



## Clinical

Shock Index as a predictor for In-hospital mortality in patients with non-ST-segment elevation myocardial infarction<sup>☆</sup>Akihiro Kobayashi<sup>a,\*</sup>, Naoki Misumida<sup>a</sup>, Daniel Luger<sup>a</sup>, Yumiko Kanei<sup>b</sup><sup>a</sup> Department of Internal Medicine, Mount Sinai Beth Israel, New York, USA<sup>b</sup> Department of Cardiology, Mount Sinai Beth Israel, New York, USA

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## ABSTRACT

**Background/Purpose:** Shock index (SI), a ratio of heart rate/systolic blood pressure, has been reported to predict increased mortality in patients with ST-segment elevation myocardial infarction. However, the prognostic value of SI has not been fully elucidated in patients with non-ST-segment elevation myocardial infarction (NSTEMI).

**Methods/Materials:** We performed a retrospective analysis of 481 consecutive NSTEMI patients who underwent coronary angiography from January 2013 to June 2014. Systolic blood pressure and heart rate on presentation were recorded, and SI was calculated as heart rate/systolic blood pressure. Patients were divided into those with  $SI \geq 0.7$  and those with  $SI < 0.7$ . Baseline and angiographic characteristics were recorded. In addition, cardiogenic shock and in-hospital mortality were recorded and compared between the two groups.

**Results:** Among 481 patients, 103 patients (21.4%) had  $SI \geq 0.7$ . No statistically significant difference was observed in baseline characteristics between the two groups. Patients with  $SI \geq 0.7$  had a lower left ventricular ejection fraction than those with  $SI < 0.7$  (56 [35–60] % vs. 60 [45–64] %,  $p = 0.035$ ). Patients with  $SI \geq 0.7$  had a higher rate of cardiogenic shock on admission (2.9% vs. 0.3%,  $p = 0.032$ ). Patients with  $SI \geq 0.7$  had a higher, albeit statistically insignificant, incidence of cardiogenic shock after admission (5.0% vs. 1.9%,  $p = 0.074$ ). The total incidence of cardiogenic shock was higher in patients with  $SI \geq 0.7$  (7.8% vs. 2.1%,  $p = 0.001$ ). Patients with  $SI \geq 0.7$  had higher in-hospital mortality (4.9% vs. 0.5%,  $p = 0.006$ ) than those with  $SI < 0.7$ .

**Conclusion:** Elevated SI was associated with higher in-hospital mortality in patients with NSTEMI.

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## 1. Introduction

Early revascularization is a key therapeutic strategy to improve clinical outcomes in patients with non-ST-segment elevation acute coronary syndrome (ACS) who have high-risk features [1,2]. Therefore, prompt recognition of patients with high-risk profiles is the cornerstone of managing patients with non-ST-segment elevation ACS.

Several risk scoring models, such as Thrombolysis In Myocardial Infarction (TIMI) risk score and Global Registry of Acute Coronary Events score, have been shown to identify patients at increased risks for adverse outcomes in ACS populations [3–5]. However, these scoring models require detailed patients' demographic, hemodynamic, electrocardiographic findings as well as troponin values, which makes it difficult to access patients' risk profiles soon after arrival to the hospital.

Therefore, a risk-stratification tool that promptly identifies high-risk patients will be of great clinical use.

Recently, shock index (SI), a ratio of heart rate/systolic blood pressure, has been reported to predict increased mortality in patients with ST-segment elevation myocardial infarction (STEMI) [6–9]. However, the prognostic value of SI has not been previously addressed in a non-ST-segment elevation myocardial infarction (NSTEMI) population.

Thus, the aim of this study is to evaluate the prognostic value of SI in patients with NSTEMI.

## 2. Material and methods

A retrospective analysis was performed on NSTEMI patients who underwent coronary angiography between January 2013 and June 2014 at our institution. Myocardial infarction was diagnosed in accordance with the European Society of Cardiology and American College of Cardiology criteria [10]. Inclusion criteria were: 1) troponin I level greater than the 99th percentile reference value before cardiac catheterization; 2) chest pain (or anginal equivalent) or ischemic change on electrocardiogram including horizontal or down-sloping ST-segment depression ( $\geq 0.05$  mV) or T-wave inversion ( $\geq 0.10$  mV) in two or more contiguous leads; and 3) the absence of ST-segment elevation

Abbreviation: SI, shock index.

