Sarcopenia of the Psoas Muscles Is Associated With Poor Outcomes Following Lung Transplantation

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Background. **Sarcopenia, a known component of frailty, defined by diminished cross-sectional area of the psoas muscles, is associated with poor outcomes after a range of surgical procedures. However, little is known of the relationship between sarcopenia of the psoas muscles (SPM) and long-term survival, decline in pulmonary function, and graft failure after lung transplantation.**

Methods. **We reviewed patients who underwent primary lung transplantation at our institution from 2011 to 2014. Cross-sectional areas of the psoas muscles at the L4 vertebral level were measured using preoperative computed tomography. Gender-based cutoff values for sarcopenia were generated and validated. The primary outcomes were 1-, 2-, and 3-year all-cause mortality, forced expiratory volume in 1 second values, and graft function. Adjusted logistic regression and survival analysis was used to analyze outcomes.**

Results. **Ninety-five patients were included in this study; 39 (41.1%) patients were considered sarcopenic. SPM was significantly associated with short-term and midterm mortality on multivariate analysis (1 year: odds ratio [OR], 8.7, $p = 0.017$; 2 years: OR, 12.7, $p < 0.01$; 3 years: OR, 13.4, $p < 0.01$). Survival analysis showed significantly decreased survival in sarcopenic patients at 3 years (35.9% versus 76.8%; $p < 0.01$). SPM is also associated with decreased forced expiratory volume in 1 second (coefficient, –17.3; $p = 0.03$). Adjusted Cox analysis showed an increased hazard for all-cause mortality (hazard ratio, 5.8, $p < 0.01$) and graft failure (hazard ratio, 14.7, $p < 0.01$) in sarcopenic patients.**

Conclusions. **This study demonstrates a significant association between SPM and death, pulmonary function, and graft failure in patients receiving a lung transplant. Determining SPM preoperatively may be a useful component of frailty assessment and a predictor of survival in this patient population.**

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Frailty has emerged as a potent predictor for poor outcomes after a range of surgical procedures in various patient populations [1], but its value as a prognostic tool after lung transplantation has not been thoroughly investigated. Conventional frailty assessments such as the Fried Index often include gait speed and 6-minute walk tests [2]. These measurements can be subjective and inconvenient for healthcare professionals and is especially difficult to perform if the patients are bedridden or severely ill [3, 4]. Consequently, sarcopenia—the state of pathologically low muscle mass—has become a favored component of assessing frailty [5] and has also been associated with an increased risk for adverse outcomes, such as physical disability, poor quality of life, and early death [6].

Previous studies have demonstrated that sarcopenia can be determined by assessing the cross-sectional area of the psoas muscle at the L4 level on computed tomography (CT) [7, 8]. Assessment of sarcopenia of the psoas muscles (SPM) is a readily available diagnostic tool to assess severity of preoperative sarcopenia for patients receiving either a unilateral or bilateral lung transplant, as institutional protocols may already require abdominal CT scans on patients undergoing lung transplantation evaluation. Diminished muscle cross-sectional areas determined by single-slice CT scan are known to be associated with increased length of stay, preoperative complications, and in the case of the composite paravertebral muscle area, increased likelihood of death [9, 10]. However, no study has investigated the association of decreased cross-sectional area of the isolated psoas

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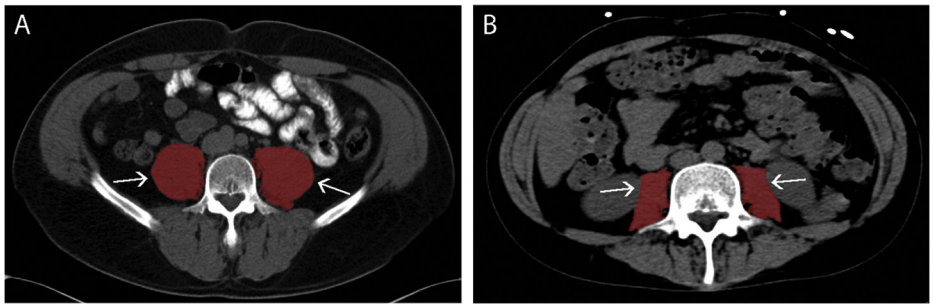

