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Is Sarcopenia a Risk Factor for Postoperative Surgical Site Infection After Posterior Lumbar Spinal Fusion?

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ABSTRACT

Background: This retrospective observational study aimed to evaluate the impact of sarcopenia on surgical site infection (SSI) risk in patients who undergo posterior lumbar fusion. While many studies have investigated the impact of sarcopenia on postoperative morbidity both in general and orthopedic surgery, none of them examined the risk of postoperative infection after lumbar spine surgery in sarcopenic vs nonsarcopenic patients.

Methods: Consecutive 55- to 75-year-old patients who underwent short posterior lumbar fusion for degenerative pathology between 2004 and 2019 were included. Charts were reviewed, and the psoas:lumbar vertebral index (PLVI) was used as a measure of central sarcopenia. Patients were stratified according to low vs high PLVI and then according to postoperative infection status. SSI was assessed as an outcome. A statistical analysis was performed to identify risk factors for infection.

Results: A total of 304 patients were included; 24 (7.9%) developed postoperative SSI. The average follow-up was 26.2 months. The sarcopenic group was found to not have a higher likelihood of experiencing postoperative SSI ($P = 0.947$). Only Charlson Comorbidity Index and American Society of Anesthesiology score were significantly associated with infectious complications ($P = 0.008$ and $P = 0.017$, respectively).

Conclusions: Low PLVI was not associated with postoperative SSI in this study. This finding is in contrast with the findings of other authors who found sarcopenia to be a risk factor for postoperative complications. However, these studies did not consider SSI as the only primary endpoint, and patients were not stratified by indication (degeneration, infection, tumor, and trauma) or surgical procedure.

Clinical Relevance: Low PLVI was not associated with postoperative SSI in patients who undergo short posterior lumbar fusion for degenerative pathology.

Level of Evidence: 3.

Complications

Keywords: sarcopenia, surgical site infection, lumbar fusion, lumbar degenerative disease, infection risk

INTRODUCTION

Surgical site infection (SSI) is the third most common complication after spinal surgery, leading to hospital readmissions and a resulting high economic burden.[1,2] Since elective lumbar spine surgery procedural volumes have increased rapidly over recent years,[3] interest has grown in identifying risk factors associated with infectious complications. In fact, a better assessment of the infection risk level could allow surgeons to optimize treatment. Sarcopenia, currently defined as a "syndrome of progressive and generalized loss of skeletal muscle mass and strength,"[4] has gained significant interest[5,6] in this field. Indeed, sarcopenia has been associated with greater complication rates, discharge disposition, increased length of stay, and perioperative morbidity and mortality, both in general and orthopedic surgery.[5,7,8] Calculating the psoas:lumbar vertebral index (PLVI), a recently validated tool for sarcopenia assessment, has been proven to predict perioperative morbidity after lumbar spine surgery.[7–9] Similarly, it has been found to be an independent predictor for postoperative infection in orthopedic surgery.[7] Nevertheless, to our knowledge, there are no studies that have examined the risk of postoperative SSI after lumbar spine surgery in sarcopenic vs nonsarcopenic patients.

The theorized pathophysiology and implications of sarcopenia (neurodegeneration, hormone imbalance, metabolic disorders, chronic inflammatory state, physical inactivity, etc) include numerous potential risk factors for SSI, leading us to hypothesize that sarcopenia may be a good summary risk indicator. In particular, we sought to establish this point in short lumbar fusion (3 levels or less). We selected short lumbar fusion because it requires less surgical exposure, has a shorter surgical time, and results in less blood loss compared with other types of spinal surgery. Hence, the aim of the present study was to evaluate sarcopenia—as quantified

by PLVI—as a systemic risk factor for SSI in patients undergoing a short posterior lumbar spinal fusion.

MATERIALS AND METHODS

A retrospective review of medical records for patients with degenerative lumbar spine diseases who underwent a short (3 levels or less) posterior lumbar fusion at our institution over a 15-year period (2004–2019) was performed.

