Original Article

Exploring the Correlation between Pulmonary Hypertension and Pectoralis Muscle Area and Density

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Highlights

- Markers of sarcopenia showed a weak correlation with the prognosis of PH.
- Some indices including right atrial pressure and systemic arterial oxygen saturation were considered as predictors of PH prognosis.

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ABSTRACT

Background: Sarcopenia is a predictor of mortality in multiple conditions, but the potential prognostic value of sarcopenia indices in pulmonary hypertension (PH) has not been clarified. This study aimed to determine whether there is an association between computed tomography (CT) scan-measured pectoralis muscle area (PMA) and density (PMD) and adverse clinical outcomes in PH patients.

Methods: In this cross-sectional study, the medical records of PH patients (clinical classes I and IV) referred to Rajaie Cardiovascular Institute from March 2016 through March 2021 were retrospectively reviewed. CT scan-measured PMA and PMD were compared between survivors and non-survivors, along with blood biomarkers and right heart catheterization variables. Binary logistic regression analysis was performed to identify potential predictors of mortality.

Results: A total of 45 patients with PH (34 survivors and 11 non-survivors) were included in the analysis. PMA was not significantly different between the two groups (P=0.12), whereas PMD differed weakly between survivors and non-survivors (survivors: 45 HU [25.8–51.3] vs. non-survivors: 31 HU [23–36]; P=0.062). In logistic regression analysis, none of the sarcopenia indices predicted mortality (P >0.05). Nonetheless, phosphodiesterase-5 (PDE-5) inhibitor use, right atrial pressure, and systemic arterial oxygen saturation were identified as potential predictors (P<0.05).

Conclusions: Although CT scan-measured PMA and PMD showed only a weak correlation with the prognosis of PH, these factors may serve as potential markers of mortality in patients with idiopathic and chronic thromboembolic PH. Further confirmation is needed through future studies with larger sample sizes.

Keywords: Pulmonary Hypertension; Pectoralis Muscle Area; Pectoralis Muscle Density; Sarcopenia; Right heart Catheterization

RHYTHM Research in Heart Yield and Translational Medicine

Introduction

Pulmonary hypertension (PH) is a rare but life-threatening disease with an incidence of 1.1–7.6 per one million adults and a 1-year mortality rate of 2.8–9.9% in developed countries.[1]

PH is defined as a mean arterial pressure (mPAP)>25 mm Hg at rest, as measured by right heart catheterization. An alternative definition with a lower cutoff (mPAP>20 mm Hg) has also been suggested for PH to include patients in the earlier stages of the disease. The contemporary classification recommended by the World Health Organization (WHO) divides PH into five clinical subgroups based on etiology and underlying pathophysiology.[2]

PH is a severe and potentially fatal condition with a 3-year survival rate of 39–77%, irrespective of the underlying etiology, based on different registries worldwide.[1] Several factors have been identified for predicting mortality in PH. It has been shown that patients with a baseline 6-minute walk distance (6MWD)>440 m have a better 1-year survival outcome than those with lower 6MWD values. Moreover, a decline in 6MWD is a predictor of worse outcomes, although an improvement in 6MWD does not affect survival.[3] Other potential prognostic factors for survival include being a male older than 60 years,[4] echocardiographic and catheterization variables,[5] right atrial size,[6] peak oxygen consumption (VO_2)/kg,[7] and ECG parameters.[8, 9]

Sarcopenia, defined as a progressive disorder of skeletal muscles characterized by reduced muscle strength, quantity, and quality,[10] has been increasingly studied in clinical contexts. Recent research highlights the potential role of pectoralis muscle volume and density measurements as indicators of disease severity and poor prognosis in various pulmonary and systemic conditions, including chronic obstructive pulmonary disease (COPD),[11] COVID-19,[12] advanced non-small cell lung cancer,[13] and gastric cancer.[14]

In this study, we aimed to evaluate the prognostic value of computed tomography (CT)-measured pectoralis muscle area (PMA) and density (PMD) as markers of sarcopenia and their association with clinical outcomes in patients diagnosed with PH. Further, we examined whether right heart catheterization parameters could predict mortality.