Fig 1. Cross-sections of psoas muscles on computed tomography. Axial computed tomography image of the abdomen was obtained preoperatively. 3D Slicer was used to highlight the psoas major muscles at L4 (arrows). (A) A patient who is not sarcopenic and (B) a sarcopenic patient.

muscles and mortality, long-term pulmonary function, and graft survival in lung transplant patients. Additionally, contemporary studies of muscle area in lung transplantation do not provide validated cutoffs of sarcopenia with which these assessments could be made prospectively. Cutoffs also provide the ability to enroll patients into studies of sarcopenia or direct therapy, when appropriate [11].

This study aims to provide validated cutoffs for the cross-sectional area of the psoas muscles, below which patients can be considered sarcopenic, as well as to study the association between SPM and death, long-term pulmonary function tests, and graft function after lung transplantation.

Patients and Methods

Patient Population

We conducted a retrospective study of all patients who underwent either unilateral or bilateral orthotopic lung transplant at Johns Hopkins Hospital from January 2011 to December 2014. Patients were included in the study if they had an abdominal CT imaging within 6 months before operation as sarcopenia represents chronic muscle wasting. They were excluded from the study if they lacked relevant CT imaging, or had a CT that did not allow for analysis of the psoas muscles at the L4 level. Patient demographics, operative characteristics, lung allocation score at the time of transplant, and outcomes were obtained from chart review and our institutional transplant registry. Steroid use indicated an active prescription for systemic glucocorticoid at the time of transplant. Data on mortality, graft function, and pulmonary function were also collected. This study was approved by the Johns Hopkins Institutional Review Board.

Image Analysis

To assess sarcopenia, cross-sectional imaging of the abdomen was reviewed to determine the size of the psoas major muscles. The psoas muscle area was measured at the L4 level on the first slice where both transverse processes were visible. Cross-sectional areas were obtained utilizing 3D Slicer (https://www.slicer.org), a software platform used for radiographic image analysis [12]. Density thresholds were set between –29 HU and 150 HU to exclude vasculature and areas of fatty infiltration from the calculations [13]. Measurements were performed by highlighting the psoas muscle groups using the paint tool and using the sum of the left and right psoas muscles (Fig 1). A board-certified thoracic radiologist with 4 years of postgraduate experience (C.T.L.) validated the cross-sectional area measurements on 10 randomly selected patients to ensure accuracy. Authors who participated in image analysis (J.H. and A.K.) were blinded to outcomes.

Exposure Variables

The exposure variable was SPM at L4. Cross-sectional areas for each patient was adjusted for the patient's height in m^2. Possible sex-specific cutoffs were generated using locally weighted scatterplot smoothing and spline modeling of indexed psoas muscle area and 1-year mortality to determine natural inflection points. These cutoff candidates were tested using log-rank chi-square statistical analysis to optimize sensitivity for 1-year mortality, as previously described in other patient subsets [11, 14, 15]. The male cutoff was 7.8 cm^2/m^2 and the female cutoff was 6.4 cm^2/m^2; these cutoffs are consistent with those of similar acuity patient populations, including those undergoing liver transplantation [14]. Patients were considered sarcopenic if their calculated cross-sectional area standardized to height-squared was below the cutoff value.

Outcomes

The primary endpoint was all-cause mortality up to 4 years after lung transplantation. Secondary endpoints included decreased forced expiratory volume in 1 second (FEV_1), and graft failure following lung transplantation and the incidence of any-grade bronchiolitis obliterans syndrome (BOS) [16].

