Original Article

The Prognostic Yield of Admission Shock Index in Patients with ST-Segment Elevation Myocardial Infarction: SEMI-CI Study

Abstract

Background: Early identification of high-risk patients presenting with ST-segment elevation myocardial infarction (STEMI) helps prevent complications. The shock index (SI) is a bedside risk-stratification tool used in emergency departments. In this study, we aimed to assess the SI's predictive value for prognosticating in-hospital and one-year mortality, as well as one-year major cardiovascular events (MACEs). As secondary endpoints, we assessed the age SI's performance and the influence of prehospital transport factors on SI's predictive value. **Methods:** This prospective cohort study is named SEMI-CI and enrolled patients with STEMI who were referred to a cardiology hospital in Isfahan. We analyzed data on 867 patients with STEMI. Systolic blood pressure (SBP) and heart rate (HR) upon admission were used to calculate SI. Patients were divided into two groups based on SI, and 277 patients had SI > 0.7. **Results:** In-hospital death, one-year mortality, and MACE were more prevalent in those patients presenting with SI ≥ 0.7. However, after multivariate adjustment, SI was an independent predictor of in-hospital mortality and MACE, but it was not associated with one-year mortality. Furthermore, mortality rates increased from lower to higher age groups. Among patients transferred by emergency medical services to our hospital, SI showed prognostic implications for in-hospital mortality but not for one-year mortality. **Conclusions:** The current study showed that a positive SI and age SI are valuable risk-stratification tools to identify high-risk patients presenting with STEMI.

Keywords: *In-hospital mortality, MACE, one-year mortality, shock index, STEMI*

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Introduction

ST-segment elevation myocardial infarction (STEMI) is one of the most debilitating cardiovascular diseases. According to the Global Registry of Acute Coronary Events (GRACEs) follow-up program in 2015, STEMI accounts for 35% of all acute coronary syndrome (ACS) presentations.[1] Although the incidence and case-fatality rates of STEMI have declined in recent years,[2-4] the growing population size and aging of the population contribute to a rise in the number of individuals dealing with myocardial infarction complications.[5,6]

STEMI complications are of great importance. Patients with STEMI have a higher risk of experiencing some complications, such as death, recurrent MI, heart failure (HF), and unscheduled cardiovascular rehospitalization, compared to those with non-ST-segment elevation myocardial infarction (NSTEMI).[1] Sudden cardiac death, myocardial reinfarction, and HF are the single dominant causes of cardiac death.[7] Thus, the identification of high-risk patients is crucial for the management of this entity. Several scoring systems have been created to pinpoint high-risk STEMI patients. However, some of these systems are impractical for bedside use due to their complex and time-consuming calculations.[8] Allgower and Burri first introduced the SI in 1967, which is defined by HR (bpm) divided by SBP (mmHg).[9] The initial purpose of this index was to assess hypovolemia in the setting of septic and hemorrhagic shock.[9,10] SI is a simple-to-use, bedside risk-stratification tool that can identify high-risk patients soon after arrival.[11] The normal SI range is between 0.5 and 0.7 for healthy adults.

Moreover, both the modified shock index (MSI), calculated as the heart rate (HR) divided by the mean arterial pressure,[12] and the age-related shock

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index (age SI), which incorporates the patient's age into the SI,[13] are derived from the original SI.

SI has been studied mainly in critically ill patients, such as those with septic shock.[14-16] It also has predictive value in several other medical conditions, such as traumatic injuries,[17] pulmonary embolism,[18] post-partum hemorrhage,[19] and community-acquired pneumonia.[20] Elevated SI is also associated with mortality rates in patients with STEMI.[11,21-23] The SI is often calculated using hemodynamic parameters measured at the time of hospital admission. Meanwhile, prehospital transport factors, including primary treatment that patients receive before admission, may affect SI's predictive value.