All consecutive patients aged between 55 and 75 years who received a short lumbar spine fusion by 3 experienced surgeons (>10 years of activity, >500 surgeries per year) were included in screening. Patients who had received previous spinal surgeries, had less than 2 years of follow-up, had a diagnosis other than degenerative lumbar spine disease (such as traumatic or neoplastic diseases), had degenerative or idiopathic lumbar scoliosis, or lacked a preoperative lumbar spine magnetic resonance images (MRIs) were excluded from the study. We identified 4568 patients who underwent spine surgery in the 15 years examined in the study. After applying the study's exclusion criteria (including all nondegenerative etiologies, nonlumbar localizations, spine deformities, revision surgeries, and lumbar fusions more than 3 levels), we identified 712 patients. Of these, 304 patients had both at least a 2-year follow-up and preoperative MRI available in our archives.

Demographic data, age, gender, smoking history, body mass index (BMI), Charlson Comorbidity Index (CCI), length of stay after surgery, and American Society of Anesthesiology (ASA) classification were included for analysis. Postoperative septic complications were also recorded. The diagnosis of SSI was made by an infectious disease specialist based on clinical data, radiographic findings, blood tests, and/or a documented positive culture obtained at the time of revision or debridement surgery, up to 2 years from the primary fusion.

Next, MRIs were evaluated. The PLVI was calculated by dividing the average cross-sectional area (CSA) of the psoas by the average area of the L4 vertebrae—similar to a previously validated method[7,10,11]: PLVI = (left psoas CSA + right psoas CSA)/2 /L4 vertebral body CSA. These values were calculated on a single axial cut at the level of the L4 pedicles; images were evaluated independently by 2 reviewers, both blinded to their respective measurements and to the patient's name. The average of the 2 measurements was then recorded.

Patients were initially stratified into high vs low PLVI with the mean value (0.74) to identify baseline characteristic differences, a similar value and method to what has previously been described in literature.[7,10] The high PLVI cohort was defined as ≥0.74, and low PLVI cohort was defined as <0.74. A secondary expost statistical analysis was performed stratifying according to postoperative infective status in 2 groups: infectious vs noninfectious. Parametric test was used to compare samples in case of continuous variables and normal distribution. The Shapiro-Wilk test was used to verify normal distribution. The Levene test was used to analyze homogeneity of the variances. For the parametric test, we used the 2-tailed Student *t* test to compare the average of the variables for homoscedastic unpaired groups and the Welch *t* test for nonhomoscedastic unpaired groups. For the nonparametric test, we used the 2-tailed Mann-Whitney *U* test for unpaired groups. Continuity correction was applied in case of discrete distribution. Odds ratios were used to quantify the strength of the association between categorical variables using the χ^2 test to establish significance. Spearman coefficient was used to make correlations. "Post hoc" power analysis was not performed because it has recently been considered an improper statistical tool in retrospective studies and has been used to discredit the nonsignificance of the evidence obtained.[12,13] *P* values <0.05 were considered to be significant. All statistical analyses were performed using the Statistical Package for Social Science (IBM SPSS Statistics for Windows, Version 26.0; IBM Corp., Armonk, NY).

RESULTS

Patients and Baseline Demographics

In total, 304 patients (155 women and 149 men) met the inclusion criteria and were included in the study. The mean follow-up was 26.2 months (range 24–45 months). Patients' demographics, baseline characteristics, PLVI, and surgical variables are summarized in the Table. Mean PLVI was 0.74, ranging between 0.29 and 1.13.

Among all patients, 24 developed postoperative SSI at an mean time of 32 days after surgery (range 18–43 days). The cultures were positive for Methicillin-susceptible *Staphylococcus aureus* (12 patients), Methicillin-resistant *S aureus* (6 patients), *Enterobacter cloacae* (4 patients), and *Escherichia coli* (2 patients).

High vs Low PLVI Patients

Among the patients' demographic and health marker differences between low PLVI and high PLVI groups, only gender was found statistically significant: low

Table. Baseline characteristic differences for high vs low PLVI groups and for infected vs noninfected groups.