Methods

Study design and patient selection

This retrospective, cross-sectional, single-center study reviewed the medical records of patients with PH referred to Rajaie Cardiovascular Institute, a tertiary PH center, between March 2016 and March 2021. All diagnoses were confirmed by right heart catheterization. Patients from PH clinical classes I and IV, according to the 2015 ESC/ESR guidelines for PH,[15] were included in the study.

Other PH groups (group II: due to left heart disease; group III: due to lung diseases such as COPD; and group V: due to unknown or multifactorial causes, including sickle cell anemia or thalassemia) were excluded because they may have other causes for sarcopenia, which could confound the outcomes of interest. Additional exclusion criteria were as follows: (1) other pulmonary conditions, such as chronic lung diseases and pulmonary embolism; (2) a diagnosis of malignancy; (3) severe malnutrition; and (4) no available CT scans.

Baseline data on demographic characteristics (age, sex, weight, height, body mass index, and body surface area), clinical signs and symptoms (chest pain, dyspnea, palpitations, syncope, edema, and ascites), comorbidities (diabetes, hypertension, and dyslipidemia), laboratory results (proBNP, albumin, uric acid, liver function tests, thyroid function tests, and creatinine levels), and imaging/hemodynamic findings from the first visit (CT scans and right heart catheterization—including mPAP, cardiac output, cardiac index, right atrial pressure, pulmonary vascular resistance, and systemic vascular resistance) were collected.

Echocardiographic indices were categorized according to established guidelines.[16]

The study protocol received approval from the local ethics committee (IR.RHC.REC.1401.035).

CT scan and image analysis

All participants underwent a non-contrast chest CT scan in the supine position within 6 months of enrollment. The right pectoralis major muscle above the aortic arch was selected for the evaluation of PMA and PMD on a single axial slice. CT images were acquired using a first-generation dual-source CT scanner (Siemens Healthcare, Germany). The borders of the right pectoralis muscle were identified using an attenuation threshold of –29 to 150 Hounsfield units (HU). PMA (cm^2) and PMD (HU) were then measured for each participant by an experienced radiologist.

Outcomes of interest

All-cause mortality was defined as the primary outcome. The secondary outcome of interest was disease duration, calculated from the time of diagnosis until the end of the study period. Outcome data were continuously updated and added to our PH database through follow-up visits or, when necessary, telephone interviews. All events were verified by reviewing medical records, death certificates, or confirmation from first-degree relatives.

Statistical Analysis

Categorical data are presented as numbers and percentages, and continuous data are expressed as mean ± standard deviation (SD) or median (1st quartile–3rd quartile). The one-sample Kolmogorov-Smirnov test was performed to assess the normal distribution of continuous variables. The Mann-Whitney U test was used to compare non-parametric variables between the survivor and non-survivor groups. The Kruskal-Wallis test was utilized to compare PMA and PMD values between PH groups stratified by severity. The Pearson chi-square test or the Fisher exact test, as appropriate, was drawn upon to evaluate potential associations between outcomes and categorical variables.

The effects of baseline parameters, pulmonary function variables, and CT scan parameters on all-cause mortality were assessed using univariable and multivariable logistic regression analyses. After the univariable logistic regression was performed, predictors with a significant P-value were included in the multivariable logistic regression to determine independent predictors. Linear regression analysis was also applied to evaluate the relationship between CT scan-measured PMA and PMD and the secondary outcome of interest. This was assessed using the Pearson correlation coefficient (R) and its corresponding P-value.

The sample size included all available patients referred during the specified time frame. Since no similar study has compared the same endpoints in this population, no formal sample size calculation was performed. All analyses were conducted using SPSS version 26.0, and a two-sided P-value <0.05 was considered statistically significant.

