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Atherosclerosis

Systemic Inflammatory Response Syndrome is an Independent Predictor of One-Year Mortality in Patients with Acute Myocardial Infarction

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Background: Convincing evidence suggests that inflammatory biomarkers are associated with an increased risk among patients with acute myocardial infarction (AMI). However, the impact of systemic inflammatory response (SIRS) on one-year clinical outcomes remains uncertain. Herein we investigated the impact of SIRS on one-year mortality and major adverse cardiovascular events (MACE) in patients with AMI.

Methods: We conducted a retrospective study that enrolled patients admitted due to AMI and who received coronary artery intervention from January 2012 to June 2014. SIRS was defined according to standard criteria as having two or more of the following: (1) body temperature < 36 or > 38 °C, (2) heart rate > 90 beats per minute, (3) respiratory rate > 20, or (4) white blood cell count < 4000/mm^3 or > 12,000/mm^3. The primary endpoint was one-year mortality. The secondary endpoint was a one-year MACE, including revascularization, AMI, and stroke.

Results: A total of 330 AMI patients were enrolled in the study, and 121 study subjects (36.6%) met the SIRS criteria. AMI patients with SIRS on admission had significantly increased one-year all-cause mortality (control vs. SIRS: 21.1% vs. 33.1%, p = 0.026) and one-year MACE (35.9% vs. 53.7%, p = 0.022). Patients with SIRS had a higher incidence of one-year non-fatal myocardial infarction, but not non-fatal stroke. After multivariable adjustment, SIRS [hazard ratio (HR) = 1.773, 95% confidence interval (CI) = 1.097-2.886, p = 0.019] and age (HR = 1.038, 95% CI = 1.018-1.058, p < 0.001) were associated with enhanced risk of one-year mortality.

Conclusions: This study revealed that AMI patients with SIRS on initial admission were associated with increased risk of one-year all-cause mortality.

Key Words: Myocardial infarction • Systemic inflammatory response syndrome

INTRODUCTION

Acute myocardial infarction (AMI) remains the leading cause of death worldwide and imposes a large economic burden on healthcare systems. Despite recent advances in therapeutic strategies, the prognosis of patients with AMI remains poor. Atherosclerosis is a systemic inflammatory disease of the arterial wall caused by endothelial dysfunction.[1,2] Myocardial infarction occurs predominantly as a result of acute atherosclerotic plaque rupture and infiltration of inflammatory cells.[3] An acute inflammatory response is a major characteris-

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tic of ischemia/reperfusion injury following acute cardiovascular events.[2-5] Inflammatory biomarkers have also been used to assess the risk of adverse events in patients with acute coronary syndrome.[6-10] Accumulating evidence indicates that enhanced inflammatory processes are crucial mediators of the deleterious effects of AMI.[11-13] Although local vascular inflammation has been identified to play a crucial role in all stages of atherosclerosis, the impact of systemic inflammatory response syndrome (SIRS) in patients with AMI remains uncertain.

SIRS is defined as the presence of any two of the following diagnostic criteria: abnormal temperature, heart rate, respiratory rate, and leukocyte count.[14] It is generally caused by inappropriate immune responses due to infectious or non-infectious conditions, and it can occasionally lead to profound shock and multiple organ failure. Recent studies have shown that a diagnosis of SIRS is useful for severity-of-illness scoring systems in hospitalized patients,[2] surgical patients,[15,16] and that it is associated with adverse prognostic outcomes in critically ill patients.[17,18] In addition, Diepan et al. reported that a diagnosis of SIRS in patients with ST-elevation myocardial infarction (STEMI) was independently associated with death, shock, heart failure and stroke at 90 days.[19] Furthermore, Fosco et al. reported that SIRS was associated with a higher risk of hospital mortality among patients with acute coronary syndrome without heart failure.[20] However, clinical data related to the impact of SIRS on patients with AMI with regards to one-year clinical outcomes remains unknown. Herein, we conducted a single-center, retrospective study to investigate the association between SIRS and short- and long-term outcomes in patients with AMI.