Characteristics	Total	Low PLVI <0.74	High PLVI ≥0.74	*P* Value	Noninfected	Infected	*P* Value
n	304	154	150		280	24	
Age at surgery, y, mean ± SD	64 ± 5.9	64.6 ± 6.0	63.6 ± 5.9	0.273	63.9 ± 5.8	66.2 ± 7.0	0.096
Gender, M/F	149/155	47/107	104/46	**<0.001**	136/144	13/11	0.599
Body mass index, mean ± SD	26.8 ± 3.5	25.2 ± 4.3	28.1 ± 3.5	0.313	25 ± 4.7	27.2 ± 4.0	0.202
Diabetes mellitus, *n*	26	12	14	0.631	22	4	0.139
Smoking history, *n*	68	36	32	0.670	60	8	0.058
Charlson Comorbidity Index, mean ± SD	2.5 ± 1.3	2.8 ± 1.6	2.3 ± 1.0	0.218	2.4 ± 1.2	3.6 ± 1.9	**0.008**
American Society of Anesthesiology score, mean ± SD	2.0 ± 0.6	2.1 ± 0.6	2.0 ± 0.6	0.663	2 ± 0.5	2.4 ± 0.8	**0.017**
Length of stay, d, mean ± SD	7.3 ± 5.6	9.9 ± 8.6	7.9 ± 3.8	0.625	9.7 ± 4.7	12.7 ± 13.5	0.075
Operative time, min, mean ± SD	191.4 ± 60.2	185 ± 62.5	197.4 ± 57.3	0.232	188.9 ± 60.0	214 ± 58.7	0.138
Infection, *n* (%)	24 (7.9%)	12 (7.8%)	12 (8%)	0.947			
PLVI, mean ± SD	0.74 ± 0.2	0.56 ± 0.1	0.85 ± 0.1	**<0.001**	0.75 ± 0.2	0.73 ± 0.2	0.686
PLVI, low/high , *n*	154/150				142/138	12/12	0.947

Abbreviation: PLVI, psoas lumbar vertebral index.
Boldface indicates statistical significance.

PLVI was found mostly in women ($P < 0.01$, Table). Nevertheless, nonsignificant differences were seen in many variables: in fact, patients in the low PLVI group were on average older (64.6 ± 6.0 vs 63.6 ± 5.9 years, $P = 0.273$), had a lower BMI (25.2 ± 4.3 vs 28.1 ± 3.5, $P = 0.313$), higher CCI (2.8 ± 1.6 vs 2.3 ± 1.0, $P = 0.218$), and had a higher ASA score (2.1 ± 0.6 vs 2.0 ± 0.6, $P = 0.663$). Moreover, using Spearman rank correlation, age, CCI, and ASA score were inversely related to PLVI (age, $\rho = -0.20$; ASA, $\rho = -0.16$; CCI, $\rho = -0.10$), despite a statistically significant difference that was found only for age at surgery ($P = 0.019$). The low PLVI group did not have a higher likelihood of experiencing postoperative SSI ($P = 0.947$).

Infectious Status

The infection rate in this cohort was 7.9% (24/304). When stratified according to postoperative SSI (Table), there were no significant differences in baseline characteristics. The infected group had a higher CCI (3.6 ± 1.9 vs 2.4 ± 1.2, $P = 0.008$) and higher ASA score (2.4 ± 0.8 vs 2 ± 0.5, $P = 0.017$). The infected group had no significant difference in their average PLVI when compared with the noninfected group (0.75 ± 0.2 vs 0.73 ± 0.2, $P = 0.686$). Therefore, while increasing ASA score and higher CCI acted as risk predictors of postoperative SSI, the patient's PLVI did not (Table).

DISCUSSION

SSI is a severe complication of spinal surgery, with an incidence that ranges between 0.2% and 16%.[1,2] It can be difficult to treat, resulting in repeated debridements, prolonged antibiotic therapy, and potential disability.[14] Therefore, assessing and recognizing risk factors are important steps when evaluating patients with lumbar degeneration and considering whether it is safe to proceed with elective surgery. The aim of the present study was to evaluate whether sarcopenia was a predictor of postoperative infection after short lumbar spinal fusion. Paraspinal muscle atrophy may be a local risk factor for SSI, both because of a lower amount of soft tissue to cover the surgical site and because of a lower blood supply. However, the purpose of the present study was to investigate sarcopenia as a systemic risk factor for infection because of the metabolic implications of sarcopenia. Therefore, measuring psoas muscle diameters as an index of sarcopenia allowed us to use an established and validated method for this measurement as well as to avoid local confounding factors.