Results

Baseline patient demographics

A total of 45 patients (29 female) diagnosed with groups I and IV PH were included in this study. The mean age of participants was 44.64±15.03 years (range: 16–81 years), with a mean PH duration of 2.09±1.47 years at enrollment. All patients underwent right heart catheterization, which demonstrated the following hemodynamic measurements: mPAP 52.12±16.42 mm Hg, pulmonary capillary wedge pressure 12.32±4.21 mm Hg, right atrial pressure 12.07 ± 8.01 mm Hg, and pulmonary vascular resistance 13.29±8.93 Wood units (WU). During the follow-up period, all-cause mortality occurred in 11 patients (24.4%), consisting of 1 male and 10 females. Baseline characteristics, echocardiographic and hemodynamic parameters, CT scan findings, and blood biomarkers for all participants are summarized in (Table 1).

Patients were stratified into survivor and non-survivor groups, with baseline characteristics and blood biomarkers compared between the groups (Table 2). The non-survivor group demonstrated significantly elevated levels of fasting blood glucose (108 mg/dL [98–157.5] vs. 95 mg/dL [82–106]; P=0.031), alkaline phosphatase (218 U/L [179–294] vs. 160 U/L [132–192]; P=0.013), and pro-B-type natriuretic peptide (1633 pg/mL [384–

3828] vs. 342 pg/mL [91–1143]; P=0.041) than survivors. Conversely, survivors showed significantly higher baseline levels of SaO (92% [88.8–95] vs. 88% [74–90]; P=0.002), SvO (62%[57–64.25] vs. 52% [41–60]; P=0.010), and triglycerides (88.5 mg/dL [71–155] vs. 68 mg/dL [62.5–83.5]; P=0.015) than non-survivors.

Table 1. Baseline characteristics of the study population

Demographic Characteristics		Total (n=45)
Age		44.64±15.03
Female, n (%)		29 (64.4)
Height		162.49±10.25
Weight		66.16±14.36
WHO-FC, n (%)	2	34 (75.6)
	3	2 (4.4)
	4	9 (20.0)
Systolic blood pressure (mm Hg)		117.57±19.80
Diastolic blood pressure (mm Hg)		75.98±12.86
Smoking, n (%)		5 (11.1)
Comorbidities		
Diabetes, n (%)		6 (13.3)
Hypertension, n (%)		7 (15.6)
Dyslipidemia, n (%)		6 (13.3)
Echocardiographic Characteristics		
LVEF (%)		48.56±6.09
RV dilation, n (%)	No dilation	4 (8.9)
	Mild	7 (15.6)
	Moderate	9 (20)
	Severe	25 (55.6)
RV dysfunction, n (%)	No dysfunction	3 (6.7)
	Mild	7 (15.6)
	Moderate	24 (53.3)
	Severe	11 (24.4)
RA dilation, n (%)	No dilation	12 (26.7)
	Mild	13 (28.9)
	Moderate	8 (17.8)
	Severe	12 (26.7)
LA dilation, n (%)	No dilation	37 (82.2)
	Mild	4 (8.9)
	Moderate	1 (2.2)
	Severe	3 (6.7)
TR severity (n=44), n (%)	Mild	12 (26.7)
	Moderate	22 (48.9)

Severe	10 (22.2)
IVC dilation, n (%)	29 (64.4)
Right Heart Catheterization Parameters	
mPAP (mm Hg)	52.12±16.42
sPAP (mm Hg)	85.89±28.56
dPAP (mm Hg)	36±13.06
RAP (mm Hg)	12.07±8.01
PVR (WU)	13.29±8.93
SVR (WU)	21.97±6.04
PCWP (mm Hg)	12.32±4.21
Medications	
Prednisolone, n (%)	5 (11.1)
Immunosuppressive, n (%)	4 (8.9)
CCBs, n (%)	5 (11.1)
Statin, n (%)	3 (6.7)
Diuretics, n (%)	35 (77.8)
Anticoagulants, n (%)	23 (51.1)
Digoxin, n (%)	11 (24.4)
Sildenafil/Tadalafil, n (%)	36 (80)
Bosentan/Macitentan, n (%)	32 (71.1)
Prostaglandin, n (%)	12 (26.7)
CT scan findings	
PMA (cm^2)	8.44±4.13
PMD (HU)	35.17±17.55