Previous studies have evaluated the predictive power of SI in STEMI patients using different cut-off points. The study conducted by Spyridopoulos *et al*.[24] was evaluated the prognostic value of SI >1 in patients with STEMI treated with primary percutaneous intervention (PPCI). The study showed that a positive pre-PPCI SI significantly predicted long-term mortality. In another study conducted by Huang *et al*.,[25] it was reported that short-term cardiovascular events were higher in patients with admission SI >0.7. In patients with positive SI, seven-day and 30-day mortality rates increased 2.2-fold and 1.9-fold, respectively.

This study aims to investigate the utility of SI and age SI as independent predictors, highlighting their potential integration into clinical decision-making for patients presenting with STEMI. The primary endpoints of this study are in-hospital mortality, one-year mortality, and one-year major adverse cardiovascular events (MACEs). Additionally, we specifically examined the association of SI with short-term and long-term outcomes in patients transferred by ambulance, providing insights into this subset of STEMI patients.

Method

Study design and patient population

This prospective cohort study was conducted at a referral cardiology center in Isfahan, Iran. Individuals who presented with STEMI to our hospital between March 2016 and February 2017 were enrolled in the study. Our patient population consisted of individuals who were directly admitted to our hospital from the community and patients who were referred from the affiliated hospitals. STEMI diagnosis was defined as the following: (1) presence of chest pain in addition to dynamic 12-lead ECG changes, characteristic of STEMI, including new ST elevation in two contiguous leads of >0.1 mV in all leads except for leads V2-V3 or as for lead V2-V3, ST elevation ≥ 0.2 mV in men ≥40 years, ≥0.25 mV in men <40 years or ≥ 0.15 mV in women, or presence of new left bundle branch block and (2) an elevated Troponin I level above the 99th percentile reference value.[26,27]

Exclusion criteria were the presence of a severe infection or a diagnosis other than STEMI, absence of sinus rhythms at the time of arrival at the hospital, and age under 18. After excluding patients who met the criteria mentioned above, 867 patients with STEMI were included in this study.

All participants signed written informed consent. This study complied with the Helsinki Declaration and was approved by the review board of Isfahan University of Medical Sciences.

Data collection and definitions

All data were collected retrospectively. We obtained patients' baseline data, including age, gender, body mass index (BMI), current smoking status, and past medical histories such as other cardiovascular diseases (previous MI, stroke), diabetes mellitus (DM), hypercholesterolemia, and history of treated hypertension. Patients' admission vital signs (SBP and HR) were measured upon arrival at the hospital to calculate SI. Admission blood sugar (BS) and glycosylated hemoglobin (HbA1c) levels were recorded, in addition to the Killip class. Coronary blood flow in the culprit's vessel was assessed using the TIMI flow grading system pre- and post-percutaneous intervention (PCI). Patients with TIMI flow grades 0 and 1 were considered group 1, and those with TIMI flow grades 2 and 3 were considered group 2. Patients received in-hospital treatments according to the existing guidelines for managing STEMI. The treatment consisted of reperfusion strategies and medications like anticoagulants, antiplatelet medications, angiotensin-converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARBs), and statins. The SI was computed using the patient's admission HR and SBP. The age SI was calculated for all patients and then categorized into six groups. Age SI groups ranged between 10 and 60.

Hypercholesterolemia was defined as total cholesterol >200 mg/dL, or taking lipid-lowering agents. Patients with FBS ≥126 mg/dL, HbA1c ≥6.5%, or specific treatment use were defined as having DM. The following are the definitions of our endpoints. All-cause mortality was defined as cardiac and noncardiac death. MACE does not have an absolute definition. We described MACE as a composite of myocardial reinfarction, stent thrombosis, stroke, unstable angina, and HF.

Endpoints

Patients were followed up for one year after hospital discharge. Clinical follow-ups were performed through in-clinic visits or phone interviews. Our primary endpoints were in-hospital and one-year all-cause mortality rates, as well as MACE. The secondary endpoints included age SI association with one-year mortality and the influence of prehospital means of transport on SI's predictive value.