FEV_1 represents the volume of air a patient can expire in the first second of a forced breath [16]. It is also described as a percentage representing the ratio of the actual volume of air expired in 1 second over the predicted volume of air expired in 1 second. In our study, we report decrease in FEV_1 as a secondary endpoint, defined as a decline in FEV_1 between the first posttransplant outpatient pulmonary function test and FEV_1 at last available follow-up.

Graft failure was defined as retransplantation or death primarily due to primary graft dysfunction, acute

rejection, chronic rejection, or pulmonary failure. Any-grade BOS was identified at follow-up visits.

Statistical Analysis

Recipient characteristics were described using means ± SD if continuous and parametric and median (interquartile range) if nonparametric. Student's *t* tests were used to compare parametric continuous characteristics, and Wilcoxon's rank sum test were used for nonparametric characteristics. Categorical variables were reported as number and percent and compared using Pearson's chi-square analysis.

Unadjusted and adjusted odds ratios were calculated with 30-day, 90-day, 1-year, 2-year, and 3-year mortality; episodes of chronic rejection; and decline in FEV_1 as the dependent variables. Adjusted linear regression was used to assess the association between sarcopenia and FEV_1 at last available follow-up. Kaplan-Meier survival analysis was conducted with 4 years as the endpoint. Log-rank tests were used to compare survival between sarcopenic and nonsarcopenic lung transplant recipients. Hazard ratios (HRs) were calculated mortality and graft failure by using multivariate Cox proportional hazards analysis.

Forward stepwise variable selection was made before risk modeling. Covariates that demonstrated univariate association to the outcome ($p < 0.20$) or were deemed of clinical importance were explored in the modeling procedure. We used variate *p* values and both Akaike and Bayesian information criteria to optimize our model. Diagnostic tests were performed to confirm model fit. Median imputation was used in variables missing less than or equal to 1% data. Statistical significance was established at *p* less than 0.05. STATA version 14.0 (StataCorp, College Station, TX) was utilized for statistical analysis.

Results

Study Population

A total of 97 patients underwent orthotopic lung transplantation at Johns Hopkins over a 4-year period. Two patients were excluded from analysis due to lack of abdominal CT within 6 months before operation. Therefore, 95 patients met our inclusion criteria and had a median follow-up of 3.5 years. Patient demographic and comorbidity data are presented in Table 1. The mean age was 50.1 ± 15.2 years and 47 (49.5%) patients were women. Most patients were white (83.1%, n = 79) and the most common diagnosis was idiopathic pulmonary fibrosis (30.5%, n = 29). The median time from CT scan to lung transplant was 2.8 (interquartile range, 1.3 to 5.3) months. Most patients underwent bilateral lung transplant (73.7%, n = 70). The remaining patients underwent unilateral left lung transplant (16.8%, n = 16) or unilateral right lung transplant (9.47%, n = 9) ($p > 0.05$). A total of 39 (41.1%) patients were considered sarcopenic by our definition. Patients who were sarcopenic had a lower mean posttransplant FEV_1 ($p < 0.01$) and higher incidence of posttransplant graft failure ($p < 0.01$).

