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Association between sarcopenia index, intraoperative events and post-discharge mortality in patients undergoing percutaneous coronary intervention: a retrospective cohort study in a teaching hospital in Western China

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ABSTRACT

Objectives To examine the association between the sarcopenia index (SI) and the risk of intraprocedural events and post-discharge death during percutaneous coronary intervention (PCI).

Design A retrospective cohort study.

Setting The study was conducted at a teaching hospital in Western China.

Participants The participants were patients aged 45 years and older who underwent PCI at the hospital and had an estimated glomerular filtration rate (eGFR) of ≥15 mL/min/1.73 m^2. Patients who died during hospitalisation, as well as those with unknown death dates, those lost to follow-up and those with missing information for the SI calculation, were excluded.

Primary and secondary outcome measures The SI was calculated as serum creatinine/cystatin C (Cr/CysC) × 100. The high-SI group was defined as the highest quartile, while the remaining participants were included in the low-SI group. Intraprocedural events included intraprocedural coronary slow flow (CSF)/coronary artery no-reflow (CNR) and malignant ventricular arrhythmia (MVA). In the event of death, the date of death was recorded.

Results The study included 497 patients who underwent PCI in our hospital, of whom 369 (74.25%) were males. A total of 57 (11.47%) patients developed CSF, 100 (20.12%) developed CNR and 4 (0.8%) developed MVA. Forty-four (8.85%) patients died post-discharge. The proportion of patients in the low-SI group who developed CSF was higher than those who did not (16.94% vs 9.65%, p=0.027). In addition, the average SI was lower in patients who developed CSF than in those who did not (81.99 vs 87.11, p=0.043). After adjusting for possible confounding factors, logistic regression analysis showed that the risk of CSF in the low-SI group was higher than that in the high-SI group (OR = 2.01, 95% CI: 1.04 to 3.89). In addition, it was found that the lower the SI, the higher the risk of CSF (OR = 0.983, 95% CI: 0.967 to 0.999).

Conclusions Patients with lower SI had a greater risk of developing CSF, and the lower the SI, the higher the risk of CSF. However, these data suggest that SI is not associated with CNR and the risk of post-discharge death in patients after PCI.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ Use of sarcopenia index (SI) as a screening indicator for sarcopenia to predict disease prognosis.
⇒ Focus on intraoperative events and post-discharge mortality in percutaneous coronary intervention (PCI) patients.
⇒ This is a single-centre retrospective analysis.
⇒ The study population included is relatively small.
⇒ No further study was conducted based on the length of time from PCI to death and the cause of death.

INTRODUCTION

Coronary artery disease (CAD) is among the most common causes of morbidity and mortality throughout the world.[1] Percutaneous coronary intervention (PCI) is one of the key treatments for patients with CAD. It can also reduce angina symptoms, improve the patient's quality of life and enhance their functional capability.[1] The development of PCI over more than 40 years has seen significant improvements in its safety and efficacy.[2] However, PCI intraprocedural events (including intraprocedural coronary slow flow (CSF)/coronary artery no-reflow (CNR), malignant ventricular arrhythmia (MVA)) and high postoperative mortality rates post-discharge from the hospital remain important clinical issues associated with this procedure. The incidence of CSF/CNR ranges from 11.1 to 28.8%[3–7] and is closely associated with poor clinical outcomes.[3 7] MVA occurs in 2.8% of cases and may lead to sudden cardiac death.[8] In addition, the risk of post-discharge death in patients after PCI is also a concern.[9–11]

Sarcopenia has been linked to increased rapidity of cardiovascular disease progression, mortality, increased fall risk and reduced quality of life, especially in older adults.[12] Sarcopenia is defined by reduced muscle mass together with decreased muscular strength and/or physical function.[13] While an assessment of muscle mass is necessary for the diagnosis of this disease, this requires instrumentation (such as InBody and CT), which may increase the hospitalisation costs and radiation risks for patients admitted with acute CAD. In addition, muscle strength and physical function tests need to be completed. Muscle strength is generally evaluated by testing the grip strength of both hands, whereas physical function is generally evaluated based on 6-metre walking speed or the completion of five sit-ups. For patients with CAD, these tests may also be associated with a risk of worsening their condition, and patients may be reluctant to cooperate due to physical discomfort.

