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Psoas muscle index as a predictor of mortality following percutaneous coronary intervention

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ABSTRACT

Background Percutaneous coronary intervention (PCI) is a widely utilized revascularization technique for coronary artery disease (CAD). While clinical and biomarker-based prognostic tools are standard for predicting outcomes, there is growing interest in sarcopenia as a marker of frailty and its potential role in long-term prognosis. The prognostic value of the psoas muscle index (PMI), a sarcopenia metric, remains underexplored in PCI populations regarding long term survival.

Methods This single-center retrospective cohort study evaluated 177 patients undergoing PCI from 2015 to 2019. PMI was calculated from computed tomography (CT) imaging at the L3 vertebral level using the formula: (left psoas area + right psoas area)/height^2 and expressed in cm^2/m^2. Sarcopenia was defined as the lowest sex-specific PMI quartile. Primary outcomes included 5-year all-cause mortality and 3-point major adverse cardiovascular events (MACE: non-fatal myocardial infarction, ischemic stroke, and cardiac death). Binary linear regression and Cox proportional hazards models were utilized to determine associations between PMI and outcomes

Results Sarcopenic patients exhibited significantly higher 5-year all-cause mortality compared to non-sarcopenic counterparts (64.4% vs. 35.6%, $P < 0.001$), while no significant difference was observed in 3-point MACE incidence (55.6% *vs.* 51.4%, $P = 0.520$). Sarcopenia was independently associated with all-cause mortality on binary logistic regression (OR = 3.49; 95% CI: 1.69–7.19; $P = 0.0007$), but not MACE (OR = 1.00; 95% CI: 0.50–1.98; $P = 0.99$). In a multivariable Cox regression model, sarcopenia was associated with increased hazard of mortality (HR = 1.60; 95% CI: 0.96–2.66; $P = 0.071$), though this did not reach statistical significance. Kaplan-Meier analysis demonstrated significantly reduced survival among sarcopenic patients ($\chi^2 = 6.13$, $P = 0.0133$).

Conclusions PMI is a significant independent predictor of 5-year all-cause mortality in PCI patients, underscoring the prognostic importance of assessing skeletal muscle mass in this population.

Percutaneous coronary intervention (PCI) has become a cornerstone of treatment for coronary artery disease (CAD) worldwide, with more than two million procedures performed annually.[1] Despite its widespread use, ongoing debate remains regarding the most effective methods for long term risk stratification in PCI patients, particularly in predicting major adverse cardiac events (MACE) and overall mortality.[2-5] Traditional biomarkers such as troponins and brain natriuretic peptide (BNP) may not fully capture the overall physiological state or frailty of the patient, which can significantly impact long-term outcomes after PCI.[6,7]

Sarcopenia, defined as the loss of skeletal muscle mass and physical performance, is emerging as a significant predictor in outcomes in various cardiovascular conditions, particularly in cardiac surgery populations.[8-10] While skeletal muscle index (SMI) at the L3 level has traditionally been used, the psoas muscle index (PMI), a cross-sectional area measurement of the bilateral psoas muscles, has gained traction as a practical alternative due to its ease of measurement on CT, reproducibility, and strong correlation with total skeletal muscle mass and clinical outcomes.[11-15] Despite these findings, there is limited data on the role of PMI in patients undergoing both elective and emergent PCI, particularly in relation to long-term outcomes. Existing studies have primarily focused on short-term outcomes, leaving a gap in under-

standing the long-term prognostic utility of PMI in diverse PCI settings.[16]

This study aims to evaluate the prognostic significance of PMI in patients undergoing both elective and emergent PCI. By investigating this association, we hope to provide further insight into the utility of PMI as a prognostic tool to support long-term risk stratification and post-PCI management in this vulnerable population.

METHODS

This single-center retrospective analysis was conducted at the University of Pittsburgh Medical Center and included 177 adult patients who underwent PCI between 2015 and 2019. Inclusion criteria were: (1) age ≥ 18 years and (2) availability of abdominal or pelvic CT imaging performed within 90 days prior to or following PCI. Patients were excluded if they (1) lacked CT imaging within this timeframe or (2) lacked 5-year follow-up data. Patients with a history of heart failure and/or chronic kidney disease were not excluded and were included in the final analysis to reflect real-world PCI populations. A flowchart summarizing the inclusion and exclusion process is provided in Figure 1. This study was approved by the University of Pittsburgh Medical Center Institutional Review Board (IRB), with a waiver of informed consent due to its retrospective design.