Statistical analysis

In the current study, we defined the elevated SI as SI ≥ 0.7, according to the previous studies of STEMI populations. (23) All data analyzes and descriptions were performed using IBM SPSS statistics (Version 25, SPSS Inc., Chicago, USA). Continuous variables are presented as mean ± SD, and categorical variables are given in numbers and percentages. We examined the normality of continuous variables via the Kolmogorov-Smirnov test. All continuous variables had a normal distribution. Categorical variables were compared with the Pearson Chi-square test or Fisher's exact test. To determine whether, after adjusting for age, gender, and other confounders, SI is still associated with in-hospital death, one-year mortality, and death, multivariate Cox proportional hazard regression was performed. The adjusted hazard ratio with 95% confidence intervals (CIs) was also calculated. A two-sided $P < 0.05$ was considered statistically significant.

Results

A total of 867 patients with STEMI, with a mean age of 60.9 ± 12.77, were included in this study. Of the total population, 26 patients were unavailable for a one-year follow-up. 81.9% of the study population was male. SI >0.7 was considered to be positive in this study. Among the total patient population, 277 patients had SI >0.7. No significant age difference was found between the two groups: normal and elevated SI groups. Table 1 shows patients' characteristics and demographics according to the value of the SI. Patients with SI higher than 0.7 were more likely males compared to patients with normal admission SI ($P = 0.001$). There was no meaningful difference between the two groups in terms of BMI ($P = 0.09$). The previous myocardial infarction and stroke had no statistically significant impact on SI at the time of admission ($P > 0.05$). Diabetic patients were more likely to have an admission SI higher than 0.7 ($P = 0.003$). The presence of other cardiovascular risk factors, such as hypercholesterolemia, hypertension, and smoking, was not related to elevated admission SI in our study population ($P > 0.05$). HR was significantly higher in patients with SI > 0.7 than those with SI < 0.7; however, systolic blood pressure (SBP) was significantly lower in patients with elevated SI.

On admission, worse Killip class, lower hemoglobin levels, and higher plasma glucose levels were more prevalent in patients with SI > 0.7 ($P < 0.001$). Patients with normal or high SI were comparable in terms of pre-PCI TIMI flow in the culprit's vessel ($P = 0.1$). However, patients with SI > 0.7 were more likely to have post-PCI TIMI grade flow 0 and 1 ($P = 0.035$).

Table 2 provides information on in-hospital management, including reperfusion therapies and medications. Although both groups were similar regarding thrombolysis and

Table 1: Baseline characteristics

Variables	All patients (867)	SI <0.7 (590)	SI ≥0.7 (277)	*P*
Age	60.9±12.77	60.67±12.54	61.45±13.24	0.404
Gender (male)	710 (81.9%)	500 (75.8%)	210 (84.7%)	0.001
BMI, Kg/m^2	26.3±4.05	26.5±4.04	25.9±4.07	0.09
Previous myocardial infarction	112 (14%)	70 (12.6%)	42 (17.1%)	0.089
Previous stroke	51 (6.1%)	37 (6.4%)	14 (5.4%)	0.58
Current smoker	342 (40.5%)	235 (40.4%)	107 (40.7%)	0.95
Diabetes mellitus	255 (29.4%)	155 (26.3%)	100 (36.1%)	0.003
Hypercholesterolemia	255 (36.6%)	171 (35.7%)	84 (38.5%)	0.47
Treated hypertension	295 (36.8%)	205 (37%)	90 (36.4%)	0.1
Systolic blood pressure at first presentation	127±27.25	136±24.6	108±22.31	<0.001
Heart rate	80.37±31.3	71±14.4	101±44.8	<0.001
Killip class				<0.001
I	796 (91.8%)	559 (94.8%)	237 (85.6%)	
II	50 (5.8%)	27 (4.6%)	23 (8.3%)	
III	5 (0.6%)	2 (0.3%)	3 (1.1%)	
IV	16 (1.8%)	2 (0.3%)	14 (5.1%)	
TIMI flow in culprit vessel PRE				0.1
TIMI flow in culprit vessel PRE (group 1)	389 (40.6%)	269 (40.6%)	120 (40.6%)	
TIMI flow in culprit vessel PRE (group 2)	266 (59.4%)	184 (59.4%)	82 (59.4%)	
TIMI flow in culprit POST				0.035
TIMI flow in culprit POST (group 1)	29 (95.5%)	15 (3.3%)	14 (7%)	
TIMI flow in culprit POST (group 2)	620 (4.5%)	435 (96.7%)	185 (93%)	
Earliest hemoglobin level	14.31±1.9	14.5±1.7	14±2.16	<0.001
Glucose plasma level	169.5±81	161±73	187.5±93	0.001