The sarcopenia index (SI) is a surrogate marker of sarcopenia. The SI is defined as the ratio of serum creatinine (Cr) to serum cystatin C (CysC).[14] Serum Cr and CysC are two commonly used indicators of renal function. The loss of skeletal muscle during muscle atrophy is accompanied by a decrease in the serum Cr level, as Cr is released from the skeletal muscle, while serum CysC is produced by all nucleated cells and is therefore relatively less affected.[15] Therefore, a low Cr/CysC ratio can be used as a marker for low skeletal muscle mass.[15] Furthermore, multiple studies have shown that the Cr/CysC index can predict adverse clinical outcomes, such as mortality in patients diagnosed with non-dialysis-treated chronic kidney diseases,[15] as well as 3 year mortality in hospitalised patients,[16] and all-cause mortality in those with obstructive CAD.[17] The SI can thus serve as a surrogate indicator in patients with acute CAD without the need for additional examinations or financial expenditure. The SI is also strongly associated with poor outcomes, including major adverse cardiovascular events (MACE) and both cardiovascular and non-cardiovascular-related death, in patients after PCI.[18 19]

To date, there has been no exploration of the relationship between the SI and PCI-associated intraprocedural events (CSF/CNR, MVA) or postoperative post-discharge death. Therefore, the purpose of this study was to evaluate this relationship based on the hypothesis that the SI is related to the risk of PCI intraprocedural events and/or postoperative post-discharge death.

METHODS

Study design and patients

This was a retrospective observational study, conducted between 1 February 2016 and 31 December 2022, at a single teaching hospital in Western China. Patients were included if they were aged 45 years and older, had undergone PCI and had an estimated glomerular filtration rate (eGFR) of ≥15 mL/min/1.73 m^2. Patients were excluded if they died in-hospital, if their time of death was not available, if they were lost to follow-up or if variables used to calculate the SI were missing.

Ethics

The protocol for this retrospective study was overseen by the Centre for Health Informatics, which also ensured that all relevant information remained anonymous and that the data remained confidential at all times. No patient consent was required to review the medical records used for this study as these analyses were retrospective in nature. This study received approval from the Research Ethics Committee of the Affiliated Traditional Chinese Medicine Hospital of Southwest Medical University (No. BY2023032) and was consistent with the Declaration of Helsinki.

Intraprocedural events

Intraprocedural events were collected based on clinician diagnoses recorded in the medical record system. For this study, the relevant intraprocedural events were CSF/CNR and MVA. The TIMI grading method was used to determine CSF/CNR, using a grade 0 for CNR, 1–2 for CSF and 3 for normal blood flow status.

Post-discharge death

Telephone interviews were conducted from 9 May2023 to 16 May 2023 to obtain information regarding patient mortality (survival status and time of death). The overall survival of a patient refers to the interval between discharge and death from any cause or the last known date of survival.

SI

As reported previously, the SI was calculated by dividing serum Cr levels by CysC levels and multiplying by 100.[14] The highest quartile SI value was then used to separate patients into two groups, with individuals in the 'low SI' group being all patients with scores below this quartile and all other patients being categorised in the 'high SI' group.[20]

Covariates

The clinical parameters of the patients were considered as possible covariates. These included age, sex, the reasons for PCI, time from onset to PCI, number of stent implants, smoking history, drinking history, heart rate, ejection fraction (EF), left ventricular end-systolic diameter (LVESD), left ventricular end-diastolic diameter (LVEDD), eGFR, chronic diseases and blood test results. Chronic diseases included hypertension, diabetes, pulmonary disease and arrhythmia. The blood test results used in the analysis included preoperative levels of low-density lipoprotein (LDL), high-density lipoprotein (HDL), total cholesterol (TC) and albumin (ALB), as well as the prothrombin time (PT), fibrinogen degradation product (FDP) and activated partial thromboplastin time (APTT). Details of the normal threshold levels of HDL, LDL, TC, PT, APTT, FDP and ALB are provided in the Supplementary Materials (online supplemental table S1). Indicators