METHODS

Study population

In this retrospective study, we screened 427 patients admitted to the cardiac care unit (CCU) of Taipei Veterans General Hospital due to AMI from January 2012 to June 2014. Patients older than 18 years who were diagnosed with AMI at admission, either STEMI or non-ST-segment elevation myocardial infarction (NSTEMI), were screened for the study. AMI was defined as an elevated troponin I level $\geq$ 0.1 ng/mL, accompanied by either typical chest pain for > 30 minutes and/or electrocardiographic changes (including ischemic ST-segment depression, ST-segment elevation, or pathologic Q waves).[21] The inclusion criterion was a diagnosis of AMI on presentation (n = 427). The exclusion criteria were: (1) patients refusing coronary interventions or if the physician chose conservative therapy due to any reasons (n = 65); and (2) patients with end-stage renal disease (ESRD) at initial presentation (n = 32). After excluding these 65 patients, the remaining 330 patients were enrolled for analysis. The flowchart of patient enrollment is shown in Figure 1.

After enrollment, the chart of each patient was reviewed in detail and data on symptoms, medication, coronary risk factors, pervious cardiac events, and other systemic diseases were collected. Clinical characteristics including age, gender, presence of hypertension, hyperlipidemia, diabetes mellitus (DM), congestive heart failure (CHF), vessels of AMI, incidence of contrast-induced nephropathy (CIN), and laboratory examinations were also recorded. CIN was defined as an elevation in serum creatinine level of more than 0.5 mg/dL or more than 25% of baseline at 48 hours after a percutaneous coronary intervention (PCI).[22] Data including medical histories, physical exams, 12-lead electrocardiograms, blood exams and chest films were also obtained for each patient. Electrocardiograms were repeated in cases of recurrent symptoms. Leukocytes were counted using an automated cell counter as per standard laboratory techniques. Each patient had two or more plasma cardiac troponin I and creatine kinase (CK) analyses. One was performed at least 12 hours after the onset of symp-

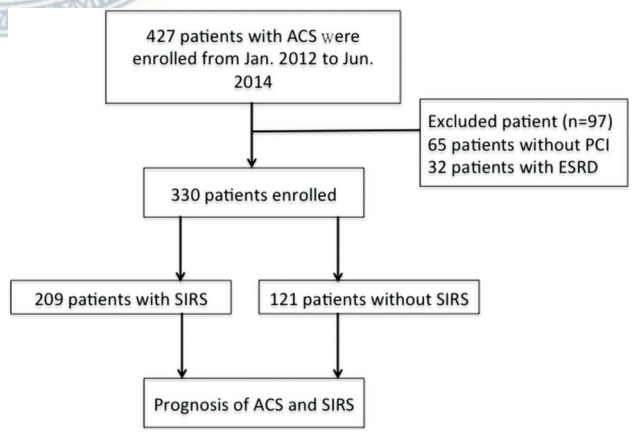

Figure 1. *Flowchart of patient enrollment. ACS, acute coronary syndrome; ESRD, end-stage renal disease; PCI, percutaneous coronary intervention; SIRS, systemic inflammatory response syndrome.*

toms, and the levels of cardiac enzymes were checked every six hours until CK and troponin I peaked. Left ventricular ejection fraction (LVEF) was assessed by 2D-echocardiography immediately after the patients were admitted to the CCU. This study was conducted according to the principles of the Declaration of Helsinki, and it was approved by the Research Ethics Committee of Taipei Veterans General Hospital. All participants provided written informed consent.

Definition of SIRS

SIRS was defined based on the America College of Chest Physicians and Society of Critical Care Medicine Criteria as two or more of the following criteria at presentation when admitted to our intensive care unit (ICU): heart rate > 90 beats/minute, respiratory rate > 20 breaths/minutes, leukocyte count > 12 or < $4*10^9$/L, temperature > 38 °C or < 36 °C.[14]

Study endpoints

The primary endpoint was one-year mortality after AMI, and the secondary endpoint was the occurrence of a major adverse cardiovascular event (MACE) (including target vessel revascularization, non-fatal myocardial infarction, and non-fatal ischemic stroke) within 1 year after AMI. Target vessel revascularization was defined as balloon dilatation or stent deployment over a previously treated lesion. Non-fatal myocardial infarction was defined as elevation of cardiac troponin I (> 1 ng/ml) with symptoms of ischemia. Non-fatal ischemic stroke was defined as the presence of new neurological defects as verified by either computed tomography or magnetic resonance imaging. All patients received regular follow-up at our out-patient department, and their medical records were carefully reviewed.

