9(2):140-148.
13. Fu L, Chang MS, Crandall DG, Revella J. Does obesity affect surgical outcomes in degenerative scoliosis? *Spine (Phila Pa 1976)*. 2014;39(24):2049-2055.
14. Wang H, Ma L, Yang D, et al. Incidence and risk factors for the progression of proximal junctional kyphosis in degenerative lumbar scoliosis following long instrumented posterior spinal fusion. *Medicine (Baltimore)*. 2016;95(32):e4443.
15. Park P, Wang MY, Nguyen S, et al. Comparison of complications and clinical and radiographic outcomes between nonobese and obese patients with adult spinal deformity undergoing minimally invasive surgery. *World Neurosurg*. 2016;87:55-60.
16. Mummaneni PV, Shaffrey CI, Lenke LG, et al. The minimally invasive spinal deformity surgery algorithm: a reproducible rational framework for decision making in minimally invasive spinal deformity surgery. *Neurosurg Focus*. 2014; 36(5):E6.
17. Mummaneni PV, Park P, Shaffrey CI, et al. The MISDEF2 algorithm: an updated algorithm for patient selection in minimally invasive deformity surgery. *J Neurosurg Spine*. 2019; 32(2):221-228.
18. Kanter AS, Tempel ZJ, Ozpinar A, Okonkwo DO. A review of minimally invasive procedures for the treatment of adult spinal deformity. *Spine (Phila Pa 1976)*. 2016;41(suppl 8): S59-S65.
19. Park P, Okonkwo DO, Nguyen S, et al. Can a minimal clinically important difference be achieved in elderly patients with adult spinal deformity who undergo minimally invasive spinal surgery? *World Neurosurg*. 2016;86:168-172.
20. Sharma A, Tanenbaum JE, Hogue O, et al. Predicting clinical outcomes following surgical correction of adult spinal deformity. *Neurosurgery*. 2019;84(3):733-740.

21. Toombs CS, Paul JC, Lonner BS. Psychosocial factors and surgical outcomes in adult spinal deformity: do dementia patients have more complications? *Spine (Phila Pa 1976)*. 2018; 43(15):1038-1043.
22. Smith JS, Shaffrey CI, Glassman SD, et al. Clinical and radiographic parameters that distinguish between the best and worst outcomes of scoliosis surgery for adults. *Eur Spine J*. 2013;22(2):402-410.
23. Feng L, Nyunt MSZ, Feng L, Yap KB, Ng TP. Frailty predicts new and persistent depressive symptoms among community-dwelling older adults: findings from Singapore longitudinal aging study. *J Am Med Dir Assoc*. 2014;15(1): 76.e7-76.e12.
24. Livshits G, Malkin I, Bowyer RCE, et al. Multi-OMICS analyses of frailty and chronic widespread musculoskeletal pain suggest involvement of shared neurological pathways. *Pain*. 2018;159(12):2565-2572.
25. Tian X, Wang C, Qiao X, et al. Association between pain and frailty among Chinese community-dwelling older adults: depression as a mediator and its interaction with pain. *Pain*. 2018;159(2):306-313.
26. De la Garza-Ramos R, Bydon M, Abt NB, et al. The impact of obesity on short- and long-term outcomes after lumbar fusion. *Spine (Phila Pa 1976)*. 2015;40(1):56-61.
27. De la Garza Ramos R, Nakhla J, Echt M, et al. Risk factors for 30-day readmissions and reoperations after 3-column osteotomy for spinal deformity. *Global Spine J*. 2018;8(5): 483-489.
28. Safaee MM, Tenorio A, Osorio JA, et al. The impact of obesity on perioperative complications in patients undergoing anterior lumbar interbody fusion. *J Neurosurg Spine*. 2021; 33(3):332-341.
29. Soroceanu A, Burton DC, Diebo BG, et al. Impact of obesity on complications, infection, and patient-reported outcomes in adult spinal deformity surgery. *J Neurosurg Spine*. 2015; 23(5):656-664.
30. Cassilly RT, Bess S, Line B, et al. Obesity is associated with increased major complications following adult spinal deformity (ASD) surgery including implant failure, wound problems and return to surgery: a propensity score matched analysis. *Spine J*. 2016;16(10 Suppl):S131.
31. Upasani VV, Caltoum C, Petcharaporn M, et al. Does obesity affect surgical outcomes in adolescent idiopathic scoliosis? *Spine (Phila Pa 1976)*. 2008;33(3):295-300.
32. Chang MS, Fu L, Crandall DG, Revella J. Does obesity affect surgical outcomes in degenerative scoliosis? *Spine J*. 2014; 14(11 Suppl):S128.
33. Passias PG, Horn SR, Vasquez-Montes D, et al. Prior bariatric surgery lowers complication rates following spine surgery in obese patients. *Acta Neurochir (Wien)*. 2018;160(12):2459-2465.
34. Jain D, Berven SH, Carter J, Zhang AL, Deviren V. Bariatric surgery before elective posterior lumbar fusion is associated with reduced medical complications and infection. *Spine J*. 2018;18(9):1526-1532.
35. Agarwal N, Angriman F, Goldschmidt E, et al. Relationship between body mass index and sagittal vertical axis change as well as health-related quality of life in 564 patients after deformity surgery. *J Neurosurg Spine*. 2019;31(5):697-702.

