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Relation between Shock Index, Severity of Coronary Artery Disease and Outcomes in ST Elevation Myocardial Infarction Patients

Mohamed Salama Sharban[1*], Ahmed Farouk Alaarag[1], Mona Adel Elsaidy[1] and Magdy Mohamed El-Masry[1]

[1]Department of Cardiology, Faculty of Medicine, Tanta University, Tanta, Egypt.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Coronary artery disease is considered a major cause of death in both male and female subjects in the developed world and carries a risk of several complications.
Multiple scores have been developed in order to set risk stratification and predict the outcomes for ischemic patients. Another scores have been developed in order to assess the severity of the coronary arteries lesions.
Methods: The prospective cross sectional cohort study included 68 consecutive patients with STEMI; they were divided into two groups based on the shock index at presentation. Group A: included 43 patients with SI <0.7. Group B: included 25 patients with SI > 0.7. All participants were subjected to trans-thoracic echocardiography, PPCI, SYNTAX score calculation and follow up during the hospitalization period.
Results: There was a significant myocardial damage in group B supported by the reduced LVEF and elevated serum troponin at presentation. There was a significant more coronary artery lesion severity in group B as assessed by the SYNTAX score. As regard in-hospital outcomes, patients in group B had the worst outcomes during the hospitalization period.

**Corresponding author: E-mail: dr.msalama9@gmail.com;*

Conclusions: Shock index is a useful and quick tool to predict the severity of the underling coronary artery disease and correlate with the SYNTAX score in patients with STEMI. Shock index is a good indicator of the hemodynamics and the extent of myocardial damage. Shock index is a quick bedside tool with a good prediction value for the in-hospital outcomes in STEMI patients undergoing PPCI.

Keywords: Shock index; coronary artery disease; ST elevation; myocardial infarction.

1. INTRODUCTION

According to the World Health Organization (WHO), 17.9 million people die each year from cardiovascular diseases (CVDs), an estimated 31% of all deaths worldwide. 85% of all CVDs are due to heart attacks and strokes [1].

Myocardial injury is defined as detection of an elevated cardiac troponin values (cTn) above the 99th percentile of the upper reference limit (URL). The injury is considered acute if there is a rise and/or fall of (cTn). The clinical definition of myocardial infarction (MI) denotes the presence of acute myocardial injury detected by abnormal cardiac biomarkers in the setting of evidence of acute myocardial ischemia [2].

Several risk stratification systems have been developed, such as thrombolysis in myocardial infarction (TIMI) and global registry of acute coronary events (GRACE), to estimate patients' prognosis and help physicians to identify those patients at higher risk for complications [3-5]. One problem when dealing with these systems is that they are time-consuming and difficult to perform routinely at bedside.

The shock index (SI), defined as the ratio between the heart rate (HR) and systolic blood pressure (SBP), was first introduced by Allgöwer and Burri, in 1967 [6,7] SI gives reliable data about the hemodynamic instability of the patient and is better than using HR alone, SBP alone or even better than some risk stratification systems e.g. Triage sort (TSO) for secondary triage in mass-casualty situation [8]. Number of studies have shown that SI can be used to assess the prognosis in different settings including STEMI [8-11].

SYNTAX scores was developed by (The synergy between percutaneous coronary intervention with Taxus and cardiac surgery (SYNTAX) Trial). This score classifies patients according to the severity of coronary artery disease depending on the complexity of the lesions, their location and numbers [12].

The aim of this study is to assess the relation between the shock index SI at time of presentation of ST elevation myocardial infarction patients undergoing primary percutaneous intervention, the severity of coronary artery lesion and the outcomes during hospitalization.

2. PATIENTS AND METHODS

This study was carried out on 68 patients from October 2019 till October 2020 at the cardiovascular department, Tanta University Hospital after approval from Ethical Committee and obtaining informed written consent. The study was conducted during the covid-19 pandemic during which thrombolytic therapy was the preferred choice for STEMI patients.

The study included 68 patients from the cardiovascular department, divided in to 2 groups depending on the shock index value. Group A: includes 43 patients with normal shock index at presentation (<0.7). Group B: Includes 25 patients with elevated shock index at presentation (> 0.7).

