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ORIGINAL RESEARCH

Prognostic Value of Psoas Muscle Mass Index in Patients With Non–ST-Segment–Elevation Myocardial Infarction: A Prospective Observational Study

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BACKGROUND: Muscle wasting is an important predictor of long-term outcome in patients with cardiovascular disease, but the prognostic value of muscle wasting in patients with non–ST-segment–elevation myocardial infarction is not established. The aim of this study is to investigate the prognostic value of muscle wasting, defined by psoas muscle mass index (PMI), in patients with non–ST-segment–elevation myocardial infarction.

METHODS AND RESULTS: A total of 132 consecutive patients with non–ST-segment–elevation myocardial infarction were prospectively enrolled between 2015 and 2018. Primary end point was incidence of cardiovascular events including cardiovascular deaths, non-fatal myocardial infarction, or non-fatal stroke. Cross-sectional area of the psoas muscle at the L3 vertebral level was obtained by computed tomography and PMI was calculated. The median follow-up period was 2.4 years (interquartile range, 1.1–4.0 years). There were 45 cardiovascular events (34%) during the study periods. The optimal cutoff value of PMI to predict cardiovascular events was 772 mm^2/m^2, as assessed by receiver operating curve analysis. Patients with reduced PMI (PMI<772 mm^2/m^2) had significantly higher cardiovascular events than those with preserved PMI (PMI≥772 mm^2/m^2) (48% versus 21%; log-rank test $P<0.001$). Multivariate Cox proportional hazards model revealed that reduced PMI was a statistically significant predictor of cardiovascular events (hazard ratio, 3.30; 95% CI, 1.70–6.40; $P<0.001$).

CONCLUSIONS: Muscle wasting defined as PMI is a simple and useful objective marker to predict future cardiovascular outcome in patients with non–ST-segment–elevation myocardial infarction.

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Key Words: acute myocardial infarction ■ muscle wasting ■ psoas muscle ■ sarcopenia

Long-term prognosis of acute myocardial infarction (AMI) remains poor, despite the advancement of contemporary treatment strategies including early invasive revascularization, intensive risk factor management, and multidisciplinary treatment.[1,2] Because of the lower incidence of total coronary occlusion and smaller infarct size, non–ST-segment–elevation myocardial infarction (NSTEMI) has less myocardial injury compared with ST-elevation AMI.[3,4] On the other hand, worse long-term prognosis has been reported in patients with NSTEMI compared with ST-elevation AMI, because patients with NSTEMI are older, have higher incidence of geriatric condition including muscle wasting, sarcopenia, or frailty as well as greater comorbidity which strongly affects long-term prognosis rather than infarct myocardial damage.[2,3,5-8]

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CLINICAL PERSPECTIVE

What Is New?

- Reduced psoas muscle mass index was a statistically significant predictor of cardiovascular events in patients with non–ST-segment–elevation myocardial infarction.
- The addition of reduced psoas muscle mass index improved the prognostic capacity of the prediction model.

What Are the Clinical Implications?

- Muscle wasting defined by psoas muscle mass index is a useful measure to predict future cardiovascular events beyond conventional cardiac prognostic markers in non–ST-segment–elevation myocardial infarction.

Nonstandard Abbreviation and Acronym

PMI	Psoas muscle mass index

Muscle wasting is a significant medical problem in patients with cardiovascular disease and is positively affected by adequate interventional managements such as rehabilitation and nutritional treatment.[9] Therefore, early diagnosis of this condition is important to improve survival. However, relationship between muscle wasting and NSTEMI has not been elucidated. Psoas muscle mass index (PMI) has been growing recognition as an objective and quantitative marker to assess muscle wasting and has been suggested as a useful predictive marker for long-term outcome after cardiovascular surgery and surgical oncology.[10,11] Accordingly, we investigated prognostic value of muscle wasting by measuring PMI in patients with NSTEMI.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Population

Three-hundred sixty-one consecutive patients with AMI admitted to Kansai Medical University Hospital between January 2015 and April 2018 were prospectively enrolled. Patients with ST-elevation AMI (n=165), out-of-hospital cardiac arrest (n=52), malignant disease with life expectancy ≤1 year (n=4) and loss of follow-up (n=8) were excluded. Thus, 132 patients were included in the final analysis. The study protocol was approved by the ethics committee of Kansai Medical University (No.20130181) and was registered at the University Hospital Medical Information Network Clinical Trial Registry (Unique identifier: UMIN000013445). All patients gave written informed consent. The investigation conforms with the principles outlined in the 1975 Declaration of Helsinki.