Table 1 Baseline characteristics of participants according to the intraoperative events

Characteristics	CSF		P value
	No (n=440)	Yes (n=57)	
Age years, median (IQR)	65 (55.25, 73)	64(55, 74)	0.687
Sex, n (%)			0.132
Male	322 (73.18)	47 (82.46)	
Female	118 (26.82)	10 (17.54)	
The reasons for PCI, n (%)			>0.99
Stable angina pectoris	12 (2.73)	2 (3.51)	
ACS	428 (97.27)	55 (96.49)	
Time from onset to PCI, hour, median (IQR)	11 (4, 40)	8 (4, 29.5)	0.098
Number of stent implants, n (%)			0.748
1	231 (52.5)	27 (47.37)	
2–3	178 (40.45)	26 (45.61)	
>3	31 (7.05)	4 (7.02)	
Smoking history, n (%)			0.807
No	185 (42.05)	23 (40.35)	
Yes	255 (57.95)	34 (59.65)	
Drinking history, n (%)			0.115
No	237 (53.86)	37 (64.91)	
Yes	203 (46.14)	20 (35.09)	
Heart rate, median (IQR)	75 (65, 88)	78 (69, 90)	0.099
Hypertension, n (%)			0.655
No	187 (42.5)	26 (45.61)	
Yes	253 (57.5)	31 (54.39)	
Diabetes, n (%)			0.564
No	324 (73.64)	44 (77.19)	
Yes	116 (26.36)	13 (22.81)	
Pulmonary disease, n (%)			0.987
No	394 (89.55)	51 (89.47)	
Yes	46 (10.45)	6 (10.53)	
Arrhythmia, n (%)			0.557
No	373 (84.77)	50 (87.72)	
Yes	67 (15.23)	7 (12.28)	
EF%, median (IQR)	62 (55, 65)	61 (50.75, 64.25)	0.36
LVESD, mm, median (IQR)	31 (29, 35)	33 (30, 39)	0.023
LVEDD, mm, median (IQR)	47 (44, 50)	49 (47, 51)	0.004
eGFR, ml/min/1.73m^2, median (IQR)	70.93 (56.83, 86.08)	69.49 (56.24, 89.54)	0.954
eGFR, ml/min/1.73m^2, n (%)			0.582
<60	131 (29.77)	19 (33.33)	
≥60	309 (70.23)	38 (66.67)	
HDL, mmol/l, median (IQR)	1.15 (0.97, 1.38)	1.1 (0.97, 1.31)	0.435
LDL, mmol/l, median (IQR)	3.01 (2.42, 3.65)	3.13 (2.7, 4.03)	0.126
TC, mmol/l, median (IQR)	4.64 (4, 5.43)	4.7 (4.14, 5.7)	0.263
ALB, g/l, median (IQR)	41.2 (38.6, 43.7)	42.2 (39.9, 43.65)	0.183
ALB, g/l, n (%)			0.038
Normal	270 (61.36)	43 (75.44)	
Abnormal	170 (38.64)	14 (24.56)	

Continued

Table 1 Continued

Characteristics	CSF		P value
	No (n=440)	Yes (n=57)	
PT, n (%)			0.727
Normal	341 (77.5)	43 (75.44)	
Abnormal	99 (22.5)	14 (24.56)	
APTT, n (%)			0.294
Normal	302 (68.64)	43 (75.44)	
Abnormal	138 (31.36)	14 (24.56)	
FDP, n (%)			0.411
Normal	417 (94.77)	56 (98.25)	
Abnormal	23 (5.23)	1 (1.75)	

ACS, acute coronary syndrome; ALB, albumin; APTT, activated partial thromboplastin time; CSF, coronary slow flow; EF, ejection fraction; eGFR, estimated glomerular filtration rate; FDP, fibrinogen degradation product; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; PCI, percutaneous coronary intervention; PT, prothrombin time; TC, total cholesterol.

with normal threshold levels that remained similar over different years were analysed as continuous variables, while indicators with normal threshold levels that differed over different years were analysed as binary variables.