2.1 Inclusion Criteria

All patients aged from 42 to 80 years presented with ST-elevated myocardial infarction (STEMI) eligible for Primary Percutaneous Coronary Intervention (PPCI) were included in this study.

2.2 Exclusion Criteria

1. Cardiogenic shock at presentation which can be defined as persistent hypotension (SBP < 90 mm Hg) that did not respond to fluid titration and requires an intra-aortic balloon pump or intravenous inotropic therapy 104.
2. Arrhythmias with irregular heart rate including atrial fibrillation (AF) and atrial flutter.

3. Previous PCI or coronary artery bypass grafting (CABG).
4. Sinus bradycardia and second- or third-degree heart block (HB).
5. Patients on dialysis.
6. Patients with malignancy or bleeding disorders.

All patients included in this study were subjected to complete demographic and medical history including risk factors of ischemic heart disease, hypertension, diabetes mellitus, peripheral vascular disease, chronic kidney disease. Clinical examination including a twelve-lead electrocardiogram (ECG), Shock Index calculation **(**SI) was calculated to every patient at presentation using the ratio between the heart rate (HR) and systolic blood pressure (SBP) (Shock Index = Herat Rate / Systolic Blood Pressure) with normal value ranging from 0.5 to 0.7 [6,13] (106,102), trans-thoracic echocardiography, PPCI, SYNTAX score [12] calculation and follow up during the hospitalization period.

2.3 Statistical Analysis

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. Qualitative data were described using number and percent. The Kolmogorov-Smirnov test was used to verify the normality of distribution.. Quantitative variables were expressed as using range (minimum and maximum), mean, standard deviation (SD) and median. Correlation coefficients were used to assess the correlation between different variables with value of + 1.0 means perfect positive correlation, value of – 1.0 means perfect negative correlation and value of 0.00 means no correlation. Significance of the obtained results was judged at the 0.05 level.

3. RESULTS

There was no significant difference between the two groups as regard basal demographic data. Table 1.

There was a significant difference between the two groups as regard clinical presentation (SBP, DBP, MAP, HR, LVEF %, dyspnea and Killip class). There was no significant difference between the two groups as regard the typicality of chest pain at presentation and RBS. **Error! Reference source not found.**.

There was a significant difference between the two groups as regard serum troponin (P = 0.001). There were no significant difference between the two groups regarding other laboratory parameters **Error! Reference source not found.**.

There were no subjects with left main coronary artery disease. There was a significant difference between the two groups as regard total ischemic time with patients in group B tend to take longer time. There was no significant difference in the contrast volume injected into the subjects, culprit vessel and multivessels between both groups. There was a significant difference between the two groups as higher percentage of subjects in group B tend to have TIMI grade flow II than those of group A. **Error! Reference source not found.**.

Table 1. Comparison between the studied groups regarding demographic data

Demographic Data	Group A N= 43	Group B N= 25	Test	p. value
Age (Range/Mean ± SD)	(42 – 80 / 61.42 ± 7.56)	(47 – 70 /59.32 ± 6.63)	T: 1.153	0.253
BMI (Range/Mean ± SD)	(24 – 32 /28.16 ± 1.90)	(25 – 32 /27.92 ± 1.89)	T: 0.509	0.613
Sex (male) (n,%)	27 (62.8%)	21 (84.0%)	X2: 3.425	0.064
(female) (n,%)	16 (37.2%)	4 (16.0%)		
DM (n, %)	17 (39.5%)	15 (60.0%)	X2: 2.658	0.103
HTN (n, %)	21 (48.8%)	16 (64.0%)	X2: 1.465	0.226
Smoking (n,%)	20 (46.5%)	12 (48.0%)	X2: 0.014	0.906
History of IHD (n, %)	6 (14.0%)	2 (8.0%)	X2: 0.540	0.463
History of PVD (n, %)	3 (7.0%)	0 (0%)	X2: 1.825	0.177
Family History of IHD (n, %)	7 (16.3%)	2 (8%)	X2: 0.944	0.331

BMI: Body mass index, DM: Diabetes mellitus, HTN: Hypertension, IHD: Ischemic heart disease, PVD: Peripheral vascular disease S.D.: standard deviation

There was a significant difference in SYNTAX score as patients in group B tend to have more complex coronary lesions in the angiography making them to have a higher SYNTAX score as compared to the patients in group A. **Error! Reference source not found.**.