Study Protocol and Treatment

Diagnosis of NSTEMI was made by chest discomfort or ischemic-equivalent symptoms at rest, laboratory assay of troponin I or creatine kinase-myocardial band > upper limit of normal and no persistent ST-segment elevation.[12,13] Patients with alternative diagnosis after coronary angiography (eg, Takotsubo cardiomyopathy, coronary vasospasm, myocarditis, and pulmonary embolism) were not included in this study. All patients underwent guideline-directed medical management and coronary angiography.

Data Collection

Patient characteristics and past medical history were obtained from the medical records. Laboratory parameters were collected on admission and cardiovascular status was assessed using the Killip classification. Echocardiography was performed by a cardiologist on admission using Vivid 7 or Vivid E9 (GE Healthcare, Marlborough, MA, USA). Left ventricular ejection fraction (LVEF) was obtained by the Simpson method. Culprit vessel and number of coronary artery disease were assessed by coronary angiography.

Analysis of Psoas Muscle Mass Index

Plain abdominal computerized tomography (CT) with 5-mm slice imaging was performed during the hospitalization using 80 (Aquilion Prime, Canon, Japan) or 64 (Somatom Perspective, Siemens, Germany) multidetector CT. Cross-sectional area of psoas muscle (mm^2) and CT value (Hounsfield units) were measured using a dedicated workstation (Virtual Place, AZE Ltd, Japan). Both left and right cross-sectional area of the psoas muscle at the L3 vertebral level were traced semi-automatically by 2 independent physicians who were masked to the clinical history.[14,15] PMI (mm^2/m^2) was calculated by body muscle cross-sectional area divided by body surface area.[14] Mosteller formula was used to obtain body surface area: body surface area (m^2)=(height (cm)×weight (kg)/3600)$^{1/2}$.

End point and Follow-up

Primary end point was cardiovascular event including cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke. Secondary end point was all-cause death and cardiovascular death. The incidence of events was identified from the medical records or

mailed questionnaire to the follow-up hospital. For patients experiencing >1 acute event, only the first event was considered in the analysis.

Statistical Analysis

Continuous variables are presented as means±SDs or medians with interquartile ranges. Categorical variables are presented as number of total (percentages). Equality of means between the 2 groups were tested using the Student *t*-test or the Mann-Whitney *U* test for continuous variables and the Chi-square test for categorical variables. Inter-observer validity of 2 independent readers for psoas size measurements was assessed by Pearson correlation. To obtain the optimal cut-off value of PMI to predict cardiovascular event, the receiver operating characteristic curve analysis was performed. Long-term cardiovascular event was illustrated by Kaplan-Meier survival analysis and compared using log-rank test. All secondary end-point comparisons were performed at α=0.05 without adjustment for multiplicity. Univariate and multivariate Cox proportional hazards models were conducted using conventional risk factors for AMI death and variables with significant difference.[16] Net reclassification improvement and integrated discrimination index were calculated to evaluate the quality of improvement of the PMI to the model. The JMP 14.2.0 software (SAS Institute Inc., Cary, NC, USA) and R 3.6.3 with additional packages, including Rcmdr, Epi, pROC, and Predict ABEL were used for all statistical analyses. A *P* value <0.05 was considered significant.

RESULTS

Baseline Characteristics

During the median follow-up of 2.4 years (interquartile range, 1.1–4.0 years), 45 patients (34%) experienced cardiovascular events. Lower hemoglobin and serum albumin levels, and higher serum creatinine and high-sensitivity C-reactive protein levels were observed in patients who had cardiovascular events (Table 1). Although there were no significant differences in coronary angiographic characteristics between the 2 groups, patients with cardiovascular events had a significantly higher rate of LVEF<50% and a significantly lower rate of revascularization therapy compared with those without cardiovascular events.