Statistical analysis

The data were analysed with SPSS 25.0. Continuous data are given as means±SD or medians with IQRs depending on whether or not the data were normally distributed, while percentages are used for categorical data. The results were then compared with Pearson's chi-square tests, Student's t-tests and the rank-sum test before using logistic regression or Cox regression approaches to identify a possible link between SI values and intraprocedural events or all-cause mortality after hospital discharge. P<0.05 was the cut-off used to define significance. Two models were eventually established. The first model (Model 1) was unadjusted, whereas the second model (Model 2) was adjusted for variables that had shown statistical significance (p<0.05) in the univariate analysis and for variables (heart rate and time from onset to PCI) showing significance levels of p<0.1 but were associated with adverse events.

Table 2 Differences in sarcopenia index and coronary slow flow

Characteristics	CSF		P value
	No N=440	Yes N=57	
SI, n (%)			0.027
Low SI	103 (23.41)	21 (36.84)	
High SI	337 (76.59)	36 (63.16)	
SI,* mean (SD)	87.11 (17.95)	81.99 (17.17)	0.043

Low SI: SI≤74.48; High SI: SI>74.48.
*Stands for continuity variable.
CSF, coronary slow flow; SI, sarcopenia index.

Patient and public involvement statement

None.

RESULTS

This study included 497 patients who underwent PCI in our hospital, of whom 369 (74.25%) were males. Overall, 57 patients (11.47%) developed CSF, 100 (20.12%) developed CNR and 44 (8.85%) died post-discharge. Four patients (0.8%) developed MVA, and these were not included in further analyses. One hundred and twenty-four patients (24.95%) were classified as having low SI, and 373 (75.05%) were classified as having high SI. Significant differences were observed in the LVESD, LVEDD and ALB values between patients with and without CSF, while no significant differences were found in terms of age, sex, reason for PCI, time from onset to PCI, number of stents implanted, drinking history, smoking history, heart rate, hypertension, diabetes, lung disease, arrhythmia, EF, eGFR, HDL, LDL, TC, PT, APTT and FDP between the groups (table 1). There were significant differences between the high- and low-SI groups in terms of age, sex, time from onset to PCI, smoking history, drinking history, eGFR, HDL, APTT and FDP (online supplemental table S2).

The proportion of patients in the low-SI group who developed CSF was higher than those who did not (16.94% vs 9.65%, p=0.027). In addition, the mean SI of patients with CSF was lower than that of patients without CSF (81.99 vs 87.11, p=0.043; table 2, figure 1).

Further logistic regression analysis showed that the risk of CSF was higher in the low-SI group compared with the high-SI group (OR = 1.91, 95% CI: 1.1 to 3.41; table 3). There was no difference in the incidence of low SI and high SI in patients who developed CNR (online supplemental table S3) and died (online supplemental table S4). When SI was used as a continuous variable, the mean SI

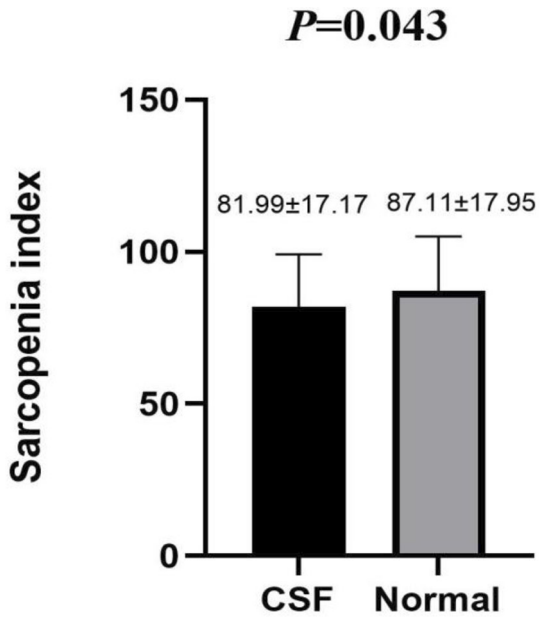

Figure 1 Distribution diagram of sarcopenia index in CSF group and normal group. CSF, coronary slow flow.