Data Collection

Patient data were securely stored and managed using a password-protected database. Collected information included demographic variables (e.g., age, gender), comorbidities, social history (e.g., smoking status), and the nature of PCI (emergent vs. elective). The primary outcome measures were overall mortality and overall survival within five years post-PCI. Secondary outcomes included the incidence of 3-point MACE which comprised cardiac death, non-fatal myocardial infarction (MI) and ischemic stroke (IS).

Psoas Muscle Measurement and Definition of Sarcopenia

PMI was calculated using clinically indicated abdominal or pelvic CT scans performed within 90 days of PCI. These scans were obtained as part of routine care, not for research purposes, and incurred no additional cost to patients. Cross-sectional psoas muscle area was measured at the level of the L3 vertebral body using eRAD PACS Viewer. The left and right psoas muscle areas (in cm^2)

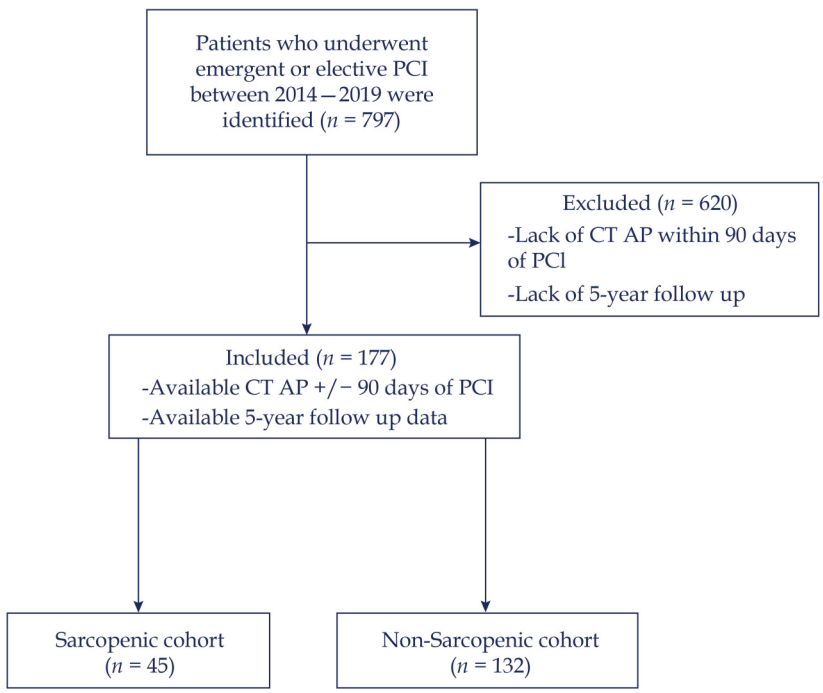

Figure 1 Exclusion criteria and cohort formation. Flow diagram of patient selection for the study cohort. A total of 797 patients who underwent emergent or elective PCI between 2014 and 2019 were identified. After excluding 620 patients due to lack of CT AP within ± 90 days of PCI or insufficient 5-year follow-up data. CT AP: computed tomography of the abdomen/pelvis; PCI: percutaneous coronary intervention.

were manually traced and summed, then divided by the patient's height in meters squared, yielding PMI in cm^2/m^2: (left psoas area + right psoas area) / $height^2$ (m^2). Informed consent was waived by the institutional IRB due to the retrospective nature of the study. As no universal PMI cutoff exists, sarcopenia was defined as the lowest sex-specific quartile, consistent with prior studies.[17] In our cohort, sarcopenia was defined as a PMI < 4.89 cm^2/m^2 for males and < 3.27 cm^2/m^2 for females. Patients below these thresholds were classified as sarcopenic. The distribution of PMI by sex, including percentile values and standard deviations, is summarized in Table 1.