Table 1. Baseline Clinical Characteristics of Recipient Cohort

Variable	Sarcopenic Yes (n = 39)	Nonsarcopenic No (n = 56)	*p* Value
Psoas area, cm^2/m^2	593.1 ± 106.4	861.0 ± 168.0	<0.001[a]
Age, years	50.1 ± 15.2	51.8 ± 14.8	0.59
Male	51.3 (20/39)	50 (28/56)	0.9
Race			0.28
White	84.6 (33/39)	82.1 (46/56)	
African American	7.7 (3/39)	16.1 (9/56)	
Hispanic	2.6 (1/39)	0 (0/56)	
Asian	2.6 (1/39)	0 (0/56)	
Other	2.6 (1/39)	1.8 (1/56)	
Body mass index, kg/m^2	22 ± 4.1	26.6 ± 5.7	<0.001[a]
Diagnosis			0.39
Pulmonary fibrosis	30.7 (12/39)	41.1 (23/56)	
Cystic fibrosis	35.9 (14/39)	21.4 (12/56)	
COPD	10.3 (4/39)	14.3 (8/56)	
Other[b]	30 (9/30)	23.2 (13/56)	
LAS at match	52.5 ± 18.1	45.4 ± 12.6	0.03[a]
Posttransplant FEV_1	55.1 ± 33.8	73.9 ± 27.2	0.006[a]
Posttransplant graft failure	62 (24/39)	17.9 (10/56)	<0.001[a]
Previous thoracic transplant	7.7 (3/39)	1.8 (1/56)	0.16
Steroid use	51.3 (20/39)	46.4 (26/56)	0.65
Preoperative KPS			0.96
>70	15.4 (6/39)	12.5 (7/56)	
50–70	51.3 (20/39)	62.5 (35/56)	
<50	33.3 (13/39)	25.0 (14/56)	
eGFR, ml•min•1.73 m^2	104 ± 47	96.1 ± 43.3	0.38
ICU pretransplant	18.0 (7/39)	3.6 (2/56)	0.02[a]
ECMO pretransplant	7.7 (3/39)	0 (0/56)	0.04[a]
Albumin, g/dL	3.9 ± 0.5	4.0 ± 0.7	0.36
Waitlist duration, days	43 (10–106)	44.5 (15.5–105.5)	0.81

[a] Indicates statistical significance ($p < 0.05$). [b] Sarcoidosis, rheumatoid disease, mixed connective tissue disease, constrictive bronchiolitis, short telomere deficiency, alpha-1 antitrypsin deficiency.

Values are mean ± SD, % (n/n), or median (interquartile range).

COPD = chronic obstructive pulmonary disease; ECMO = extracorporeal membrane oxygenation; eGFR = estimated glomerular filtration rate; FEV_1 = forced expiratory volume in 1 second; ICU = intensive care unit; KPS = Karnofsky Performance Status; LAS = lung allocation score.

Fourteen (14.7%) patients experienced graft failure due to chronic rejection. Patients with sarcopenia comprised 71% (10 of 14) of these patients ($p < 0.01$). There was a trend ($p > 0.05$) toward patients diagnosed with cystic fibrosis (35.9%, n = 14) and pulmonary fibrosis (30.7%, n = 12) in the sarcopenic group.

Mortality

The overall all-cause mortality rate was 36.2% (n = 35); 61.5% of sarcopenic patients died compared with 18.2% of patients who were not sarcopenic ($p < 0.01$). The most common cause of death was respiratory failure (n = 11,

Table 2. Multivariate HRs for All-Cause Mortality

Variable	HR	*p* Value	95% CI
Sarcopenia of the psoas muscles	5.8[a]	<0.001[a]	2.56–13.2[a]
Sex	0.9	0.81	0.43–1.93
Age	1.00	0.68	0.98–1.03
Steroid use	1.44	0.33	0.69–3.02
Primary diagnosis	1.0	0.7	0.99–1.02
LAS	1.04[a]	<0.001[a]	1.02–1.06[a]
Albumin	0.82	0.57	0.42–1.61

[a] Indicates statistical significance ($p < 0.05$).

CI = confidence interval; HR = hazard ratio; LAS = lung allocation score.

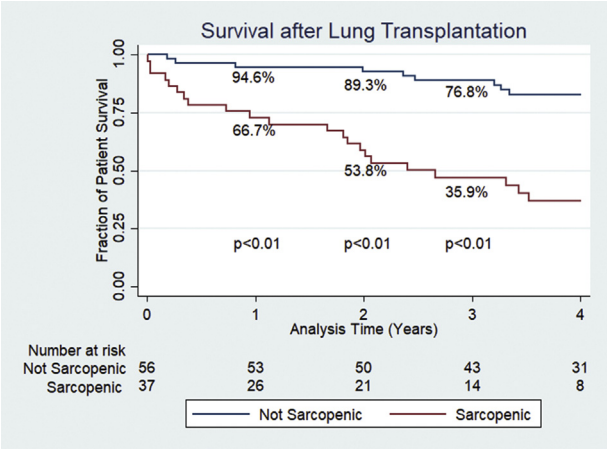

Fig 2. Kaplan-Meier survival stratified by risk group: p values shown at 1 year, 2 years, and 3 years. Patients who were sarcopenic showed decreased survival at all time points in this study. The analysis time was in years.