BMI, body mass index; TIMI, thrombolysis in myocardial infarction

PCI ($P > 0.05$), patients with SI > 0.7 received less heparin, antiplatelet therapy, and fewer beta-blockers, ACEI, and statins than those with normal SI ($P < 0.05$). Additionally, among patients who received an intra-aortic balloon pump (IABP), patients with positive SI were more likely to require IABP ($P = 0.001$).

Concerning the relationship between the SI and our study endpoints, the data analysis showed that compared with normal admission SI, a SI > 0.07 was an independent predictor of MACE within a one-year follow-up ($P = 0.011$). Furthermore, SI > 0.07 was significantly associated with both in-hospital all-cause mortality ($P < 0.001$) and long-term all-cause mortality ($P = 0.005$) [Table 3]. Even after adjusting for confounding factors, including age, gender, BMI, histories of cardiovascular diseases, and medications received during hospitalization by the Cox proportional hazards model, elevated admission SI was an independent predictor of in-hospital mortality (HR = 3.9, 95% CI 2.037 to 7.46, $P < 0.001$) [Table 4] and one-year MACE (HR = 1.67, 95% CI 1.09 to 2.54, $P = 0.018$) [Table 5]. However, it did not prognosticate one-year mortality anymore [Table 6]. Among age SI groups, one-year mortality increased from 0% to 13.8% across groups 2 and 6, respectively [Table 7].

A total of 315 out of 867 patients were transferred by ambulance. Among EMS-transferred patients, SI > 0.7 was associated with in-hospital death but not one-year mortality [Table 8]. The SI was adjusted for the length of time it took a patient to get to the hospital from home as a confounding factor. The results demonstrated a P value > 0.05 for time-adjusted SI in either group [Tables 9 and 10].

Discussion

Patients who survive STEMI are more likely to develop adverse complications. Early identification of high-risk patients presenting with STEMI is the best approach to prevent cardiovascular complications. The SI is a simple point-of-care (POC) risk-stratification tool[10] used in different clinical scenarios. SI is independent of the patient's subjective information. Thus, it is less predisposed to errors in assessing the patient's clinical status. Moreover, SI acts better than either SBP or HR alone in prognosticating short-term outcomes of STEMI.[25] In this study, we evaluated the prognostic

Table 2: In-hospital treatment characteristics in STEMI patients

Variable	All patients (867)	SI <0.7 (590)	SI >0.7 (277)	*P*
Type of initial reperfusion therapy				
Thrombolysis	396 (45.7%)	271 (45.9%)	125 (45.1%)	0.82
PCI	397 (45.8%)	276 (46.8%)	121 (43.7%)	0.4
IABP	5 (0.6%)	1 (0.2%)	4 (1.4%)	0.001
Inotrope	18 (2.1%)	8 (1.4%)	10 (3.6%)	0.03
Anticoagulants				
Heparin	767 (88.5%)	535 (90.7%)	232 (83.8%)	0.003
Antiplatelet therapy				
Aspirin	856 (99%)	586 (99.3%)	270 (97.5%)	0.044
Clopidogrel	858 (99%)	587 (99.5%)	271 (97.8%)	0.034
Other medication				
Beta-blockers	755 (87.1%)	537 (91%)	218 (78.7%)	<0.001
ACEI	465 (53.6%)	340 (57.6%)	125 (45.1%)	0.001
ARB	140 (16%)	98 (16.6%)	42 (15.2%)	0.59
Statin	832 (96%)	579 (98.1%)	253 (91.3%)	<0.001