Statistical analysis

All analyses were performed using SPSS statistical software (version 20, IBM Corporation, Armonk, NY, USA). Data were expressed as mean and standard deviation (SD) for numeric variables, and as number (percentage) for categorical variables. Comparisons of continuous variables between groups were performed using the Student's t test. Subgroup comparisons of categorical variables were assessed using the chi-square (χ^2) or Fisher's exact test. A p-value < 0.05 was considered to be statistically significance. We entered variables that have previously been reported to be independent predictors of adverse cardiac events[23] into univariate logistic analysis. Variables with statistical significance in the univariate analysis were then selected for multivariate analysis. Multivariate analysis was performed using a Cox regression hazard model to determine the independence of SIRS in predicting death and cardiovascular events. In analysis of the one-year mortality rate after AMI, SIRS and age were entered into multivariate analysis. In analysis of the one-year MACE rate after AMI, SIRS, age, hypertension, C-reactive protein (CRP), and peak CK level were selected instead. Kaplan-Meier survival curves and the log-rank test were used to determine associations with the endpoints. We also used Kaplan-Meier survival curves to compare patients with and without SIRS with regards to the rates of one-year mortality and one-year MACE after AMI.

RESULTS

Study population and baseline characteristics

A total of 330 patients with AMI (mean age: 71.8 ± 15.2 years; males, 75.1%) were enrolled in the study. The baseline characteristics of the patients with SIRS (n = 121) and without SIRS (n = 209) are outlined in Table 1. The SIRS group were older, had higher rates of hypertension, CHF, and CIN, higher heart rate, higher white blood cell count and peak CK level after the procedure, and had a higher percentage of total revascularization. We also stratified the study cohort according to the types of AMI (STEMI or NSTEMI). The patients with STEMI were younger, had lower rates of hypertension and heart failure, and a higher percentage of total revascularization, but higher levels of initial and peak CK (Supplement Table 1).

Patient follow-up and cardiovascular endpoints

All patients were followed up for one year after the AMI episode or until the occurrence of MACE or mortality. All of the study subjects were included in the analysis, and the mean follow-up duration was 199 ± 178 days. The primary and secondary outcomes for the patients at the presentation of acute coronary syndrome (ACS) are presented in Table 2. The outcomes of the two groups revealed a significantly higher rates of 90-day

Table 1. Baseline characteristics of patients with or without SIRS after acute myocardial infarction (AMI)

	Without SIRS (N = 209)	With SIRS (N = 121)	p value
Age (years)	71.0 ± 16.0	72.0 ± 15.0	0.591
Male, n (%)	156 (74.6)	92 (76.0)	0.732
SBP (mmHg)	116 ± 26.0	111 ± 27.0	0.122
Heart rate (beats/min)	79.0 ± 18.0	95.0 ± 20.0	< 0.001
Respiratory rate (rates/min)	19.0 ± 2.6	22.0 ± 4.6	< 0.001
Body temperature (°C)	36.4 ± 0.6	36.2 ± 1.1	0.074
Current smoker, n (%)	42 (20.3)	26 (21.5)	0.133
Diabetes mellitus, n (%)	94 (45.0)	52 (43.0)	0.724
Hypertension, n (%)	148 (70.8)	86 (71.1)	0.960
Heart failure, n (%)	20 (9.6)	16 (13.3)	0.299
Prior CABG, n (%)	13 (6.2)	1 (0.8)	0.020
Different types of AMI			0.455
STEMI	136 (65.1)	78 (64.5)	
NSTEMI	73 (34.9)	43 (35.5)	
Location of vascular lesions			0.577
LAD	109 (35.6)	79 (37.4)	
LCX	100 (32.7)	57 (27.0)	
RCA	97 (31.7)	75 (35.5)	
Medications, n (%)			
Aspirin	51 (26.2)	25 (21.7)	0.369
Beta blocker	45 (21.5)	28 (23.1)	0.837
ACEi	19 (9.0)	15 (12.3)	0.401
Laboratory data			
White blood cell (10^3/cumm)	9.8 ± 10.0	15.0 ± 22.0	0.002
Segment (%)	72.0 ± 12.0	78.0 ± 12.0	< 0.001
Hemoglobin (g/dl)	12.5 ± 2.6	12.3 ± 2.6	0.834
Creatinine (mg/dl)	1.92 ± 3.4	1.88 ± 1.6	0.832
CRP (mg/dl)	7.2 ± 7.8	8.4 ± 7.4	0.108
Initial CK (mg/dL)	632.0 ± 1332.0	889.0 ± 1586.0	0.223
Peak CK (mg/dl)	959.0 ± 1826.0	1325.0 ± 2386.0	0.123
Total revascularization	43 (20.6)	45 (37.8)	0.042
CIN	6 (2.9)	13 (10.7)	0.003
LVEF (%)	49.5 ± 11.2	46.6 ± 12.2	0.376