Data are presented as mean ± standard deviation (SD) or absolute number (%).
WHO-FC: World Health Organization functional class, LVEF: left ventricular ejection fraction, RAP: right atrial pressure, sPAP: systolic pulmonary arterial pressure, dPAP: diastolic pulmonary arterial pressure, mPAP: mean pulmonary arterial pressure, PVR: pulmonary vascular resistance, SVR: systemic vascular resistance, PCWP: pulmonary capillary wedge pressure, CCB: calcium channel blocker, PMA: pectoralis muscle area, PMD: pectoralis muscle density, CT: computed tomography

Table 2. Comparison of demographical characteristics and blood biomarkers between the studied groups

Variables	Survivors (n=35)	Non-Survivors (n=10)	P
Age (year)	40 (31-48)	51 (40-56)	0.055
Height (cm)	163 (158-173)	157 (149-162)	0.026*
Weight (kg)	66 (57-75)	67(60-72)	0.845
Systolic blood pressure (mm Hg)	112 (104-130)	115 (100-145)	0.669
Diastolic blood pressure (mm Hg)	76 (70-84)	80 (70-85)	0.886
Heart rate (bpm)	90 (80-97)	90 (80-100)	0.611
Blood Biomarkers			
AST (U/L)	21.5 (18.25-28)	21 (15-35)	0.773
ALT (U/L)	15 (11.5-24.5)	16 (13-29)	0.789
Alk-P (U/L)	160 (132-192)	218 (179-294)	0.013*

Direct bilirubin (mg/dL)	0.6 (0.4-0.9)	0.6 (0.5-1.1)	0.756
Total bilirubin (mg/dL)	1.45 (0.925-2.200)	1.95 (1.200-2.975)	0.254
Albumin (g/L)	41.5 (37-45.75)	40.5 (35.5-44.75)	0.708
Total protein (g/L)	64 (59-74)	67.5 (61.5-76.75)	0.388
BUN (mg/dL)	15 (12.5-19.5)	17.5 (12-30.5)	0.286
Creatinine (mg/dL)	1.1 (0.8-1.2)	1 (0.8-1.3)	0.915
Fasting blood sugar (mg/dL)	95 (82-106)	108 (98-157.5)	0.031*
TSH (mIU/ml)	2.1 (1.45-3.21)	2.02 (1.55-2.69)	0.686
Serum iron (mcg/dL)	58 (40-98.5)	39 (32-50)	0.079
Ferritin (mcg/L)	64.5 (30-230)	28 (15-101)	0.320
TIBC (mcg/dL)	340.5 (298.75-392)	304 (269-390)	0.384
Uric acid (mg/dL)	6.85 (4.80-7.97)	6.25(4.05-9.12)	0.503
Pro-BNP (pg/ml)	342 (91-1143)	1633 (384-3828)	0.041*
Triglyceride (mg/dL)	88.5 (71-155)	68 (62.5-83.5)	0.015*
Total cholesterol (mg/dL)	148.5 (123-179.75)	109 (120-146)	0.108
LDL-cholesterol (mg/dL)	80.5 (71.5-102.25)	70 (60-86)	0.165
HDL-cholesterol (mg/dL)	36 (33.25-43.75)	41 (34.5-59.5)	0.154
INR	1.2 (1.07-1.45)	1.45 (1.15-1.89)	0.141

Data are expressed as median (1st–3rd quartile).
AST: aspartate aminotransferase, ALT: alanine transaminase, Alk-P: alkaline phosphatase, BUN: blood urea nitrogen, TSH: thyroid-stimulating hormone, TIBC: total iron binding capacity, BNP: B-type natriuretic peptide, LDL: low-density lipoproteins, HDL: high-density lipoproteins, INR: international normalized ratio

Comparison of right heart catheterization and hemodynamic parameters with PMA and PMD between groups

PMA (8.44±4.13 cm²) and PMD (35.16±17.55 HU) were assessed in all participants. After the categorization of PH patients as survivors and non-survivors, PMA and PMD were compared between the two groups. Survivors had higher PMA values than non-survivors (8.16 cm² [5.99–10.74] vs. 6.36 cm² [4.62–8.84]), although the difference failed to constitute statistical significance (P=0.124). PMD exhibited a weak statistical difference between survivors and non-survivors (45 HU [25.75–51.25] vs. 31 HU [23–36]; P=0.062) (Table 3, Figure 1).