PCI, percutaneous coronary intervention; IABP, intra-aortic balloon pump; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker

Table 3: Clinical outcomes of STEMI patients according to the shock index

Variable	All patients (867)	SI <0.7 (590)	SI >0.7 (277)	*P*
In-hospital mortality	72 (8.3%)	21 (3.6%)	52 (18.8%)	<0.001
One-year mortality	34 (4.4%)	17 (3.1%)	17 (7.7%)	0.005
Myocardia reinfarction	7 (0.9%)	4 (0.7%)	3 (1.4%)	0.41
Unstable angina	31 (4.1%)	17 (3.1%)	14 (6.5%)	0.036
Stent thrombosis	4 (0.5%)	4 (0.7%)	0 (0%)	0.58
Stroke	4 (0.5%)	3 (0. 5%)	1 (0. 5%)	1
Heart failure	33 (4.3%)	25 (4.6%)	8 (3.7%)	0.59
MACE	97 (12.2%)	59 (10.4%)	38 (16.9%)	0.011

MACE, major adverse cardiovascular event

Table 4: Predictors of in-hospital mortality in STEMI patients by multivariate Cox analysis

Variable	In-hospital mortality		*P*
	HR_S	95% CI	
SI ≥0.7 (vs SI <0. 7)	3.9	2.037–7.46	<0.001
Sex (female vs male)	2.17	1.117–4.21	0.022
Age (years)	1.04	1.01–1.06	0.008
History of stroke	1.51	0.6–3.84	0.38
History of hypertension	1.03	0.51–2.076	0.94
Thrombolysis	0.79	0.40–1.55	0.49
Aspirin	1.69	0.9–3.14	0.1
B-Blocker	0.94	0.45–1.97	0.87
Lipid-lowering agents	0.84	0.41–1.73	0.64
Diabetes mellitus	1.57	0.84-2.55	0.16

Table 5: Predictors of MACE in STEMI patients by multivariate Cox analysis

Variable	MACE		*P*
	HR_S	95% CI	
SI≥0.7 (vs SI<0. 7)	1.67	1.09–2.54	0.018
Sex (male vs female)	1.205	0.69–2.09	0.51
Age (years)	1.029	1.01–1.05	0.002
History of stroke	1.22	0.58–2.59	0.6
History of hypertension	1.61	0.99–2.63	0.56
Thrombolysis	0.79	0.51–1.22	0.29
Aspirin	1.22	0.8–1.9	0.36
B-Blocker	0.84	0.5–1.45	0.54
Lipid-lowering agents	0.9	0.55–1.48	0.69
Diabetes mellitus	0.88	0.55–1.41	0.61

Table 6: Predictors of one-year mortality of STEMI patients by multivariate Cox analysis

Variable	One-year mortality		*P*
	HR_S	95% CI	
SI ≥0.7 (vs SI <0.7)	1.99	0.94-4.19	0.07
Sex (male vs female)	1.14	0.51–2.52	0.75
Age (years)	1.10	1.06–1.15	<0.001
History of stroke	1.40	0.51–3.84	0.51
History of hypertension	2.72	1.19–6.62	0.03
Diuretics	2.79	1.05–7.4	0.04
Aspirin	1.08	0.51–2.29	0.84
B-Blocker	0.65	0.28–1.5	0.32
Lipid-lowering agents	0.53	0.22–1.24	0.14
Diabetes mellitus	1.70	0.8–3.62	0.17

implications of admission SI for clinical outcomes following STEMI.