<sup>☆</sup> Conflict of interests: None.

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and new left bundle branch block on electrocardiogram. Among 728 patients reviewed, 247 patients were excluded due to following exclusion criteria. Exclusion criteria were: 1) cardiac catheterization more than 5 days after presentation ( $n = 143$ ); 2) severe aortic stenosis, hypertrophic cardiomyopathy, self-reported cocaine use within 5 days, cardiac arrest on presentation, ventricular tachycardia, supraventricular tachycardia with heart rate greater than 150 beats per minute, implantable cardioverter defibrillator shock, and blood pressure on presentation  $>230/130$  mmHg ( $n = 49$ ); 3) subsequent documented diagnosis of Takotsubo cardiomyopathy, myocarditis, and pulmonary embolism ( $n = 15$ ); and 4) insufficient data for analysis ( $n = 40$ ). As a result, a total of 481 patients with NSTEMI were included in the final analysis.

The present study complied with the Declaration of Helsinki and was approved by the institutional review board.

### 2.1. Demographic, hemodynamic and laboratory data

Patients' demographic data such as age, gender, body mass index, history of hypertension, history of diabetes mellitus, history of hyperlipidemia, history of chronic kidney disease defined as estimated glomerular filtration rate (eGFR)  $< 60$  mL/min/1.73m<sup>2</sup>, family history of coronary artery disease (CAD), smoking status, use of  $\beta$ -blocker, previous percutaneous coronary intervention (PCI), previous coronary artery bypass grafting (CABG), and previous myocardial infarction were recorded. TIMI risk score was calculated and classified into three groups: low risk (0–2), intermediate risk (3–4), and high risk (5–7). Systolic blood pressure, diastolic blood pressure, and heart rate were recorded upon arrival to our hospital and SI was calculated. In addition, Killip class on admission was recorded.

Laboratory data on admission including white blood cell count, hemoglobin level, and eGFR were recorded. Cardiac troponin I was measured using the second-generation VITROS® troponin I assay (Ortho-Clinical Diagnostics Inc., NJ, USA). The upper limit of normal for cardiac troponin I was 0.034  $\mu$ g/L, which represented the 99th percentile reference value.

Transthoracic echocardiography was performed in a standard manner during hospitalization. Left ventricular ejection fraction was obtained using either the Teichholz or biplane Simpson's method.

### 2.2. Coronary angiography

All patients underwent cardiac catheterization within 5 days of presentation. An independent cardiologist blinded to the clinical data reviewed all coronary angiography, and the assessment was compared to the primary assessment by the treating cardiologist. In the event of a discrepancy between the assessments, a third investigator made the final interpretation. Revascularization procedures including PCI and CABG were performed at the discretion of the treating physician. Obstructive CAD was defined as stenosis greater than or equal to 50% in the left main coronary artery and 70% in any other epicardial coronary arteries. In addition, coronary blood flow was graded according to TIMI criteria [11].

### 2.3. End points.

The primary end point was in-hospital mortality. The secondary end point was cardiogenic shock, defined as sustained systolic blood pressure less than 90 mmHg requiring either vasopressors or inotropes without other identifiable causes of shock status, such as sepsis or hypovolemia.

### 2.4. Statistic analyses.

In our present study, elevated SI was defined as  $SI \geq 0.7$  consistent with previous studies of STEMI populations [7,9]. Patients were divided into those with  $SI \geq 0.7$  and those with  $SI < 0.7$ . In addition, a subgroup

analysis was performed after excluding patients who presented with systolic blood pressure  $< 90$  mmHg.

Data was expressed as either a number (percentage) or median (interquartile range). Continuous variables were compared using the Wilcoxon rank sum test. Dichotomous variables were compared using the chi-squared test or Fisher's exact test. Two-sided  $p$  values  $< 0.05$  were considered statistically significant. All statistical analyses were performed using R software (version 3.0.1).