ACEi, angiotensin converting enzyme inhibitor; CABG, coronary artery bypass surgery; CIN, contrast induce nephropathy; CK, creatine kinase; CRP, C-reactive protein; LAD, left anterior descending artery; LCX, left circumflex artery; LVEF, left ventricular ejection fraction; NSTEMI, non ST segment elevation myocardial infarction; RCA, right coronary artery; SBP, systolic blood pressure; STEMI, ST segment elevation myocardial infarction.

Table 2. Short-term and long-term outcomes of patients with and without SIRS after acute myocardial infarction

Outcomes	Without SIRS (N = 209)	With SIRS (N = 121)	p value
90-days mortality, n (%)	26 (12.4)	25 (20.7)	0.049
90-days MACE, n (%)	32 (15.3)	30 (24.8)	0.039
90-days non-fatal MI, n (%)	26 (12.4)	24 (19.8)	0.042
90-days non-fatal stroke, n (%)	2 (1.0)	3 (2.5)	0.132
90-days revascularization, n (%)	4 (1.9)	3 (2.5)	0.726
One-year mortality, n (%)	44 (21.1)	40 (33.1)	0.026
One-year MACE, n (%)	75 (35.9)	65 (53.7)	0.022
One-year non-fatal MI, n (%)	56 (26.8)	50 (41.3)	0.032
One-year non-fatal stroke, n (%)	7 (3.3)	6 (5.0)	0.322
One-year re-vascularization, n (%)	12 (5.7)	9 (7.4)	0.532

MACE, major adverse cardiovascular events; MI, myocardial infarction.

all-cause mortality (AMI without SIRS vs. AMI with SIRS: 12.4% vs. 20.7%, p = 0.049), 90-day MACE (15.3% vs. 24.8%, p = 0.039), 90-day non-fatal myocardial infarction (MI) (12.4% vs. 19.8%, p = 0.042), one-year all-cause mortality (21.2% vs. 33.1%, p = 0.026), one-year MACE (35.9% vs. 53.7%, p = 0.022), and one-year non-fatal MI (26.8% vs. 41.3%, p = 0.032) in the patients with AMI and SIRS compared to those without SIRS. However, there were no significant differences in the incidence of one-year non-fatal stroke (p = 0.322) and one-year revascularization (p = 0.532) between the two groups. Kaplan-Meier analysis showed that in comparing the one-year mortality and one-year MACE rates, the patients without SIRS had a likelihood of being disease free (one-year all-cause mortality: log rank p < 0.001, one-year MACE: log rank p = 0.024; Figure 2).

Multivariate Cox regression analysis

The hazard ratios (HR) associated with individual confounders for all-cause mortality within one year are presented in Table 3. SIRS and age were associated with an unadjusted higher risk of one-year mortality at the time of presentation [SIRS: HR = 1.71, 95% confidence interval (CI), 1.06-2.76, p = 0.028; age: HR = 1.04, 95% CI, 1.02-1.06, p < 0.001]. After multivariate adjustments, SIRS and age were still associated with an adjusted higher prevalence of one-year mortality at the time of presentation (SIRS: HR = 1.77, 95% CI, 1.10-2.89, p = 0.019; age: HR = 1.04, 95% CI, 1.02-1.06, p < 0.001). As shown in Table 4, SIRS was also associated with an unadjusted higher risk of one-year MACE (HR = 1.50, 95% CI, 1.05-2.14, p = 0.028). However, the association between SIRS and MACE became insignificant after adjusting for the confounding factors (HR = 0.89, 95% CI, 0.55-1.48, p = 0.659).