To begin with, we defined an elevated SI as SI > 0.7. Our study revealed that patients with elevated SI are at higher risk of both in-hospital and one-year mortality. Besides, the incidence of long-term MACE was considerably higher in patients with high admission SI. After the multivariate adjustment, SI was not a predictor of one-year mortality. However, it was still an independent predictor of one-year MACE and in-hospital mortality. The sensitivity and specificity of this SI threshold were 71% and 72%, respectively.

Huang *et al*.[25] reported that short-term cardiovascular events, including all-cause mortality and MACE, were more significant in patients with high admission SI. An SI > 0.7 was considered elevated, with a sensitivity and specificity reported as 59.0% and 74.4%, respectively. In a study of 644 patients with STEMI, SI >0.8 was significantly associated with higher death rates.[21] Spyridopoulos *et al*.[24] studied 3049 STEMI patients undergoing primary PCI. Patients were followed up for a median of 454 days. The study suggested that an SI >1 measured pre-PCI invasively strongly predicts both in-hospital and long-term (up to four years) mortality. In another study, Abe *et al*.[28] studied the prognostic implication of SI on STEMI prognosis with a threshold of 0.66. They found that although in-hospital cardiac mortality and one-year MACE were more frequent in the elevated SI group, these patients were not different from normal SI patients in terms of the incidence of one-year cardiac events.

The association between SI and physiologic parameters may address its correlation with poor clinical outcomes in patients with STEMI. This index is inversely associated with left ventricular stroke volume, mean arterial pressure, and left ventricular stroke work.[29,30] Thus, an elevated SI represents organ failure, specifically cardiovascular collapse.[31] Cardiac imaging findings may further explain the higher rates of long-term events in high SI patients. Cardiac magnetic resonance has shown that SI is a marker of myocardial damage. Patients with admission SI > 0.7 have significantly larger myocardial "infarction size" and "areas at risk." These patients have higher amounts of microvascular obstructions as well.[23] Patients with microvascular obstruction are significantly more likely to experience future MACE, hospital admissions, and premature cardiac deaths.[32] According to these findings, SI can be used for the early recognition of patients on the verge of hemodynamic collapse. Therefore, it enables clinicians to improve prognosis by providing patients with timely management.

Diabetic patients were more likely to present with elevated SI after an episode of STEMI. It has been reported that adverse outcomes, such as HF and death, are more prevalent in diabetic patients who develop STEMI.[33] In addition, patients with SI > 0.7 had higher admission plasma glucose levels. Hyperglycemia upon admission is highly correlated with STEMI complications. In-hospital[34] and long-term[35] mortality rates are more prevalent in patients with higher mean initial blood glucose. High admission blood glucose reflects a hyperadrenergic state following the acute phase of MI.[36] This hyperadrenergic state triggers several events, such as an acute increase in free fatty acids,[37] impaired myocardial glucose uptake,[37] and free oxygen radicals[38] formed by hyperglycemia, which may worsen myocardial ischemia.

Table 7: One-year mortality and age SI

	Gender		Age group			One-year mortality
	Male	Female	<18	18–60	>60	
Age shock index						
Group 2 (10–20)	2 (4.5%)	42 (95.5%)	1 (2.3%)	38 (86.4%)	5 (11.4%)	0%
Group 3 (20–30)	30 (12.6%)	208 (87.4%)	0%	181 (76.1%)	57 (23.9%)	4 (1.7%)
Group 4 (30–40)	36 (14.9%)	206 (85.1%)	0%	129 (53.3%)	113 (46.7%)	4 (1.8%)
Group 5 (40–50)	38 (23.2%)	126 (76.8%)	0%	50 (30.5%)	114 (69.5%)	8 (5.5%)
Group 6 (>50)	51 (28.5%)	128 (71.5%)	0%	40 (22.3%)	139 (77.7%)	18 (13.8%)