## 3. Results

Demographic, hemodynamic and laboratory data are summarized and presented in Table 1. Among 481 patients included in the final analysis, 103 patients (21.4%) had  $SI \geq 0.7$ . No statistically significant difference was observed in baseline characteristics between patients with  $SI \geq 0.7$  and those with  $SI < 0.7$ .

Systolic and diastolic blood pressure were lower in patients with  $SI \geq 0.7$  than those with  $SI < 0.7$ . Heart rate was significantly higher in patients with  $SI \geq 0.7$ . Patients with  $SI \geq 0.7$  had a lower hemoglobin level than those with  $SI < 0.7$ . Patients with  $SI \geq 0.7$  had a significantly lower left ventricular ejection fraction than those with  $SI < 0.7$ .

Angiographic characteristics and in-hospital procedures are summarized and presented in Table 2. No statistically significant difference was observed in the rate of multi-vessel disease or left main and/or three-vessel disease (LM/3VD). Pre-procedural TIMI flow was similar between the two groups. With respect to in-hospital procedures, although the rates of PCI were similar between the two groups, the rate of CABG was significantly lower in patients with  $SI \geq 0.7$ .

**Table 1**  
Demographic, hemodynamic and laboratory characteristics.

	Shock index $\geq 0.7$ ( $n = 103$ )	Shock index $< 0.7$ ( $n = 378$ )	$p$ value
<b>Demographics</b>			
Age (years)	66 [57–78]	66 [57–76]	0.94
Male	66 (64.1)	232 (61.4)	0.62
Body mass index (kg/m <sup>2</sup> )	26.7 [22.8–30.1]	27.8 [24.0–31.9]	0.14
Hypertension	77 (74.8)	285 (75.4)	0.89
Diabetes mellitus	42 (40.8)	147 (38.9)	0.73
Hyperlipidemia	60 (58.3)	216 (57.1)	0.84
Chronic kidney disease	32 (31.1)	127 (33.6)	0.63
Family history of coronary artery disease	26 (25.2)	73 (19.3)	0.19
Current smoker	21 (20.4)	86 (22.8)	0.61
Use of $\beta$ -blocker	40 (38.8)	176 (46.6)	0.16
Previous PCI	30 (29.1)	118 (31.2)	0.68
Previous CABG	16 (15.5)	45 (11.9)	0.33
Previous myocardial infarction	17 (16.5)	64 (16.9)	0.92
TIMI risk score			0.72
Low risk (0–2)	20 (19.4)	68 (18.0)	
Intermediate risk (3–4)	54 (52.4)	188 (49.7)	
High risk (5–7)	29 (28.2)	122 (32.3)	
<b>Hemodynamic and laboratory data</b>			
Systolic blood pressure (mmHg)	123 [112–135]	148 [133–165]	$< 0.001$
Diastolic blood pressure (mmHg)	74 [69–83]	82 [73–93]	$< 0.001$
Heart rate (beat/minute)	105 [94–115]	76 [66–87]	$< 0.001$
Shock index	0.80 [0.74–0.96]	0.53 [0.45–0.60]	$< 0.001$
Hemoglobin (g/L)	12.6 [11.1–14.1]	13.2 [11.9–14.2]	0.025
White blood cell count ( $10^9/L$ )	9.1 [7.5–11.1]	8.1 [6.5–10.1]	0.002
eGFR (mL/min/1.73m <sup>2</sup> )	77 [49–89]	71 [52–89]	0.66
Peak troponin I ( $\mu$ g/L)	1.17 [0.11–6.25]	0.61 [0.10–5.34]	0.32
Killip class on admission			0.060
Killip class 1 on admission	84 (81.6)	330 (87.3)	
Killip class 2 on admission	14 (13.6)	39 (10.3)	
Killip class 3 on admission	2 (1.9)	8 (2.1)	
Killip class 4 on admission	3 (2.9)	1 (0.3)	
Left ventricular ejection fraction (%)	56 [35–60]	60 [45–64]	0.035

Data are expressed as a number (percent) or median (interquartile range). CABG; coronary artery bypass grafting, eGFR; estimated glomerular filtration rate, PCI; percutaneous coronary intervention, TIMI; Thrombolysis in Myocardial Infarction.

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