Disclosures

Dr. Than: consultant for Bioventus; honoraria from LifeNet Health and DJO. Dr. Park: consultant for Globus, NuVasive, Zimmer Biomet, Medtronic, and Allosource; royalties from Globus. Dr. Uribe: consultant for NuVasive, SI Bone, and Misonix; direct stock ownership in and royalties from NuVasive. Dr. Eastlack: consultant for NuVasive, Aesculap, K2M, SI Bone, Stryker, SeaSpine, Baxter, Titan, and Carevature; direct stock ownership in NuVasive, SeaSpine, Alphatec Spine, Invuity, and Spine Innovation; ownership in Spine Innovation; royalties from Globus, NuVasive, and Aesculap; patent holder with Invuity, Spine Innovation, and NuTech; research support from NuVasive, Alphatec Spine, and SeaSpine; and board member/committee appointments to Nocimed, Matrisys, San Diego Spine Foundation, Society of Lateral Access Surgery, and Scoliosis Research Society. Dr. Chou: consultant for Globus and Medtronic; royalties from Globus. Dr. Fu: consultant for SI Bone, Johnson & Johnson, and Globus. Dr. Wang: consultant for DePuy Synthes, K2M, Spineology, and Stryker; direct stock ownership in Spinicity/ISD and Medical Device Partners; medical advisory board of Vallum. Dr. Anand: consultant for Medtronic, Globus, GYS Tech, TheraCell, and Spinal Balance; direct stock ownership in Medtronic, Globus, GYS Tech, TheraCell, Atlas Spine, AF Cell, Bonovo, Paradigm Spine, and Spinal Balance; royalties from Medtronic, Globus, and Elsevier; scientific advisory board for Globus, GYS Tech, TheraCell, and Spinal Balance; speaker for DePuy Synthes and Stryker; editor of *Gray's Anatomy*. Dr. Passias: consultant to Zimmer Biomet, Medicrea, and Spinewave. Dr. Shaffrey: consultant to Medtronic, Biomet, NuVasive, and Globus; patent holder for Medtronic and Biomet. Dr. Okonkwo: consultant for Stryker; royalties from NuVasive and Zimmer Biomet. Dr. Kanter: consultant for and royalties from NuVasive and Zimmer Biomet. Dr. Nunley: consultant for Spineology, K2M, Centinel Spine, Zimmer Spine, Vertiflex, Camber, and Integrity; direct stock ownership in Amedica, Paradigm, Spineology, and Camber; patent holder with Camber; patent holder with and royalties from K2M, Zimmer Spine, and Safewire. Dr. Mundis: consultant for, receives royalties and research funding from, holds a patent with, and is on the speakers bureau and advisory council for NuVasive; board of directors for SOLAS and Global Spine Outreach; consultant for, receives honoraria and royalties from, and holds a patent with K2M; has received an honorarium and research funding from DePuy Synthes. Dr. Fessler: consultant for and royalties from DePuy Synthes; teaching and education for Benvenue; ownership in InQ Innovations. Dr. Mummaneni: consultant for Globus, DePuy Synthes, and Stryker; direct stock ownership in Spinicity/ISD; support of non–study-related clinical or research effort from AO Spine, ISSG, NIH, and NREF; royalties from DePuy Synthes, Thieme Publishers, and Springer Publishers. International Spine Study Group: funded through research grants from DePuy Synthes and individual donations.

Author Contributions

Conception and design: Than. Acquisition of data: Than. Analysis and interpretation of data: Mehta, Than. Drafting the article: Mehta, Than, Le, Park, Uribe, Eastlack, Chou, Fu, Wang, Anand, Passias, Shaffrey, Okonkwo, Kanter, Nunley, Mundis, Fessler, Mummaneni. Critically revising the article: all authors. Reviewed submitted version of manuscript: Mehta, Than, Le, Park, Uribe, Eastlack, Chou, Fu, Wang, Anand, Passias, Shaffrey, Okonkwo, Kanter, Nunley, Mundis, Fessler, Mummaneni. Approved the final version of the manuscript on behalf of all authors: Mehta. Statistical analysis: Le.

Correspondence

Vikram A. Mehta: Duke University Medical Center, Durham, NC. vikram.mehta@duke.edu.