31.4%). Other causes of death included sepsis or septic shock (n = 5, 14.2%), bacterial or viral pneumonia (n = 4, 11.4%), chronic rejection (n = 5, 14.2%), primary graft dysfunction (n = 2, 5.7%), posttransplant lymphoproliferative disorder (n = 2, 5.7%), or miscellaneous (n = 6, 17.1%). Miscellaneous causes of death included cardiac arrest, ischemic bowel disease, or unknown cause of death.

SPM was significantly associated with all-cause mortality on univariate Cox analysis (HR, 3.0; 95% confidence interval [CI], 1.32 to 6.70; $p < 0.01$). Our multivariate model included lung allocation score, sex, age, preoperative albumin levels, primary diagnosis, and preoperative steroid use. Neither ethnicity nor whether a patient underwent a bilateral or unilateral lung transplant met our significance threshold and thus were not included in the final multivariate regression model.

Multivariate Cox proportional hazards analysis demonstrated an increased risk of mortality in sarcopenic patients (HR, 5.8; 95% CI, 2.6 to 13.2; $p < 0.01$) (Table 2). Kaplan-Meier survival curves were constructed and analyzed (Fig 2). Wilcoxon rank sum test showed significantly decreased survival at 1 year ($p < 0.01$), 2 years ($p < 0.01$), and 3 years ($p < 0.01$) in the sarcopenic group compared with the nonsarcopenic patients. Analysis of the area under the receiver-operating characteristic curve plotted with multivariate SPM displayed a C-statistic of 0.91 for predicting 1-year mortality (Fig 3).

Graft Failure

Multivariate Cox proportional hazards analysis for graft failure identified an increased risk of graft failure in the sarcopenic population (HR, 12.8; 95% CI, 3.3–48.8; $p < 0.01$) (Table 3). Multivariate Cox proportional hazards analysis also identified an association between SPM and any-grade BOS in patients (HR, 3.4; 95% CI, 1.4 to 8.0; $p < 0.01$) (Table 3).

Pulmonary Function

Multivariate linear regression of posttransplant FEV_1 showed sarcopenic patients had a 17% lower FEV_1 than did those who were not sarcopenic (coefficient, –17.3; 95% CI, –32.8 to –1.8; $p = 0.03$). Decline in FEV_1 between first pulmonary function test posttransplant and last available pulmonary function test was associated with SPM on multivariate regression (odds ratio, 4.6; 95% CI, 1.2 to 17.6; $p = 0.03$) (Table 3).

Comment

Our study establishes statistically validated cutoffs for sarcopenia in lung transplant candidates based on indexed psoas muscle cross-sectional area determined by single-slice CT scan. We showed that SPM is significantly associated with death, diminished pulmonary function, any-grade BOS, and graft failure after lung transplantation. Additionally, our multivariate model of SPM exhibited a high degree of sensitivity and specificity for predicting mortality.

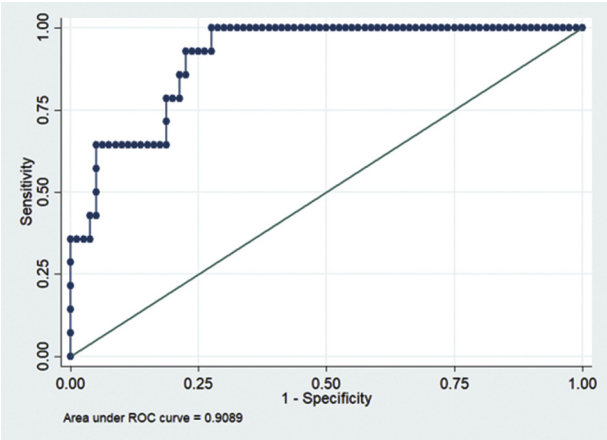

Fig 3. Receiver-operating characteristic (ROC) curve of sarcopenia of the psoas muscles. Area under the ROC curve for predicting 1-year mortality was 0.91.