Table 1. Baseline Clinical and Coronary Angiographic Characteristics

	Cardiovascular event		*P* Value
	Absent (n=87)	Present (n=45)	
Age, y	73 (66–77)	74 (70–80)	0.14
Men	65 (75%)	32 (71%)	0.66
Body mass index, kg/m^2	23.7 (21.4–25.7)	22.1 (19.6–24.5)	0.02
Hypertension	61 (70%)	30 (67%)	0.69
Hyperlipidemia	69 (79%)	26 (58%)	0.01
Diabetes mellitus	51 (59%)	24 (53%)	0.56
Past smoking	61 (70%)	32 (71%)	0.91
Prior myocardial infarction	17 (20%)	11 (24%)	0.52
Laboratory parameters			
Hemoglobin, g/dL	13.2 (11.4–14.7)	11.8 (10.6–13.0)	0.0001
Serum creatinine, mg/dL	1.0 (0.7–1.3)	1.3 (0.8–2.8)	0.01
Serum albumin, g/dL	4.0 (3.6–4.3)	3.6 (3.3–4.0)	0.002
Cardiac troponin I, ng/mL	0.5 (0.1–4.6)	1.4 (0.3–7.7)	0.13
High-sensitivity CRP, mg/dL	0.21 (0.07–0.71)	0.60 (0.20–2.92)	0.003
LVEF, %	56 (46–65)	48 (36–65)	0.06
LVEF<50%	29 (33)	25 (56)	0.01
Killip class			0.39
1	54 (62%)	21 (47%)	
2	12 (14%)	8 (18%)	
3	13 (15%)	9 (20%)	
4	8 (9%)	7 (16%)	
Culprit lesion			0.08
LMT	14 (16%)	6 (13%)	
LAD	23 (26%)	18 (40%)	
LCX	20 (23%)	14 (31%)	
RCA	30 (34%)	7 (16%)	
Multiple vessel disease	64 (74%)	33 (73%)	0.98
IABP	19 (22%)	9 (20%)	0.81
Revascularization	83 (95%)	38 (84%)	0.04

Data presented as median (25th to 75th percentiles), or number (%). BP, blood pressure; CRP, C-reactive protein; HDL, high-density lipoprotein; IABP, intra-aortic balloon pumping; LAD, left anterior descending artery; LCX, left circumflex artery; LDL, low-density lipoprotein; LMT, left main trunk; LVEF, left ventricular ejection fraction; RCA, right coronary artery.

Psoas Muscle Assessments

The mean PMI was physician 1, 804 ± 231 mm^2/m^2; and physician 2, 806 ± 230 mm^2/m^2. The mean CT value was physician 1, 34.5 ± 9.2 Hounsfield units; and physician 2, 35.5 ± 9.2 Hounsfield units. Excellent correlation of PMI was shown by 2 observers (Figure 1). Psoas muscle mass and PMI were significantly higher in patients without cardiovascular events, whereas there was no significant difference in CT value between the 2 groups (Table 2). The optimal cut-off value of PMI to predict cardiovascular events was 772 mm^2/m^2 (sensitivity, 67%; specificity, 63%; area under the curve, 0.637). When the studied patients were classified into 2 groups according to PMI; preserved PMI (PMI ≥772 mm^2/m^2; n=70)

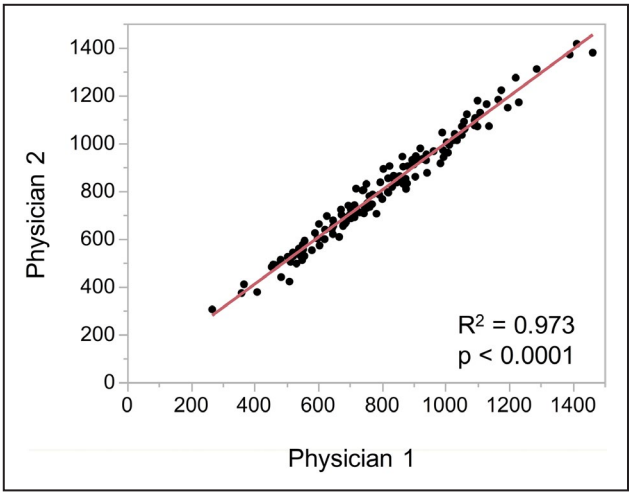

Figure 1. Correlation coefficient analysis.
Psoas muscle mass index measurements identified excellent correlation coefficient between physician 1 and physician 2.

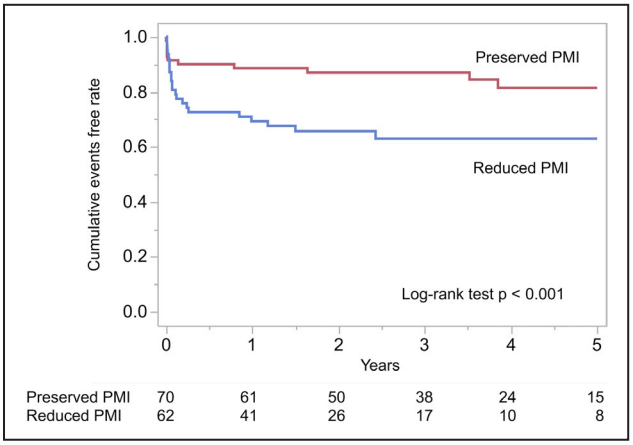

Figure 2. Kaplan-Meier estimates of cardiovascular events based on psoas muscle mass index.
The cut-off values defining the psoas muscle mass index was 772 mm^2/m^2. Blue line indicates patients with PMI≥772 mm^2/m^2; red line indicates patients with PMI<772 mm^2/m^2. PMI indicates psoas muscle mass index.