of patients with or without CNR and death did not differ either (online supplemental tables S1 and S2). After adjusting for possible confounders (LVESD, LVEDD, ALB, time from onset to PCI, heart rate), low SI was still associated with a higher risk of CSF (OR=2.01, 95% CI: 1.04 to 3.89; table 3). In addition, it was also found that the lower the SI, the higher the risk of developing CSF (OR=0.984, 95% CI: 0.968 to 0.999; table 3). Even after adjusting for possible confounders, the lower the SI, the higher the risk of CSF (OR=0.983, 95% CI: 0.967 to 0.999; table 3). Specifically, as a continuous variable, the OR was 0.983, indicating that for every unit increase in SI, the probability of CSF occurrence decreases by approximately 1.7%.

DISCUSSION

The results showed a significant association between low SI and CSF risk in patients undergoing PCI, and the lower the SI value, the greater the CSF risk. In contrast, SI was not found to be associated with CNR and the risk of death post-discharge in patients undergoing PCI. The novelty of this study is that it is the first investigation of the relationship between SI values and the risk of intraprocedural events and post-discharge death in patients undergoing PCI. In clinical practice, this indicator could be used as a surrogate marker of sarcopenia to convey the risk of possible intraprocedural events (CSF) to patients' families. It remains unknown whether the SI could also be used as a biomarker and surrogate indicator of sarcopenia to predict other conditions in these patients, such as the length of hospital stay or the need for re-admission for PCI. Future studies may be able to explore this further.

Our results showed that the SI could be used as a surrogate marker of sarcopenia to predict the risk of intraprocedural events (CSF) in patients undergoing PCI. It has been shown that surrogate markers of sarcopenia are effective for predicting adverse outcomes in PCI patients. For example, the lower L1 skeletal muscle index quartile used as a surrogate marker of sarcopenia was found to independently predict MACE,[21] and the SI based on serum Cr and CysC could be used as a surrogate marker of sarcopenia to predict the risk of MACE within 1 year of undergoing PCI.[19] There are two possible explanations for the mechanisms by which sarcopenia can predict intraprocedural events. First, one of the core features of sarcopenia is the impairment of physical function.[22] The presence of sarcopenia may thus be a necessary background for organ-specific pathophysiology, potentially leading to the development of heterogeneous clinical symptoms.[18 23] Second, the muscle is an endocrine organ that can produce and release a series of cytokines and peptides.[24] The presence of sarcopenia will thus also weaken its endocrine function.[25] Muscle-derived follistatin-related protein 1 has been shown to promote endothelial cell function and vascular recanalisation under conditions of ischaemic stress.[26] This may thus be one of the mechanisms influencing the risk of intraprocedural events. However, the explanation for the lack of association between the SI and CNR risk is still unclear and requires further research.

Table 3 Association between sarcopenia index and coronary slow flow

	Model 1		Model 2	
Variable	**P value**	**OR (95% CI)**	**P value**	**OR (95% CI)**
SI				
High SI	–	1	–	1
Low SI	0.029	1.91 (1.1 to 3.41)	0.038	2.01 (1.04 to 3.89)
SI*	0.043	0.984 (0.968 to 0.999)	0.045	0.983 (0.967 to 0.999)

Model 1: a non-adjusted model. Model 2: adjusted for LVESD, LVEDD, ALB level, time from onset to PCI, heart rate. Low SI: SI≤74.48; High SI: SI>74.48.
*Stands for continuity variable.
ALB albumin; CSF, coronary slow flow; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; PCI, percutaneous coronary intervention; SI, sarcopenia index.

Our study did not find that SI was associated with the risk of out-of-hospital mortality in post-PCI patients, whereas Lee *et al* previously found that SI was associated with cardiovascular and non-cardiovascular mortality in post-PCI patients.[18] The main reasons for this difference may include the limited sample size and the low number of death events, the failure to distinguish between cardiovascular and non-cardiovascular post-discharge deaths and the lack of any comparisons of short-term and long-term post-discharge deaths. Different results may manifest with the prolongation of the follow-up time. Second, we used indicators on admission as a proxy for muscle mass, and these indicators may not have sufficient predictive value for long-term death.