Surprisingly, we established that patients with a low PLVI prior to elective 1- to 3-level lumbar spine fusion surgery are not more likely to experience postoperative SSI ($P = 0.947$). This is in contrast with the findings of other authors who found sarcopenia to be a risk factor for postoperative infection.[8,9] Bokshan et al[8] evaluated 46 patients undergoing thoracolumbar spine surgery and found patients with sarcopenia to have a threefold increase in perioperative complications (deep venous thrombosis, infection, and wound drainage). Similarly, Zakaria et al[9] evaluated 395 patients undergoing posterior lumbar fusion: those with lower psoas muscle area were found to have an increased risk of postoperative complications (including SSI).

However, none of the previously cited authors considered postoperative SSI alone; their endpoint was any kind of severe postoperative complication (including deep venous thrombosis, pulmonary embolism, and urinary tract infection).[8,9] Moreover, patients were not stratified by either indication (degenerative, infection, tumor, and trauma) or surgical procedure (any lumbar spine surgery

was included, such as multilevel operations and/or revisions). Another important difference between the present study and the previously cited studies is the choice of a limited age range (55–75 years), which helped in separating sarcopenia as a pathological entity from the physiological muscle mass loss associated with senescence. In fact, the use of PLVI alone as a measure of sarcopenia (not associated with functional tests) would have generated a strong bias. Zakaria et al[9] included patients of any age, with an average age similar to our patients (63.3 years) but an extremely high SD (±12.48, range 23–88 years). Bokshan et al[8] included any patient older than 55 years, obtaining a high average age difference between sarcopenic and nonsarcopenic groups (76.4 vs 69.9 years).

As for our other results, they are mostly in line with the current literature, including the infection rate for this type of surgery. Not surprisingly, age was inversely related to PLVI ($\rho = -0.204$, $P = 0.019$), and women had lower PLVI than men ($P < 0.001$). However, neither of these variables is associated with infection risk, diabetes, smoking history, or BMI. Only high CCI and ASA score were significantly associated with infectious complications ($P = 0.008$ and $P = 0.017$, respectively). While the negative impact of comorbidities (CCI and ASA score) on the outcome of spine surgery has been widely demonstrated,[15–18] some controversy still exists on some of the other variables (especially age and BMI). According to the literature, we believe that specific risk factors may be more or less relevant depending on the type of surgery—for example, in "short" vs more demolitive or invasive spinal surgery. Furthermore, investigation of these risk factors was not the purpose of our study, so we limited our study to a qualitative exploration of the data without considering the severity and level of control of diabetes with therapies or the extent of smoking. However, we emphasize that a negative trend (although not significant) was found between these risk factors and the incidence of SSI, particularly for diabetes and smoking (Table). In this regard, a recent meta-analysis by Zhou et al on 27 studies and 603 SSI cases in 22,475 patients found no strong association between age >60 years or BMI >25 and SSI.[19] Interestingly, according to our results, the operative time was also not associated with postoperative SSI ($P = 0.188$). Even though a prolonged operative duration certainly increases the chance of contamination in the surgical wounds, some authors did not describe it as a significant risk factor,[20–22] and others found direct correlation between time and SSI only after more than 3 hours of operative time.[19,23]

The main limitation of this study is its retrospective nature. Prospective studies (with proper power analysis and sample size calculation to assess adequate odds ratios) are necessary to further assess the usefulness of sarcopenia in predicting outcomes following lumbar spine surgery. An additional limitation is that only PLVI was used to define sarcopenia, although there are numerous other methods, such as measures of muscle strength (hand grip, knee flexion/extension, and peak expiratory flow) and physical performance (gait speed, timed get-up-and-go test, and stair-climb power test)[4]; however, these parameters are not possible to assess retrospectively and furthermore may be inaccurate in patients undergoing spinal surgery due to neurological compression and spinal malalignment. Additional concerns may arise regarding the secondary involvement of the psoas muscle in the context of atrophy caused by wasting due to chronic low back pain, affecting its ability to act as a systemic index of sarcopenia. However, the literature shows that psoas involvement is ancillary, identifying only the paraspinal muscles (particularly the multifidus) as the main subjects of atrophy.[24,25] Patients with spinal deformity were excluded because of the unpredictable and probably inhomogeneous consequences on the paraspinal musculature.

CONCLUSION

The results of the present study indicate that sarcopenia is not associated with SSI. Nevertheless, this condition has been widely demonstrated to have a great impact on clinical outcome of spine surgery. Therefore, further prospective investigation is needed to deepen the role of sarcopenia in predicting morbidity and mortality in patients undergoing spinal surgery.

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