Table 8: Shock index predictive value according to the mode of transport of STEMI patients

Variable	EMS patients		*P*	Non-EMS patients		*P*
	SI >0.7	SI <0.7		SI >0.7	SI <0.7	
In-hospital mortality	15	5	00	37	15	00
One-year mortality	11	6	0.28	12	11	0.007

Table 9: Time-adjusted shock index in patients transported using EMS

Variable	One-year mortality		*P*
Time to arrive at the hospital	HR_S	95% CI	
	1	1-1.003	0.17
Variable	**In-hospital mortality**		***P***
Time to arrive at the hospital	**HR_S**	**95% CI**	
	0.99	1.001–1.004	0.8

Table 10: Time-adjusted Shock index in STEMI patients transported by any way other than EMS

Variable	One-year mortality		*P*
	HRS	95%CI	
Time to arrive at the hospital	0.99	0.99-1002	0.5
variable	**In-hospital mortality**		***P***
	HRS	**95%CI**	
Time to arrive at the hospital	0.99	0.99-1002	0.5

Age is a predicting factor associated with clinical outcomes after STEMI.[24] Older people have a higher risk of developing poor outcomes following AMI. The age SI is a derivative of SI. Our study's values of the age SI groups showed that the rate of death increases from lower to higher age SI groups. Several studies have shown that age SI has a higher predictive power in several medical conditions, including trauma[31,39] and Emergency Severity Index level 3.[40] In Kim's study, age SI compared to either SI or MSI was a stronger predictor of in-hospital mortality in geriatric patients with traumatic injuries.[39]

Yu *et al*.[41] showed that age SI was similar to the GRACE score in prognosticating AMI's long-term outcomes. However, it acted better than admission SI and MSI in patients with AMI undergoing PCI. In a second study, age SI and age MSI were superior to SI and modified SI in predicting short- and long-term mortality rates in patients with STEMI undergoing PCI.[42] Conversely, one further study reported comparable SI, age SI, and MSI performance in patients admitted to a tertiary center with various medical conditions. The only advantage of the age SI over the two other indices was that it predicted the hospital's length of stay.[43]

Medications such as β-blockers, calcium channel blockers, and fluid therapy may affect a patient's SBP and HR. To evaluate the influence of prehospital treatment on SI performance, we compared the SI performance between the two groups of patients transferred to the hospital via non-EMS and those transferred via EMS. Positive SI was still an independent predictor of in-hospital mortality among patients transferred via EMS. However, similar to our total patient population, SI > 0.7 was not associated with one-year mortality rates in this group. Besides, our results outline that the time to get to the hospital does not significantly influence SI's predictive power. Hence, it could be suggested that clinicians can rely on SI to predict in-hospital mortality, irrespective of modes of transport.

Limitations

Our study included data on all-cause mortality, not cardiac mortality alone. Considering all-cause mortality as an endpoint may include noncardiac mortality cases, which subject our findings to bias. Although we investigated the predictive value of the SI in EMS-transported patients separately, we did not have access to data on the exact drugs patients may have received on their way to the hospital.

Conclusions

The current study showed that positive SI and age SI are valuable risk-stratification tools to identify high-risk patients presenting with STEMI. In conjugation with other clinical parameters, SI and age SI help in the early application of hemodynamic support and other treatment strategies, thereby modifying poor outcomes of STEMI. In patients transferred with EMS, the SI is still an independent predictor of in-hospital mortality in patients with STEMI. However, further studies are needed to evaluate the influence of prehospital factors on SI predictive value.

Ethical issues

This study was approved by the Ethical Committee of the Isfahan University of Medical Science (Ethical code: IR.MUI.MED.REC.1399.537). Written informed consent was obtained at the beginning of enrollment in the cohort study after explaining the study protocol.

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Conflicts of interest

There are no conflicts of interest.

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