and reduced PMI (PMI<772 mm^2/m^2; n=63), cardiovascular events were significantly higher in patients with reduced PMI than those with preserved PMI (48% versus 21%, log-rank test, *P*<0.001, Figure 2). In subgroup analysis according to LVEF, predictive value of reduced PMI (PMI<772 mm^2/m^2) for cardiovascular events improved in patients with LVEF≥50% (sensitivity, 90%; specificity, 64%; area under the curve, 0.769), whereas worsened in patients with LVEF<50% (sensitivity, 48%, specificity, 62%, area under the curve, 0.550). Cardiovascular death and all-cause death were significantly higher in patients with reduced PMI (Table 3).

Prognostic Value of PMI

Univariate and multivariate Cox proportional hazard models revealed that PMI<772 mm^2/m^2 was an independent predictor for cardiovascular events (hazard ratio, 3.30; 95% CI, 1.70–6.40; *P*<0.001, Table 4) Model fit and discrimination improvement was evaluated by adding PMI<772 mm^2/m^2 to the known predictors including age, male sex, hypertension, hyperlipidemia, diabetes mellitus, hemoglobin, serum albumin, and serum creatinine. As a result, net reclassification improvement and integrated discrimination index significantly improved by adding PMI<772 mm^2/m^2 (Table 5).

DISCUSSION

This prospective observational study investigated the prognostic value of muscle wasting using PMI for cardiovascular events in patients with NSTEMI. When the studied patients were classified by PMI=772 mm^2/m^2, patients with reduce PMI had significantly higher incidence of cardiovascular events than those with preserved PMI. Multivariate analysis revealed that reduced PMI was statistically significant predictor for cardiovascular events. Moreover, the prediction model by adding reduced PMI improved prognostic capacity in patients with NSTEMI.

Recently, age-related skeletal muscle alterations recognized as sarcopenia or frail have become an crucial issue for patient management because elderly patients admitted to the hospital have increased remarkably worldwide.[2] Age-related skeletal muscle alterations is progressive syndrome and skeletal muscle is lost earlier than fat tissue and weight loss indicating

Table 2. Psoas Muscle Assessment

	Cardiovascular event		
	Absent (n=87)	Present (n=45)	*P* Value
Cross sectional psoas muscle area, mm^2	1404 ± 48	1170 ± 67	0.005
PMI, mm^2/m^2	840 ± 229	743 ± 220	0.02
CT value (Hounsfield unit)	35.8 ± 8.9	33.2 ± 9.2	0.13

Data presented as mean ± SD. CT indicates computed tomography; and PMI, psoas muscle mass index.

Table 3. Comparison of Primary and Secondary End Points According to PMI

	Reduced PMI (n=62)	Preserved PMI (n=70)	*P* Value
Cardiovascular events	30 (48%)	15 (21%)	<0.001
Cardiovascular death	22 (35%)	11 (16%)	0.006
All-cause death	28 (45%)	15 (21%)	0.002

Data presented as number (%). Significance was assessed by the log-rank test. PMI indicates psoas muscle mass index.

Table 4. Univariate and Multivariate Cox Proportional Hazards Model for Cardiovascular Events

	Univariate			Multivariate		
	HR	95% CI	*P* Value	HR	95% CI	*P* Value
Age, y	1.02	0.99–1.06	0.19			
Men	0.88	0.46–1.68	0.71			
Hypertension	0.78	0.42–1.46	0.45			
Hyperlipidemia	0.44	0.24–0.80	<0.01	0.49	0.27–0.90	0.02
Diabetes mellitus	0.79	0.44–1.43	0.44			
Smoking	0.94	0.49–1.79	0.85			
Hemoglobin, g/dL	0.80	0.71–0.90	<0.001	0.87	0.73–1.03	0.10
Serum creatinine, mg/dL	1.08	0.98–1.17	0.10			
Serum albumin, g/dL	0.40	0.25–0.66	<0.001	0.71	0.36–1.40	0.33
High-sensitivity CRP, mg/dL	1.18	1.07–1.27	0.001	1.10	0.99–1.21	0.06
LVEF<50%	2.27	1.26–4.09	0.006	1.81	0.99–3.33	0.06
Killip class 4	2.00	0.89–4.50	0.12			
Revascularization	0.34	0.15–0.76	0.02	0.18	0.07–0.43	<0.001
Reduced PMI	2.84	1.52–5.30	<0.001	3.32	1.73–6.39	<0.001