Table 3. Multivariate Analysis for Graft Failure, BOS, and Decline in FEV_1

	Graft Failure			BOS			FEV_1 Decline		
Variable	HR	*p* Value	95% CI	HR	*p* Value	95% CI	OR	*p* Value	95% CI
Sarcopenia of the psoas muscles	14.5[a]	<0.001[a]	3.58–58.7[a]	3.4[a]	<0.01[a]	1.4–8.0[a]	4.6[a]	0.03[a]	1.17–17.6[a]
Sex	0.45	0.24	0.12–1.71	0.98	0.95	0.4–2.3	4.5[a]	0.01[a]	1.38–14.8[a]
Age	1.01	0.4	0.97–1.07	1.00	0.8	0.97–1.03	1.03	0.20	0.98–1.08
Steroid use	3.1	0.09	0.83–11.4	2.1	0.09	0.89–5.1	2.4	0.10	0.71–11.6
Primary diagnosis	1.0	0.6	0.99–1.02	1.0	0.4	0.99–1.0	1.0	0.11	0.99–1.00
LAS	1.07[a]	<0.01[a]	1.02–1.16[a]	1.02	0.07	0.99–1.1	1.02	0.43	0.96–1.09
Albumin	0.82	0.75	0.24–2.74	0.75	0.4	0.4–1.5	0.43	0.26	0.11–1.88

[a] Indicates statistical significance ($p < 0.05$).

BOS = bronchiolitis obliterans syndrome; CI = confidence interval; FEV_1 = forced expiratory volume in 1 second; HR = hazard ratio; LAS = lung allocation score; OR = odds ratio.

Previous studies have shown the relationship between reduced psoas muscle area, increased length of stay, and postoperative complications following lung transplantation. Weig and colleagues [9] demonstrated the inverse relationship between increasing cross-sectional psoas area and prolonged mechanical ventilation, requirement of tracheostomy, and length of stay in the intensive care unit in 103 patients who had undergone lung transplantation. Psoas area was treated as a continuous variable in this study. Our study builds on this work by providing statistically validated cutoffs for sarcopenia and showing a novel association between isolated psoas area and mortality, diminished posttransplant FEV_1, and graft failure in a similarly sized cohort of lung transplant recipients, as well as a novel association with BOS. In a separate analysis of psoas muscle area treated as a continuous variable rather than a binary variable, we identified the same associations with mortality, decreased pulmonary function, and graft failure after lung transplantation.

Kelm and colleagues [10] demonstrated the association between diminished composite paravertebral muscle area, which includes the areas of the rectus abdominus, internal and external obliques, transversus abdominis, quadratus lumborum, psoas major and minor, erector spinae, and latissimus dorsi, and mortality after lung transplantation in 36 patients. We identified a similar association with mortality using simply decreased psoas muscle area, potentially simplifying and reducing the time necessary for incorporating assessment of muscle wasting into risk prognostication. We selected only the psoas major muscle for analysis because the inclusion of a multitude of muscles in an assessment of sarcopenia may introduce variation in degree of wasting dependent on use of the muscle group. In many patients, the intercostal muscles may extend into the field of the paravertebral muscles at the level of L3, complicating the assessment of relevant muscle area [17]. Furthermore, we identified a novel association between sarcopenia of the isolated psoas muscles and graft failure, and BOS.