Reproducibility of PMI Measurements

To assess measurement reliability, a subset of 30 patients was randomly selected for intra- and inter-observer reproducibility analysis. PMI was independently measured by two observers using axial CT images at the L3 vertebral level. One observer repeated the measurements after a two-week interval, blinded to initial results. Intra-class correlation coefficients (ICCs) were calculated using a two-way mixed-effects model for absolute agreement. The intra-observer ICC was 0.93 and the inter-observer ICC was 0.89, indicating adequate measurement reliability.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics Version 29.0.0.0 (IBM Corp. Released 2022. IBM SPSS Statistics for Windows, Version 29.0. Armonk, NY: IBM Corp). Baseline demographic and clinical characteristics of sarcopenic and non-sarcopenic patients were compared using independent *t*-tests for continuous variables and chi-square tests for categorical variables.

For the primary outcome of 5-year all-cause mortality and the secondary outcome of 3-point MACE, binary logistic regression was used to determine the independent predictors of these outcomes. Variables included in the regression models were sarcopenia status, sex, and emergent PCI status. Regression coefficients (B), standard errors (SE), Wald test statistics, *P*-values, and 95% confidence intervals (CI) for the odds ratios (ORs) are reported.

Cox proportional hazards regression analysis was employed to assess the impact of sarcopenia on time-to-event data for all-cause mortality. Hazard ratios (HR) with 95% CI were calculated, and significance was determined with the Wald test. Survival analysis was also performed using Kaplan-Meier curves to compare the time to death between sarcopenic and non-sarcopenic patients. All tests were two-sided, and *P*-values < 0.05 were considered statistically significant.

RESULTS

Cohort Demographics and Clinical Characteristics

The study included 177 patients who underwent PCI between 2015 and 2019, of whom 45 (25.4%) were classified as sarcopenic based on sex-specific PMI quartiles. The sarcopenic and non-sarcopenic groups were broadly similar across baseline demographic variables, cardiovascular comorbidities, procedural characteristics, and medication use.

While most variables were well balanced, there were non-significant trends toward higher diabetes prevalence (50.0% *vs.* 33.3%, $P = 0.067$) and hyperlipidemia (75.8% *vs.* 60.0%, $P = 0.065$) in the non-sarcopenic group. Procedural variables—including emergent PCI status, indication for PCI, stent burden, systolic blood pressure, and heart rate—did not significantly differ between groups. Cardiac medications at discharge were similarly prescribed. Full demographic and clinical data are provided in Table 2.

Clinical Outcomes

The primary outcome, 5-year all-cause mortality, was significantly higher in sarcopenic patients compared to non-sarcopenic patients (64.4% *vs.* 35.6%, $P < 0.001$).

Table 1 Distribution of PMI by Sex.

Sex	Q1 (25th percentile)	Q2 (Median)	Q3 (75th percentile)	Mean ± SD
Male	4.89 cm^2/m^2	5.94 cm^2/m^2	7.23 cm^2/m^2	5.97 ± 1.62 cm^2/m^2
Female	3.29 cm^2/m^2	4.33 cm^2/m^2	5.29 cm^2/m^2	4.27 ± 1.45 cm^2/m^2

This table displays the distribution of PMI values among male and female patients in the cohort. Male participants had higher PMI values across all quartiles compared to female participants, with a median PMI of 5.94 cm^2/m^2 (IQR: 4.89–7.23) and a mean ± SD of 5.97 ± 1.62 cm^2/m^2. In contrast, females had a lower median PMI of 4.33 cm^2/m^2 (IQR: 3.29–5.29) and a mean ± SD of 4.27 ± 1.45 cm^2/m^2. These values reflect expected sex-based differences in skeletal muscle mass. PMI: psoas muscle index.

Table 2 Cohort demographics, comorbidities, PCI characteristics, and cardiac medication use.