The study limitations include the study design, namely, a single-centre retrospective analysis; additional large-scale prospective multi-centre studies will be essential to verify the results of this study. Furthermore, the incidence of observed intraprocedural events was relatively high. This high rate may limit the generalisability of the findings of this study, especially in centres with lower rates of intraprocedural events. Second, the included study population was small, and there were no further examinations of the relationship between SI and short-term versus long-term post-discharge death or cardiovascular and non-cardiovascular death in patients after PCI. We will continue follow-up efforts in subsequent studies. Finally, the data were collected retrospectively over 5 years and were dependent on the actual number of patients treated; the sample size could thus not be calculated before the start of the study, which may have led to an insufficient sample size. In addition, since reagents may be replaced during this period, the reference ranges of blood test indicators may be different; therefore, the absolute values of blood test indicators cannot be compared.

CONCLUSION

Patients with lower SI had a greater risk of developing CSF, and the lower the SI, the higher the risk of CSF. However, these data suggest that the SI was not associated with CNR and the risk of post-discharge death in patients after PCI.

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Contributors Study concept and design: SL, SH, JL, XC and LZ. Acquisition of data: SL and JL. Analysis and interpretation of data: SL, SH, XC and LZ. Drafting of the manuscript: SL and SH. Critical revision of the manuscript for important intellectual content: XC and LZ. SL and SH contributed equally to this article, so they are listed as co-first authors. XC and LZ contributed equally to the guidance of this article, so they are listed as co-corresponding author. XC is the guarantor.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, conduct, reporting or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study was conducted in accordance with the Declaration of Helsinki, and the ethical approval was obtained from the Research Ethics Committee of the Affiliated Traditional Chinese Medicine Hospital of Southwest Medical University (No. BY2023032). The Research Ethics Committee of the Affiliated Traditional Chinese Medicine Hospital of Southwest Medical University waived informed consent for this study. We retrospectively systematically extracted data from medical records, thus waiving patient informed consent. For the follow-up of death, verbal consent was obtained by telephone, and patients who refused were excluded. All methods comply with relevant guidelines and regulations.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. The datasets generated and analysed during the current study are not publicly available since it is part of a cohort study. The datasets will be available 2 years after publication and are currently available from the corresponding author on a reasonable request.

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Table S1 Normal threshold levels of different blood test indicators in different years

Years	HDL (mmol/L)	LDL (mmol/L)	TC (mmol/L)	ALB (g/L)	PT (sec)	APTT (sec)	FDP (mg/L)
2016	0.77-2.25	1.27-4.13	2.9-5.7	35-55	9-16	20-42	0-7
2017	0.77-2.25	1.27-4.13	2.9-5.7	35-55	9-16	20-42	0-7
2018	0.77-2.25	1.27-4.13	2.9-5.7	35-55	11-14.5	28-44	0-5
2019	0.77-2.25	1.27-4.13	2.9-5.7	35-55	11-14.5	28-44	0-5
2020	0.77-2.25	1.27-4.13	2.9-5.7	35-55	11-14.5	28-44	0-5

Note: HDL: high-density lipoprotein; LDL: low-density lipoprotein; TC: cholesterol; ALB: albumin; PT: prothrombin time; APTT: activated partial thromboplastin time; FDP: fibrinogen degradation product.