Indices of pulmonary hemodynamics were also compared between the survivors and non-survivors (Table 3). The two groups showed no statistically significant differences concerning mPAP (50.8 mm Hg [36.2–65.8] vs. 61.6 mm Hg [40.6–73.3]; P=0.203). Similarly, pulmonary capillary wedge pressure was not significantly different between survivors and non-survivors (10 mm Hg [10–15] vs. 12 mm Hg [10–15]; P=0.348). Among catheterization parameters, a significant difference was observed in right atrial pressure, with non-survivors showing higher right atrial pressure than survivors (20 mm Hg [8–25] vs. 8.5 mm Hg [6–12]; P=0.007).

Table 3. Comparison of echocardiographic parameters and PMA and PMD between survivors and non-survivors

	Survivors (n=35)	Non-Survivors (n=10)	P
CT Scan-Measured PMA and PMD			
PMA (cm²)	8.16 (5.99-10.74)	6.36 (4.62-8.84)	0.124

PMD (HU)	45 (25.75-51.25)	31 (23-36)	0.062
	Echocardiographic and Catheterization Parameters		
LVEF (%)	50 (45-55)	50 (50-55)	0.117
SaO_2 (%)	92 (88.75-95)	88 (74-90)	0.002*
SvO_2 (%)	62 (57-64.25)	52 (41-60)	0.010*
RAP (mm Hg)	8.5 (6.0 – 12.0)	20 (8-25)	0.007*
sPAP (mm Hg)	78 (63.75-105)	95 (68-110)	0.277
dPAP (mm Hg)	35 (23.75-40.75)	40 (30-50)	0.213
mPAP (mm Hg)	50.8 (36.2-65.8)	61.6 (40.6-73.3)	0.203
PVR (WU)	11 (5.9-17.5)	10.7 (6.4-16.0)	0.927
SVR (WU)	21.4 (17.3-27.5)	22.0 (17.6-24.1)	0.575
PCWP (mm Hg)	10 (10-15)	12 (10-15)	0.348
CO L/min	3.65 (3.08-4.35)	3.40 (3.10-3.80)	0.382
CI L/min/m^2	2.05 (1.79-2.56)	2.00(1.80-2.30)	0.765

Data are expressed as median (1st–3rd quartile).

PMA: pectoralis muscle area, PMD: pectoralis muscle density, LVEF: left ventricular ejection fraction, RAP: right atrial pressure, sPAP: systolic pulmonary arterial pressure, dPAP: diastolic pulmonary arterial pressure, mPAP: mean pulmonary arterial pressure, PVR: pulmonary vascular resistance, SVR: systemic vascular resistance, PCWP: pulmonary capillary wedge pressure, CO: cardiac output, SaO: systemic arterial oxygen saturation, SvO: systemic venous oxygen saturation