Category	All patients (177)	Sarcopenic (45)	Non-Sarcopenic (132)	*P*-value
Gender (male)	103 (58.2%)	26 (57.8%)	77 (58.3%)	0.948
Age, yrs	68.7	68.4	68.8	0.762
Pack years	19.0	19.7	18.8	0.829
Diabetes	81 (45.8%)	15 (33.3%)	66 (50%)	0.067
HTN	141 (79.7%)	32 (71.1%)	109 (82.6%)	0.156
HLD	127 (71.8%)	27 (60.0%)	100 (75.8%)	0.065
CKD	26 (14.7%)	4 (8.89%)	22 (16.7%)	0.220
CVA	19 (10.7%)	3 (6.7%)	16 (12.1%)	0.326
OSA	37 (20.9%)	8 (17.8%)	29 (21.9%)	0.593
CHF	36 (20.3%)	10 (22.2%)	26 (19.6%)	0.666
COPD	50 (28.2%)	11 (24.4%)	39 (28.8%)	0.627
AF	39 (22.0%)	9 (20.0%)	30 (22.7%)	0.753
Malignancy	37 (20.9%)	10 (22.2%)	27 (20.5%)	0.749
Emergent PCI	111 (62.7%)	26 (57.8%)	85 (64.4%)	0.428
Indication:				0.516
Stable Angina		19 (42.2%)	47 (26.6%)	0.061
Unstable Angina		4 (8.89%)	24 (13.6%)	0.554
NSTEMI		15 (33.3%)	42 (23.7%)	0.260
STEMI		7	19	0.523
Mean number of stents received	1.3	1.22	1.33	0.216
Mean systolic blood pressure (mmHg)	128.1	127.2	128.4	0.741
Pulse	75.4	77.1	74.8	0.399
ACEi/ARB	107	26	81	0.671
Beta Blocker	134	31	103	0.460
SGLT2i	50	10	40	0.778
MRA	26	10	16	0.098
High-intensity statin	165	41	124	0.515
Aspirin	135	33	102	0.592

Data are presented as *n* (%) unless other indicated. Baseline demographic and clinical characteristics of the study cohort, including gender, age, smoking history (pack-years), and the prevalence of comorbidities such as diabetes, HTN, HLD, CKD, CVA, OSA, CHF, COPD, AF, and malignancy. Procedural variables include PCI urgency (emergent vs. non-emergent), indication for PCI (stable angina, unstable angina, NSTEMI, and STEMI), mean systolic blood pressure, heart rate at the time of PCI, and the average number of stents placed. Medication use includes ACEi/ARB, beta blockers, SGLT2i, MRA, high-intensity statins, and aspirin. ACEi/ARB: angiotensin-converting enzyme inhibitors or angiotensin receptor blockers; AF: atrial fibrillation; CHF: congestive heart failure; CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease; CVA: cerebrovascular accident; HLD: hyperlipidemia; HTN: hypertension; MRA: mineralocorticoid receptor antagonists; NSTEMI: ST-elevation myocardial infarction; OSA: obstructive sleep apnea; SGLT2i: sodium-glucose cotransporter-2 inhibitors.

Rates of 3-point MACE were similar between groups, occurring in 55.6% of sarcopenic and 51.4% of non-sarcopenic patients ($P = 0.520$). No significant differences were observed in the individual MACE components, including non-fatal myocardial infarction and ischemic stroke. Full outcome data are presented in Figure 2.

Regression and Survival Analysis

Sarcopenia was not associated with MACE (OR = 1.00; 95% CI: 0.50–1.98; $P = 0.99$). However, sarcopenia was significantly associated with increased 5-year all-cause mortality (OR = 3.49; 95% CI: 1.69–7.19; $P = 0.0007$). Sex and emergent PCI status were not statistically significant predictors of mortality. Full data is summarized in Figure 3 and Tables 3-4.

In a multivariable Cox proportional hazards regression model for all-cause mortality, sarcopenic status was associated with increased hazard (HR = 1.60; 95% CI: 0.96–2.66; $P = 0.071$), although this did not reach statistical significance. Emergent PCI status (HR = 1.13; 95% CI:

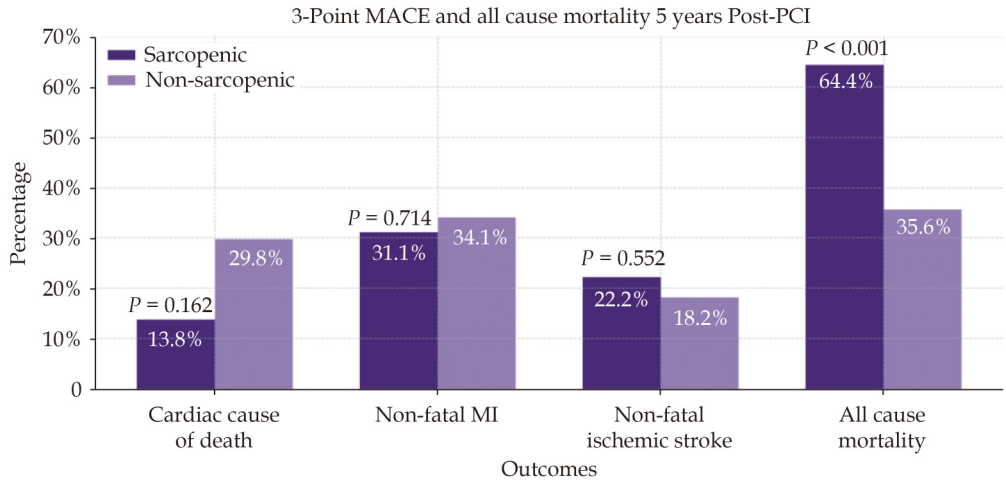

Figure 2 3-Point MACE and all-cause mortality 5 years post-PCI. The bar graph illustrates the percentage of patients experiencing cardiac-specific mortality, non-fatal MI, non-fatal ischemic stroke, and all-cause mortality within five years post-PCI. Bars are grouped by outcomes and color-coded by cohort (sarcopenic vs. non-sarcopenic). MACE: major adverse cardiovascular events; MI: myocardial infarction; PCI: percutaneous coronary intervention.

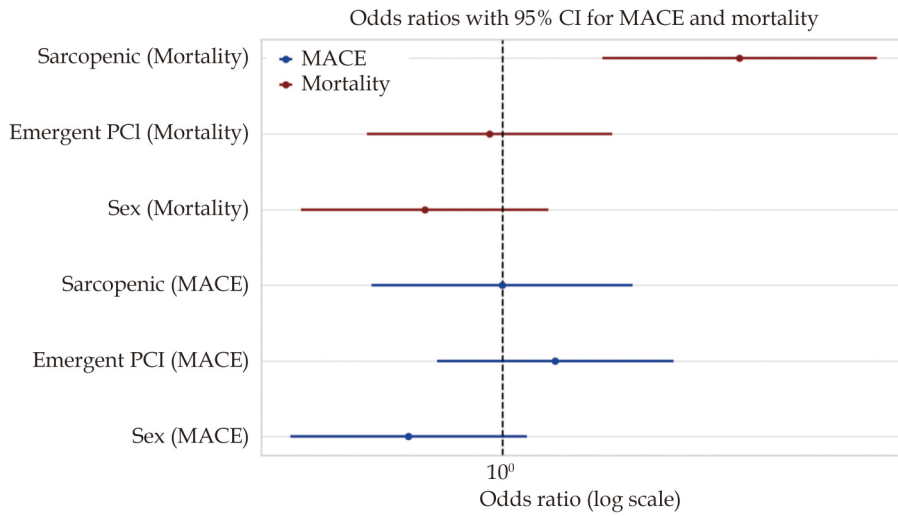

Figure 3 Forest plot of odds ratios for 5-year all-cause mortality and MACE. This figure displays odds ratios (ORs) and 95% CI for predictors of 5-year all-cause mortality (in red) and MACE (in blue) from binary logistic regression models. Sarcopenia was significantly associated with increased mortality (OR = 3.49), while emergent PCI and sex were not. In contrast, none of the variables—including sarcopenia—were significantly associated with MACE. The X-axis is shown on a logarithmic scale centered at OR = 1 (no effect). MACE: major adverse cardiovascular events; PCI: percutaneous coronary intervention.

Table 3 Binary logistic regression analysis for 5-year MACE.

Variable	OR	95% CI Lower	95% CI Upper	*P*-value
Intercept	1.2445	0.4648	3.332	0.6634
Sarcopenic	0.9965	0.5009	1.9822	0.9919
Emergent PCI	1.3179	0.7064	2.4588	0.3857
Sex	0.6082	0.3262	1.134	0.1177

This table summarizes the results of a binary logistic regression model evaluating predictors of 5-year MACE. Sarcopenia was not significantly associated with MACE (OR = 1.00; 95% CI: 0.50–1.98; $P = 0.9919$). Emergent PCI status and sex were also not statistically significant predictors. None of the variables demonstrated a meaningful association with MACE in this model. MACE: major adverse cardiovascular events; PCI: percutaneous coronary intervention.

Table 4 Binary logistic regression analysis for 5-year all-cause mortality.