DISCUSSION

Main findings

This single-center, retrospective study revealed that

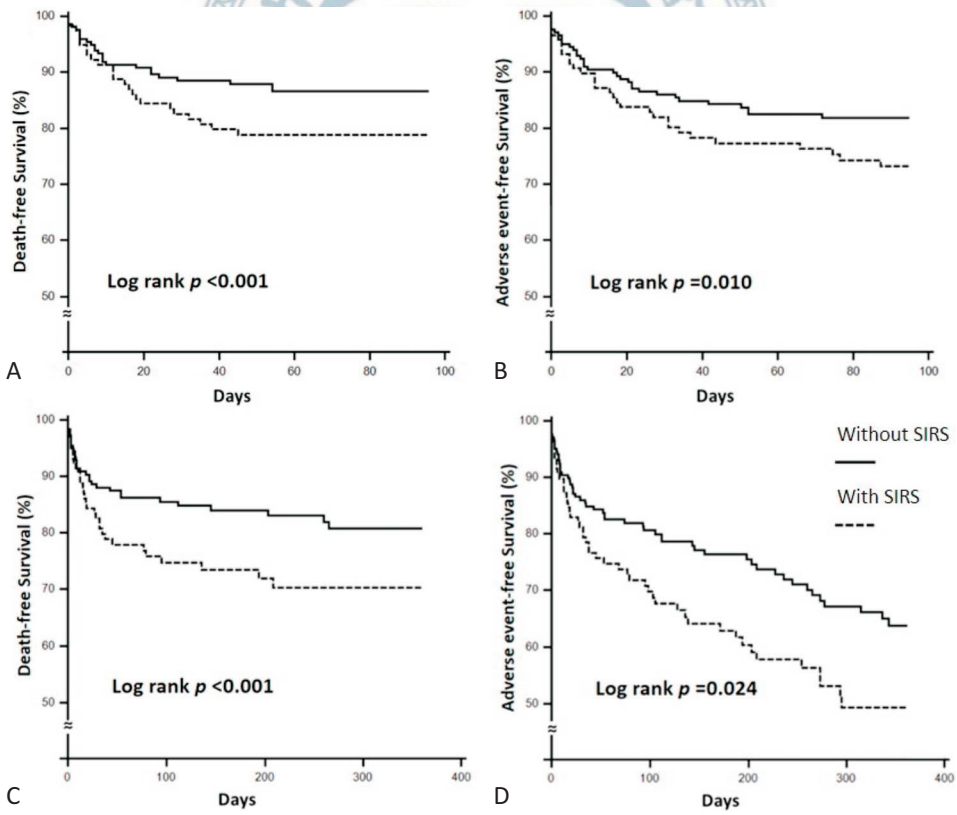

Figure 2. *Kaplan-Meier curves of 90-day mortality (A), 90-day MACE (B), one-year mortality (C) and one-year MACE (D) in patients with or without SIRS after receiving PCI. MACE, major adverse cardiovascular events, PCI, percutaneous coronary intervention; SIRS, systemic inflammatory response syndrome.*

Table 3. Cox regression for one-year mortality in patients with SIRS after acute myocardial infarction

Variable	Univariate		Multivariate	
	Hazard ratio (95% CI)	p value	Hazard ratio (95% CI)	p value
SIRS group	1.71 (1.06-2.76)	0.028	1.77 (1.10-2.89)	0.019
Age	1.04 (1.02-1.06)	< 0.001	1.04 (1.02-1.06)	< 0.001
Gender	1.49 (0.90-2.47)	0.123		
Hypertension	1.20 (0.81-1.77)	0.374		
Diabetes	1.05 (0.73-1.50)	0.799		
CHF	1.09 (0.64-1.84)	0.752		
CRP	0.97 (0.97-1.03)	0.968		
Peak CK	1.00 (0.96-1.01)	0.843		
CIN	1.46 (0.57-3.64)	0.416		

CI, confidence interval; CHF, congestive heart failure; CIN, contrast induce nephropathy; CK, creatine kinase; CRP, C-reactive protein; SIRS, systemic inflammatory response syndrome.