Assessing sarcopenia prospectively requires the use of cutoffs. Until this study, such cutoffs for SPM were not available in lung transplant patients. In other areas of surgical intervention, cutoffs for sarcopenia have been derived using the same statistical approach employed in this study [11, 14, 15]. Our cutoffs are consistent with those of similarly high-acuity surgical populations such as those undergoing liver transplantation and surgical aortic valve replacement [14, 18]. These cutoffs likely represent a lower psoas muscle area than that of a general population, given that muscle wasting is a sequelae of the end-stage pulmonary diseases that lead to transplantation [19, 20]. Once prospectively validated, our determined cutoffs may aid the eventual translation of SPM into clinical practice so clinicians may rapidly prognosticate recipient outcomes and evaluate lung transplant candidacy.

Patients who were sarcopenic in their psoas muscles at L4 level showed significantly lower survival compared with those who were not sarcopenic. This association could be explained by its direct assessment of the anatomical and physiological components of frailty, which recent evidence has suggested is a critical contributor to poor outcomes [21–23]. In addition, Shirai and colleagues [24] have also shown an association between low psoas muscle mass and poor pulmonary function in liver transplant patients. They postulated that the state of sarcopenia is "flooded with systemic inflammatory biomarkers" [24], such as tumor necrosis factor alpha, interleukin-6, and C-reactive protein. Other groups such as Garonzik-Wang and colleagues [25] demonstrated that frailty is associated with delayed graft function in kidney transplant recipients. They described frailty as a proinflammatory state—similar to pretransplant inflammation (elevated tumor necrosis factor alpha, pregnancy-associated plasma protein A) linked to delayed graft function in a study reported by Lauzurica and colleagues [26]. Taken together, a reasonable theory which links SPM to mortality, BOS, graft failure, and reduced FEV_1 could possibly be the potential inflammatory state in sarcopenic patients, leading to potentially increased rates of rejection. This would ultimately influence their systemic inflammatory response, consequently affecting graft status and muscle mass, resulting in poor physical functions.

Sarcopenic patients with end-stage pulmonary diseases identified before lung transplantation may be eligible for and should be encouraged when timing allows to undergo goal-directed preoperative medical and physical optimization and postoperative physical rehabilitation to improve survival [27]. Currently, our discharged patients receive home physical therapy as needed postoperatively. Upon completion of their home physical therapy, they are referred to outpatient pulmonary function rehabilitation. Further studies are needed to conclusively evaluate the value of preoperatively strengthening the psoas muscles in patients with SPM. Future directions may include the use of sarcopenia to target patients for "prehabilitation," or directed rehabilitation of sarcopenic patients before transplantation.

Postoperative rehabilitation is especially important for treatment of muscle wasting due to the degenerative effects of chronic immunosuppression. Additionally, if lung transplant recipients develop BOS of any grade, they receive even higher doses of immunosuppression, which may further muscle wasting after operation [28].

Finally, our multivariate SPM model demonstrated a potent C-statistic of 0.91 when assessing 1-year mortality. This suggests that including SPM into standard risk prognostication for lung transplant candidates may be beneficial in addition to the lung allocation score.

Limitations

This study faces several limitations. We provide sex-specific, but not ethnicity-specific cutoffs for sarcopenia in this study, though ethnicity may affect the baseline psoas muscle area [29]. We tried to control for potential differences by using indexed cutoffs. In addition, we examined psoas muscle area only at 1 time point (ie, within 6 months of the operation), rather than looking at changes in psoas area over a period of time. However, though changes in muscle mass may occur over time, they are not subject to short-term fluctuations such as weight [30]. Further studies are needed to prospectively validate the sarcopenia diagnostic parameters provided by our study. In addition, the measurement of sarcopenia is dependent on quality imaging that may not be available on all patients, as evidenced by the 2 patients excluded from our study.

Conclusion

Sarcopenia is an important component of the physical dimension of frailty. Preoperative SPM is associated with short-term and midterm adjusted mortality, decreased pulmonary function, and graft failure following lung transplantation. Our findings suggest SPM may serve as a key component in prognostication as well as organ allocation algorithms for patients undergoing lung transplantation in the future.

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