Variable	OR	95% CI Lower	95% CI Upper	*P*-value
Intercept	0.7411	0.2653	2.0704	0.5676
Sarcopenic	3.4873	1.6922	7.1867	0.0007
Emergent PCI	0.9324	0.4879	1.7821	0.8324
Sex	0.6628	0.3452	1.2726	0.2165

This table presents the results of a binary logistic regression model assessing predictors of 5-year all-cause mortality. Sarcopenia was a significant predictor of mortality (OR = 3.49; 95% CI: 1.69–7.19; $P = 0.0007$). In contrast, emergent PCI status and sex were not significantly associated with mortality in this model. PCI: percutaneous coronary intervention.

0.68–1.88; $P = 0.649$), BMI > 30 kg/m^2 (HR = 0.82; 95% CI: 0.50–1.33; $P = 0.415$), and age (HR = 1.01; 95% CI: 0.98–1.05; $P = 0.428$) were not significantly associated with mortality. Please see Table 5 for full display of findings.

Kaplan-Meier survival analysis demonstrated significantly worse long-term survival among sarcopenic patients compared to their non-sarcopenic counterparts. The median follow-up duration in the overall cohort was 984.5 days (IQR: 442.8–1845.5). Median follow-up was shorter in the sarcopenic group (809.0 days; IQR: 350.0–1403.0) than in the non-sarcopenic group (1,244.0 days; IQR: 518.5–1993.5). A log-rank test indicated a statistically significant difference in survival between the two groups ($\chi^2 = 6.13$, $P = 0.0133$). Data presented in Figure 4 and Table 6.

DISCUSSION

Our study demonstrates that PMI derived sarcopenia has prognostic value regarding 5-year all-cause mortality in patients undergoing PCI. However, sarcopenic PMI did not significantly predict 3-point MACE over the same period. Additionally, sarcopenia did not emerge as an independent predictor of cardiac death in regression models. Overall, this data suggests that despite their increased frailty and overall worse prognosis, sarcopenic patients tolerate PCI similarly to their non-sarcopenic counterparts from a cardiovascular standpoint.

While our findings suggest sarcopenic PMI does not predict 3-point MACE, prior studies have reported differing results. Won, *et al.*[7] revealed a significantly higher incidence of 3-point MACE a year following PCI in patients identified as sarcopenic by low PMI. Mat-

Table 5 Cox regression analysis of overall mortality.

Variable	HR	SE	Wald	df	*P*-value	Exp(B)	95% CI Lower	95% CI Upper
Sarcopenic status	0.47	0.26	3.264	1	0.071	1.6	0.961	2.664
Emergent PCI	0.119	0.261	0.208	1	0.649	1.126	0.676	1.877
BMI_> 30	−0.203	0.248	0.666	1	0.415	0.817	0.502	1.329
Age	0.014	0.017	0.627	1	0.428	1.014	0.98	1.048

The table presents the regression coefficients (B), standard errors (SE), Wald chi-square statistics, degrees of freedom (df), significance levels (*P*-values), hazard ratios (Exp(B)), and 95% CI for sarcopenic status, emergent PCI status, BMI > 30 kg/m^2, and age. BMI: body mass index; PCI: percutaneous coronary intervention.

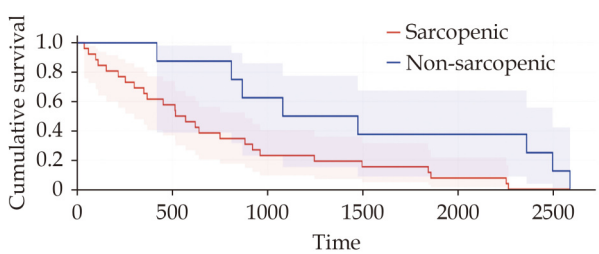

Figure 4 Kaplan-Meier survival curve. Kaplan-Meier survival curve comparing cumulative survival rates over time (in days) between sarcopenic and non-sarcopenic cohorts. The sarcopenic group is represented by the red line, while the non-sarcopenic group is represented by the blue line.

Table 6 Kaplan–Meier Survival Analysis Stratified by Sarcopenia Status.