Table S2 Baseline characteristics of participants according to the SI

Characteristics	SI		*P*-value
	Low (n=124)	High (n=373)	
Age years, median (iqr)	61(54, 71)	66(56, 73.5)	**0.021**
Sex, n (%)			**<0.01**
Male	123(99.19)	246(65.95)	
Female	1(0.81)	127(34.05)	
The reasons for PCI, n (%)			0.996
Stable angina pectoris	4(3.22)	10(2.68)	
ACS	120(96.78)	363(97.32)	
Time from onset to PCI, hour, median (iqr)	15(6, 48)	10(4, 35)	**0.003**
Number of stent implants, n (%)			0.317
1	66(53.23)	192(51.47)	
2-3	53(42.74)	151(40.48)	
>3	5(4.03)	30(8.05)	
Smoking history, n (%)			**<0.001**
No	73(58.87)	135(36.19)	
Yes	51(41.13)	238(63.81)	
Drinking history, n (%)			**<0.001**
No	89(71.77)	185(49.60)	
Yes	35(28.23)	188(50.40)	
Heart rate, median(iqr)	75.5(64, 87)	75(66, 88)	0.7
Hypertension, n (%)			0.858
No	54(43.55)	159(42.63)	
Yes	70(56.45)	214(57.37)	
Diabetes, n (%)			0.779
No	93(75.00)	275(73.73)	
Yes	31(25.00)	98(26.27)	
Pulmonary disease, n (%)			0.493
No	109(87.90)	336(90.08)	

Yes	15(12.10)	37(9.92)	
Arrhythmia, n (%)			0.303
No	102(82.26)	321(86.06)	
Yes	22(17.74)	52(13.94)	
EF%, median (iqr)	62(53, 66)	61(55, 65)	0.585
LVESD, mm, median (iqr)	31(29, 35)	31(29, 35)	0.816
LVEDD, mm, median (iqr)	47(44, 50)	47(44, 50)	0.389
eGFR, ml/min/1.73m^2, median (iqr)	75.86(58.57, 93.3)	68.85(55.79, 85)	**0.019**
eGFR, ml/min/1.73m^2, n (%)			0.584
<60	89(71.77)	258(69.17)	
⩾60	35(28.23)	115(30.83)	
HDL, mmol/l, median (iqr)	1.2(1.03, 1.37)	1.14(0.96, 1.36)	**0.033**
LDL, mmol/l, median (iqr)	3.03(2.36, 3.69)	3.03(2.5, 3.65)	0.658
TC, mmol/l, median (iqr)	4.64(4.04 5.4)	4.64(4.09, 5.44)	0.684
ALB, g/l, median (iqr)	41.25(37.7, 43.68)	41.5(38.85, 43.75)	0.215
ALB, g/l, n (%)			0.507
Normal	75(60.48)	238(63.81)	
Abnormal	49(39.52)	135(36.19)	
PT, n (%)			0.3
Normal	100(80.65)	284(76.14)	
Abnormal	24(19.35)	89(23.86)	
APTT, n (%)			**0.002**
Normal	100(80.65)	245(65.68)	
Abnormal	24(19.35)	128(34.32)	
FDP, n (%)			**0.03**
Normal	123(99.19)	350(93.83)	
Abnormal	1(0.81)	23(6.17)	

Note: SI: sarcopenia index; PCI: percutaneous coronary intervention; ACS: acute coronary syndrome; EF: ejection fraction; LVESD: left ventricular end-systolic diameter; LVEDD: left ventricular end-diastolic diameter; eGFR: estimated glomerular filtration rate; HDL: high-density lipoprotein; LDL: low-density lipoprotein; TC: cholesterol; ALB: albumin; PT: prothrombin time; APTT: activated partial thromboplastin time; FDP: fibrinogen degradation product.

Table S3 Differences in SI and CNR

Characteristics	CNR		P-value
	No	**Yes**	
	N=397	**N=100**	
SI, n (%)			0.806
Low SI	100(25.19)	24(24)	
High SI	297(74.81)	76(76)	
SI*, mean (SD)	86.17(17.94)	87.92(17.87)	0.382

NOTE:

* Stands for continuity variable.

Low SI: SI⩽74.48; High SI: SI >74.48.

Abbreviation: SI: sarcopenia index; CNR: coronary artery no-reflow; SD: standard deviation.

Table S4 Differences in SI and death

Characteristics	Death		P-value
	No	Yes	
	N=453	N=44	
SI, n (%)			0.067
Low SI	108(23.84)	16(36.36)	
High SI	345(76.16)	28(63.64)	
SI*, mean (SD)	86.85(17.85)	83.1(18.49)	0.186

NOTE:

*Stands for continuity variable.

Low SI: SI⩽74.48; High SI: SI >74.48

Abbreviation: SI: sarcopenia index; SD: standard deviation.