There was a significant difference between the two groups in MACEs as the subjects in group B had the worst outcomes compared to group A. There was no significant difference in the time at the hospital, mortality and CIN between both groups. **Error! Reference source not found.**.

The shock index and its components of blood pressure and heart rate are significant variables. SYNTAX score is also of a significant importance. From all the laboratory parameters in this study, serum troponin has a significant importance. **Error! Reference source not found.**3.

Table 1. Comparison between the two studied groups according to clinical presentation, laboratory parameters, angiographic data, SYNTAX score and in hospital outcomes

Clinical Presentation	Group A N= 43	Group B N= 25	Test	p. value
Clinical Presentation				
SBP (Range/Mean ± SD)	(110 – 210 / 139.53 ± 23.23)	(90 – 155 / 112.68 ± 16.81)	T 5.056	0.001*
DBP (Range/Mean ± SD)	(60 – 110 / 83.35 ± 10.76)	(60 – 90 / 70.28 ± 9.07)	T 5.107	0.001*
MAP (Range/Mean ± SD)	(76 – 143 / 101.74 ± 14.53)	(66 – 111 / 83.92 ± 11.60)	T 5.234	0.001*
HR (Range/Mean ± SD)	(55 – 105 / 76.86 ± 11.22)	(71 – 160 / 99.32 ± 21.47)	T 5.674	0.001*
RBS (Range/Mean ± SD)	(95 – 314 / 165.07 ± 68.41)	(45 – 320 / 172.72 ± 72.48)	T 0.435	0.665
LVEF % (Range/Mean ± SD)	(39 – 67 / 54.05 ± 6.62)	(30 – 68 / 48.28 ± 8.23)	T 3.164	0.002*
Chest pain Atypical (n, %)	1 (2.3%)	0 (0%)	X^2: 0.590	0.442
Chest pain Typical (n, %)	42 (97.7%)	25 (100%)		
Dyspnea (n, %)	2 (4.7%)	5 (20%)	X^2: 4.033	0.045*
Killip Class I (n, %)	42 (97.7%)	20 (80%)	X^2: 6.139	0.013*
Killip Class II (n, %)	1 (2.3%)	5 (20%)		
Laboratory Parameters				
Troponin (ng/ml) (Range/Mean ± SD)	(0.1–2.9 / 0.97 ± 0.47)	(0.1 – 3.6 / 1.48 ± 0.71)	T 3.570	0.001*
CKMB (U/L) (Range/Mean ± SD)	(35 – 697 / 111.72 ± 106.20)	(25 – 239 / 129.26 ± 56.40)	T 0.764	0.448
Serum Creatinine (mg/dl) (Range/Mean ± SD)	(0.56 – 1.46 / 0.88 ± 0.22)	(66 – 111 / 83.92 ± 11.60)	T 0.002	0.999
Serum Urea (mg/dl) (Range/Mean ± SD)	(18 – 49 / 28.36 ± 7.45)	(17 – 129 / 34.60 ± 23.60)	T 1.608	0.113
48 Hours Sr Cr (mg/dl) (Range/Mean ± SD)	(0.51–1.6 / 0.95 ± 0.25)	(0.7 – 2.1 / 1.02 ± 0.33)	T 1.010	0.316
Hemoglobin (g/dl) (Range/Mean ± SD)	(10.3 – 15.7 / 13.96 ± 1.35)	(9.8 –16.5 / 13.99 ± 1.54)	T 0.090	0.929
Platelets (10^3 cell/cmm) (Range/Mean ± SD)	(138 – 296 / 231.0 ± 55.32)	(138 – 326 / 228.68 ± 47.74)	T 0.175	0.862
TLC (10^3 cell/cmm) (Range/Mean ± SD)	(3.7 – 15.3 / 7.36 ± 2.50)	(4.9 – 22.7 / 8.44 ± 3.98)	T 1.370	0.175
ALT (U/L) (Range/Mean ± SD)	(9 – 60 / 28.85 ± 10.07)	(16 – 206 / 41.20 ± 40.81)	1.896	0.062
AST (U/L) (Range/Mean ± SD)	(10 – 123 / 37.29 ± 23.20)	(22 – 441 / 62.92 ± 87.59)	T 1.821	0.073
Angiographic Data				