Cohort	Median Follow-up (days)	IQR (days)
Sarcopenic	809.0	350.0–1403.0
Non-Sarcopenic	1244.0	518.5–1993.5
Overall Cohort	984.5	442.8–1845.5
Log-rank test	$\chi^2 = 6.13$	$P = 0.0133$

Median follow-up was shorter in sarcopenic patients compared to non-sarcopenic patients. A log-rank test demonstrated a statistically significant difference in survival distributions between the two groups ($\chi^2 = 6.13$, $P = 0.0133$), indicating worse long-term survival among sarcopenic patients. IQR: interquartile ranges. This table summarizes the median follow-up times and interquartile ranges (IQR) for sarcopenic and non-sarcopenic patients, as well as for the overall cohort.

sumoto, *et al*.[18] conducted a related study on non-ST elevation myocardial infarction (NSTEMI) patients and found that PMI was a significant predictor of both all-cause mortality and 3-point MACE within 2.4 years after PCI. These discrepancies may reflect differences in follow-up duration and patient populations. Shorter-term studies likely capture more immediate cardiovascular risk, whereas our 5-year follow-up may reflect broader frailty-related mortality. It's also possible that sarcopenic patients regain muscle mass over time, attenuating MACE risk.

These results have important implications for risk stratification and intervention choice in patients with CAD. Skeletal muscle mass plays a critical role in recovery following major surgery, and multiple sarcopenia metrics have been linked to an increased risk of early mortality after coronary artery bypass grafting (CABG).[8,19-21] Present risk stratification tools, such as the Synergy Between PCI With Taxus and Cardiac Surgery (SYNTAX) score and the European System for Cardiac Operative Risk Evaluation (EUROscore), do not include sarcopenic metrics.[22] Our data suggests that, while sarcopenic patients have worse overall outcomes post-PCI, their cardiovascular complication rates are similar to those without sarcopenia.

Assessing sarcopenia via PMI may help identify CAD patients who would benefit from intensive nutritional and physical rehabilitation. Cardiac rehabilitation (CR) following both elective and emergent PCI is critical for recovery. However, patients with sarcopenia may face barriers to full participation in CR due to reduced muscle mass.[23] A recent study by Shukuta, *et al*.[24] found that sarcopenic patients with heart failure who were unable to improve their sarcopenic status during CR had a higher risk of all-cause mortality. The inability to participate effectively in CR likely exacerbates muscle loss, perpetuating the cycle of frailty.[25] Utilizing PMI as an objective measure of sarcopenic status could facilitate pre- and post-PCI optimization.

Further research should focus on investigating the potential for recovery of psoas muscle area following PCI and its correlation with long-term outcomes. Given the discrepancy in our findings and those of Won *et al*.,[7] it would be valuable to explore whether PCI allows for longitudinal improvements in muscle mass which ultimately translates to a reduction in 3-point MACE. Furthermore, studying the prognostic value of PMI in CABG patients could allow for future inclusion of sarcopenia as a risk stratification tool when delineating between intervention modalities.

Several limitations of this study must be acknowledged. First, the retrospective design inherently introduces potential biases, including selection bias and the reliance on pre-existing data, which may affect the generalizability of our findings. The relatively small sample size may have limited the statistical power, preventing some trends from reaching statistical significance. Additionally, as our cohort was drawn from a single center, the broader applicability of these results to different populations and healthcare settings remains limited.

In conclusion, PMI is a significant predictor of 5-year all-cause mortality in patients undergoing PCI, though it does not independently predict 3-point MACE. These findings highlight the prognostic importance of evaluating skeletal muscle mass in this population, suggesting that PMI may enhance risk stratification and guide post-operative management strategies. Further research is warranted to explore the potential for PMI recovery after PCI and its impact on long-term outcomes, which may open avenues for targeted rehabilitation to improve survival in sarcopenic patients.

DISCLOSURE

Data Availability

The data supporting the findings of this study were obtained from UPMC and are not publicly available due to institutional policies and privacy regulations. Access to the data may be granted upon reasonable request and with permission from UPMC. For inquiries regarding data access, please contact hickeygw@upmc.edu.

Conflict of Interest

None.

Ethics Approval

This study was conducted in accordance with the ethical standards of the UPMC Institutional Review Board Name and the Declaration of Helsinki. Ethical approval was obtained from UPMC under approval number 18120143.

Patient Consent

A waiver of informed consent was approved by UPMC IRB due to the retrospective nature of the study.

Clinical Trial Registration

This study was not registered as a clinical trial as it

does not fall under the category of interventional studies requiring registration.

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