CRP indicates C-reactive protein; HR, hazard ratio; LVEF, left ventricular ejection fraction; and PMI, psoas muscle mass index.

that muscle wasting precedes frail.[17] Importantly, muscle wasting predominantly affects postural rather than non-postural muscles. Therefore, we have to detect postural muscle reduction as earlier as possible in patients with cardiovascular disease to prevent subsequent development of sarcopenia and frail.

Several methods have been advocated to assess muscle wasting, although gold standard is not established. Quantitative assessment of skeletal muscle mass by imaging including dual energy X-ray absorptiometry, ultrasonography, CT, or magnetic resonance imaging has become a simple method to determine low skeletal muscle mass.[18] Each approach has advantages and disadvantages to estimate low skeletal muscle mass. Among these approaches, psoas muscle mass assessed by CT imaging has several advantages to evaluate low skeletal muscle mass. CT imaging is a simple and quantitative method to assess psoas muscle because psoas muscle independently positions from other hip and spinal muscles. Psoas muscle is the main flexor of the hip which connects between upper and lower body trunk. Psoas muscle also provides postural support of lumbar spine, sacroiliac and hip joint which links directly to physical performance in a daily life.[18] Therefore, decrease in psoas muscle mass can precisely discriminate low postural muscle.

There is a growing body of evidence that preoperative psoas muscle mass assessed by abdominal CT imaging is a useful predictive marker for long-term outcome in the surgical field.[10,11] In contrast, prognostic value of psoas muscle mass in cardiovascular disease including AMI is unknown. In this study, we found that PMI obtained by abdominal CT had excellent inter-observer validity and was an independent predictor of cardiovascular event in patients with NSTEMI. These data indicate that PMI is a useful method to predict future cardiovascular events beyond conventional cardiac prognostic markers in NSTEMI. Interestingly, value of PMI to predict cardiovascular event was more accurate in patients with preserved LVEF than those with reduced LVEF. This implies that patients with preserved LVEF was related to muscle wasting rather than ischemic damage attributable to AMI for clinical prognosis. Therefore, more careful evaluation of muscle wasting should be provided in NSTEMI patients with smaller ischemic damage.

Table 5. Effect of Adding Psoas Muscle Mass Index to Predict Cardiovascular Event

	C-statistics	95% CI	*P* Value	NRI	95% CI	*P* Value	IDI	95% CI	*P* Value
Baseline model	0.73	0.65–0.82	Ref.			Ref.			Ref.
Baseline model+ reduced PMI	0.76	0.68–0.84	0.30	0.51	0.16–0.86	0.004	0.07	0.02–0.11	0.005

Baseline model included age, male sex, hypertension, hyperlipidemia, diabetes mellitus, hemoglobin, serum albumin, and serum creatinine. IDI, integrated discrimination index; NRI, net reclassification improvement; and PMI, psoas muscle mass index.

Limitations

Three limitations of our study should be addressed. First, the study population was relatively small in size. Thus, the findings need to be confirmed in a larger population. Second, there is a risk of patient selection bias because elderly patients with high comorbidity may have been excluded from revascularization therapy. Previous large trials have suggested that frail older patients with AMI are less likely to receive revascularization and had a higher in-hospital mortality rate as compared with non-frail patients with AMI.[19,20] Moreover, severe comorbid conditions could be considered potential contraindication to invasive procedures in the AMI patients. Third, sarcopenia or frailty parameters such as muscle strength (eg, handgrip) or muscle performance (eg, gait speed) were not systemically measured in our study. Further study is warranted to assess PMI with conventional sarcopenia or frailty parameters in patients with AMI. Nonetheless, to the best of our knowledge, this is the first report to assess the relationship between muscle wasting determined by PMI and cardiovascular event in patients with NSTEMI.

CONCLUSIONS

Muscle wasting defined by PMI is a simple and useful objective marker of future cardiovascular event in patients with NSTEMI.

ARTICLE INFORMATION

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Disclosures

None.

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