Table 4. Cox regression for one-year MACE in patient with SIRS after acute myocardial infarction

Variable	Univariate		Multivariate	
	Hazard ratio (95% CI)	p value	Hazard ratio (95% CI)	p value
SIRS group	1.50 (1.05-2.14)	0.028	0.89 (0.55-1.48)	0.659
Age	1.02 (1.01-1.03)	0.004	1.01 (0.99-1.03)	0.223
Gender	0.90 (0.60-1.36)	0.608		
Hypertension	0.64 (0.44-0.93)	0.018	0.57 (0.33-0.99)	0.041
Diabetes	0.73 (0.51-1.06)	0.095		
CHF	1.46 (0.87-2.44)	0.151		
CRP	1.04 (1.02-1.06)	< 0.001	1.02 (0.99-1.06)	0.123
Peak CK	1.01 (1.01-1.02)	0.006	1.02 (1.01-1.03)	0.008
CIN	1.54 (0.75-3.17)	0.240		

CI, confidence interval; CHF, congestive heart failure; CIN, contrast induce nephropathy; CK, creatinine kinase; CRP, C-reactive protein; MACE, major adverse cardiovascular events; SIRS, systemic inflammatory response syndrome.

SIRS on initial admission was a poor prognostic factor for both short-term and long-term outcomes in the patients with AMI. Even after adjusting for age, SIRS was independently associated with a higher one-year rate of all-cause mortality. On the other hand, the association between SIRS and one-year MACE became insignificant after multivariate adjustments. To the best of our knowledge, this is the first study to investigate the impact of SIRS on one-year adverse outcomes in patients with AMI.

Atherosclerosis, AMI, and inflammatory response

Inflammatory responses play an important role in the pathophysiology of stable coronary artery disease as well as AMI. Atherosclerosis is an inflammatory disease involving the vascular wall and endothelium,[24] and the acute rupture of atherosclerotic plaques is one of the leading causes of MI.[25] Ruptured coronary artery plaques may cause the release of cytokines and other inflammatory acute-phase proteins into the systemic circulation, which can then induce a systemic inflammation response.[3] Moreover, a post-infarction reaction can result in myocardial cell necrosis,[26] progressive myocyte apoptosis,[27] and inflammatory signal transduction.[28] Acute-phase proteins such as tumor necrosis factor-alpha and interleukin-6 (IL-6) are produced soon after myocardial ischemia, and can regulate myocyte apoptosis and trigger additional cellular inflammatory responses.[7-9] The magnitude of the acute phase response has been found to be directly correlated with short- and long-term prognoses after MI.[11,12]

Pathophysiology of SIRS, and its impact on AMI

While local inflammation is a protective response adjusted by the body, systemic, exaggerated inflamma-

tion is usually harmful.[29] SIRS is defined according to simple clinical criteria and leukocyte count,[14] and it has been shown to be an excellent predictor of morbidity and mortality in a variety of medical illnesses. Patients with SIRS have been reported to have increased ICU and hospital stays, and a higher in-hospital mortality rate.[30] SIRS has also been associated with an increased risk of mortality in patients with CHF[31,32] and STEMI.[19]

Several pathways may explain the association between SIRS and AMI. First, because of an unstable hemodynamic status caused by systemic inflammation, SIRS itself may lead to myocardial hypoperfusion or type II MI.[33,34] In addition, systemic inflammation also has a variety of effects on hemostasis,[29] such as the induction of initial coagulation via tissue factor-related thrombin, down-regulation of anticoagulant pathways, and inhibition of fibrinolysis.[35-37] Vascular endothelial cells express adhesion molecules and growth factors in response to systemic inflammation,[38] which exacerbates the condition of MI. Finally, AMI can also induce a massive release of cytokines,[3,28] resulting in a systemic inflammatory response, SIRS.

Review of previous studies and novelty of this study

The presence of SIRS has been reported to be a good predictor of cardiac outcomes inpatients with MI. In a single center study enrolling 196 patients with AMI and without heart failure, the patients with SIRS were reported to have an eight-fold higher in-hospital mortality rate than those without SIRS (with SIRS vs. without SIRS: 13.6% vs. 1.7%, $p < 0.01$).[20] In another multi-center study enrolling 1,903 patients with STEMI, SIRS on admission was found to be independently associated with 90-day mortality (10.1% vs. 6.1%, $p = 0.018$) and cardiogenic shock (6.8% vs. 3.6%, $p = 0.021$).[19] However, the relationship between SIRS and long-term cardiac outcomes in patients with AMI remains undetermined.[20,39,40] Our results suggest that patients with AMI and SIRS had a poorer short-term prognosis and higher one-year all-cause mortality rate (26.4% vs. 16.3%, $p = 0.026$). Therefore, the presence of SIRS as diagnosed with routinely available variables at the bedside, could be a simple tool for risk stratification in patients with AMI.