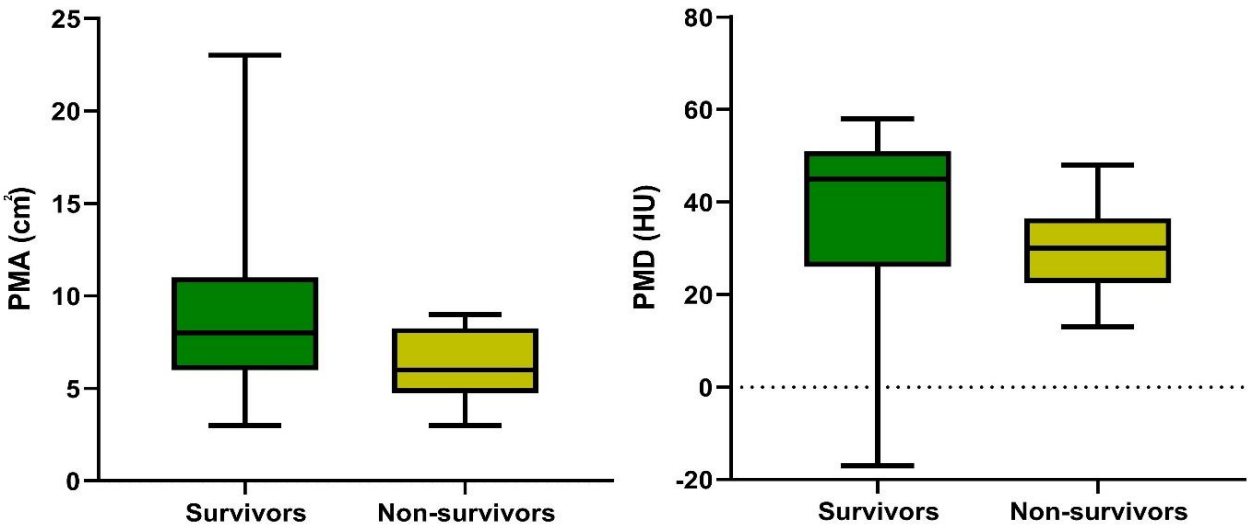

Figure 1. Pectoralis muscle area (PMA) and pectoralis muscle density (PMD) values in survivor and non-survivor groups. The upper and lower bounds of the box plots represent the minimum and maximum values, respectively.

Correlations between baseline parameters, medications, CT markers, and clinical outcomes

Analysis of medications revealed a significant association between all-cause mortality and phosphodiesterase-5 (PDE-5) inhibitor use (sildenafil/tadalafil; P=0.028). Echocardiographic parameters showed borderline significant association between right atrial dilatation and mortality (P=0.06), while no significant correlations were found with tricuspid regurgitation severity (P=0.102), right ventricular dilation (P=0.900), right ventricular dysfunction (P=0.297), left atrial dilation (P=0.246), PH severity (P=0.343), or inferior vena cava dilation (P=0.282). Linear regression analysis demonstrated no significant relationship between PMD and PH duration (r=−0.05; P=0.733) or between PMA and PH duration (r=−0.08; P=0.601). Univariable logistic regression analysis of 15 potential mortality predictors identified significant associations for

height (OR:0.91; 95% CI:0.83 to 0.99), PDE5 inhibitor use (OR:0.16; 95% CI:0.03 to 0.78), SaO (OR:0.88; 95% CI:0.80 to 0.97), SvO (OR: 0.91; 95% CI:0.84 to 0.98), alkaline phosphatase (OR:1.11; 95% CI:1.00 to 1.03), and right atrial pressure (OR:1.16; 95% CI:1.04 to 1.29). Multivariable analysis identified three independent mortality predictors: PDE5 inhibitor use (OR:0.02; 95% CI: 0.001 to 0.46), SaO (OR: 0.81; 95% CI: 0.68 to 0.97), and right atrial pressure (OR:1.35; 95% CI: 1.06 to 1.72) (Table 4).

Table 4. Univariable and multivariable logistic regression analyses regarding predictors of mortality

	Univariable Regression		Multivariable Regression	
Variables	**Odds Ratio (95% CI)**	**P**	**Odds ratio (95% CI)**	**P**
Age	1.036 (0.99-1.084)	0.129		
Height	0.904 (0.827-0.988)	0.026*		
Male sex	0.127 (0.015-1.103)	0.061		
PDE5 inhibitor use	0.160 (0.033-0.777)	0.023*	0.017 (0.001-0.456)	0.015*
SaO_2	0.880 (0.796-0.973)	0.012*	0.808 (0.676-0.966)	0.019*
SvO_2	0.905 (0.838-0.978)	0.012*		
Alk-P	1.105 (1.003-1.027)	0.015*		
Fasting blood sugar	1.022 (0.999-1.046)	0.061		
Serum iron	0.967 (0.925-1.011)	0.141		
Pro-BNP	1.001 (1.000-1.001)	0.097		
TG	0.949 (0.896-1.006)	0.078		
HDL-Cholesterol	1.072 (0.996-1.153)	0.062		
RAP	1.157 (1.038-1.289)	0.008*	1.349 (1.057-1.721)	0.016*
PMA	0.807 (0.622-1.047)	0.106		
PMD	0.980 (0.943-1.017)	0.283		

PDE5: phosphodiesterase-5, SaO: systemic arterial oxygen saturation, SvO: systemic venous oxygen saturation, Alk-P: alkaline phosphatase, BNP: B-type natriuretic peptide, TG: triglyceride, HDL: high-density lipoprotein, RAP: right atrial pressure, PMA: pectoralis muscle area, PMD: pectoralis muscle density.