Study limitations

There are several limitations to this study. First, it was a single-center, retrospective study with a small number of cases. Second, it enrolled patients with STEMI as well as NSTEMI, which involves various pathophysiologies and therapeutic approaches. We have clarified this difference in Supplement Table 1. Third, the study population was elderly (mean age: 72 ± 15 years) and had a higher incidence of overall mortality (one-year mortality rate: 20.0%), which limits the generalization of the conclusions. In addition, the primary endpoint was defined as all-cause mortality rather than cardiovascular death. This may have overestimated the inference of SIRS on cardiovascular disease. Moreover, the association between SIRS and MACEs, the secondary endpoint, became insignificant after multivariate adjustments.

CONCLUSIONS

The patients with AMI and SIRS on initial admission were associated with an increased risk of both short-term and long-term adverse outcomes, including higher rates of 90-day mortality, 90-day MACE, and one-year all-cause mortality.

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DECLARATION OF CONFLICT OF INTEREST

All authors declare no conflict of interest.

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None.

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SUPPLEMENT

Supplement Table 1. Baseline characteristics of study population grouping by different types of myocardial infarction (MI, included STEMI and NSTEMI)

	STEMI (N = 116)	NSTEMI (N = 214)	p value
Age (years)	69.2 ± 15.8	72.7 ± 14.8	0.052
Male, n (%)	94 (81.0)	152 (72.0)	0.162
SBP (mmHg)	131 ± 34.4	135 ± 28.3	0.086
Heart rate (beats/min)	85.0 ± 21.0	85.0 ± 20.6	0.590
Respiratory rate (rates/min)	20.3 ± 4.1	20.5 ± 3.6	0.582
Body temperature (°C)	36.4 ± 0.6	36.2 ± 1.1	0.500
Current smoker, n (%)	27 (23.2)	41 (19.2)	0.583
Diabetes mellitus, n (%)	44 (37.9)	102 (47.7)	0.089
Hypertension, n (%)	74 (63.8)	160 (74.7)	0.036
Heart failure, n (%)	5 (4.3)	214 (14.5)	0.005
Medications, n (%)			
Aspirin	24 (20.7)	61 (28.5)	0.134
Beta blocker	18 (15.5)	55 (25.7)	0.038
ACEi	10 (8.6)	24 (11.2)	0.473
Laboratory data			
White blood cell (10^3/cumm)	13.4 ± 21.5	11.1 ± 12.2	0.161
Segment (%)	74.5 ± 13.7	75.0 ± 12.1	0.531
Hemoglobin (g/dl)	13.0 ± 2.5	12.1 ± 2.7	0.433
Creatitine (mg/dl)	1.6 ± 1.3	2.3 ± 1.6	0.001
CRP (mg/dl)	4.4 ± 5.8	3.9 ± 6.1	0.556
Initial CK (mg/dL)	471.5 ± 1043.0	326.2 ± 541.0	0.010
Peak CK (mg/dl)	1646.6 ± 2672.2	789.7 ± 1541.2	0.001
LVEF (%)	49.0 ± 11.1	47.9 ± 11.9	0.449
Location of vascular lesions			0.482
LAD	69 (59.5)	111 (51.9)	
LCX	56 (48.3)	103 (48.1)	
RCA	64 (55.2)	114 (53.3)	
Total revascularization	48 (41.4)	40 (18.7)	0.032
CIN after procedure	4 (3.4)	15 (7.0)	0.185

ACEi, angiotensin converting enzyme inhibitor; CABG, coronary artery bypass surgery; CIN, contrast induce nephropathy; CK, creatine kinase; CRP, C-reactive protein; LAD, left anterior descending artery; LCX, left circumflex artery; LVEF, left ventricular ejection fraction; NSTEMI, non-ST elevation MI; RCA, right coronary artery; SBP, systolic blood pressure; STEMI, ST elevation MI.