Discussion

Sarcopenia has been established as an indicator of poor clinical outcomes in various chronic diseases, including heart failure and COPD, which frequently coexist with PH.[17–19] Nonetheless, the prognostic value of sarcopenia indices, such as CT scan-measured PMA and PMD, in PH with idiopathic and chronic thromboembolic causes (clinical classes I and IV) remains unclear. Accordingly, this study aimed to assess the association between sarcopenia indices and adverse clinical outcomes in PH.

We compared PMA and PMD between survivors and non-survivors to investigate whether these markers could predict mortality. Although the survivor group displayed a higher mean PMA value, the difference did not reach statistical significance. Similarly, survivors had a higher mean PMD value than non-survivors, a finding with borderline statistical significance (P=0.06). In the logistic regression analysis, neither CT scan parameter predicted mortality. Among the other parameters studied, PDE5 inhibitor use, SaO_2, and right atrial pressure were associated with all-cause mortality.

The relationship between sarcopenia and mortality has been well-established in various conditions, including COVID-19,[20] ovarian cancer,[21] and dialysis.[22] Previous studies have also shown that a higher PMA is associated with better lung function in patients with COPD.[23] Nevertheless, the predictive value of PMA for adverse clinical outcomes in PH has yet to be elucidated.

The lower survival rate in patients with sarcopenia may result from different etiologies. A state of chronic inflammation and immune dysfunction has been proposed as a potential cause of poor outcomes due to sarcopenia.[20] Furthermore, sarcopenia, which predominantly

affects the elderly, predisposes this population to falls, fractures, and mobility problems, leading to diminished quality of life and higher mortality.[10]

Skeletal and respiratory muscle atrophy in PH has been associated with lower quality of life and reduced exercise capacity through several hypothesized mechanisms, such as impaired oxygen supply and the production of catabolic inflammatory markers. It should be noted that muscle strength and exercise capacity are recognized predictors of mortality.[24] Although PH can directly affect skeletal muscle strength, this effect may not be evident in the muscle area. Breda et al.[25] reported that in idiopathic and schistosomiasis-associated PH, quadriceps muscle strength (peak torque at 60°/s and total work at 240°/s) was significantly lower than that in the control group. However, quadriceps muscle area was not influenced by PH (108.5 vs. 120.5 in the PH and control groups, respectively).

In our study, PMA and PMD values did not predict mortality or hospitalization rates in idiopathic and chronic thromboembolic PH. These findings do not confirm the prognostic value of CT scan-measured PMA and PMD in PH. Still, the potential prognostic role of skeletal muscle strength in predicting adverse outcomes remains to be clarified in future studies.

The present study has several important limitations. First and foremost, the small sample size significantly constrained our analysis, particularly given the inherent rarity of idiopathic and chronic thromboembolic PH cases. The inclusion of only groups I and IV PH patients further limited our cohort size, potentially affecting the statistical power and reliability of our findings. Additionally, as a single-center investigation, the generalizability of our results may be limited. These constraints highlight the need for future multicenter studies with larger patient populations to validate our observations.

Conclusion

In this study, we explored the possible correlation between sarcopenia indices and mortality in patients with PH. Our results demonstrated that PMA and PMD had no statistically significant impact on all-cause mortality or the duration of PH. Among the medications, blood biomarkers, and echocardiographic findings analyzed, we found that PDE5 inhibitor use, SaO_2, and right atrial pressure may be potential prognostic factors for PH.

Although CT scan-measured PMA and PMD were not found to be significant predictors in this cohort, future large-scale studies are warranted to definitively investigate the prognostic value of these sarcopenia indices in patients with PH.

Declarations:
Ethical Approval

The study protocol was approved by the local ethics committee (Approval ID: IR.RHC.REC.1401.035) in accordance with ethical standards.

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Conflict of Interest

The authors declare no conflicts of interest.

Acknowledgment

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