Clinical Presentation		Group A N= 43	Group B N= 25	Test	p. value
Total ischemic time (min) (Range/Mean ± SD)		(90 – 400) / 187.67 ± 70.33)	(90 – 360 / 229.60 ± 69.91)	T: 2.375	0.020*
Contrast Volume (ml) (Range/Mean ± SD)		(150 – 270 / 187.91 ± 35.74)	(130 – 300 194.20 ± 42.22)	T: 0.655	0.515
Culprit vessel	LAD (n, %)	19 (44.2%)	18 (72%)	X^2: 5.072	0.079
	LCX (n, %)	9 (20%)	2 (8%)		
	RCA (n, %)	15 (34.9%)	5 (20%)		
Multivessels		20 (46.5%)	17 (68%)	X^2: 2.943	0.086
Final TIMI Flow	II	1 (4%)	4 (16%)	X^2: 4.339	0.037*
	III	42 (97.7%)	21 (84%)		
SYNTAX Score					
Syntax Score (Range/Mean ± SD)		(4 – 26) / 12.56 ± 5.94)	(6 – 40.5 / 20.00 ± 8.96)	T: 4.118	0.001*
In Hospital Outcomes					
MACE		2 (4.7%)	5 (20%)	X^2 4.033	0.045*
All-Cause Mortality		0 (0%)	2 (8%)	X^2 3.544	0.060
CIN		4 (9.3%)	4 (16%)	X^2 0.683	0.408
Number of Days at Hospital (Range/Mean ± SD)		(2 – 6 / 2.35 ± 1.00)	(2 – 7 / 2.72 ± 1.67)	T 1.149	0.255

SBP: Systolic blood pressure DBP: Diastolic blood pressure MAP: Mean arterial pressure HR: Heart rate RBS: Random blood sugar LVEF: Left ventricular ejection fraction. CKMB: creatine kinase myocardial band TLC: total leukocyte count ALT: alanine aminotransferase, AST: Aspartate Aminotransferase LAD: left anterior descending LCX: left circumflex, RCA: right coronary artery TIMI: thrombolysis in myocardial infarction MACE: major adverse cardiac event CIN: contrast induced nephropathy MACE: major adverse cardiac event, CIN: contrast induced nephropathy S.D.: standard deviation

Table 2. Multivariate regression analysis of all predictors of MACE

Variable	OR	95% confidence interval	P Value
SBP	0.352	0.154 – 0.652	0.012*
DBP	0.443	0.307 – 0.654	0.031*
MAP	0.652	0.328 – 0.754	0.028*
HR	2.324	1.658 – 5.637	0.036*
LVEF %	0.637	0.348 – 2.521	0.106
Troponin (ng/ml)	1.657	1.257 – 4.205	0.041*
Total ischemic time (min)	1.865	0.579 – 2.364	0.284
Shock Index	2.506	1.628 – 5.954	0.017*
Syntax Score	1.674	1.201 – 3.258	0.025*
Killip Class	0.609	0.219 – 2.653	0.314
Final TIMI Grade Flow	0.419	0.310 – 5.309	0.307

SBP: Systolic blood pressure DBP: Diastolic blood pressure MAP: Mean arterial pressure, HR: Heart rate LVEF: Left ventricular ejection fraction TIMI: thrombolysis in myocardial infarction OR: odds ratio

Table 4. Correlation coefficient between SI and different variables

Variable	Shock Index	
	r	p
Age	-0.164	0.181
LVEF %	-0.378	0.001*
Troponin (ng/ml)	0.317	0.008*
Syntax Score	0.541	0.001*
Total ischemic time (min)	0.204	0.095
Sex	0.092	0.457
DM	0.153	0.138
HTN	0.139	0.259
Smoking	-0.015	0.907

There was a significant negative correlation between SI and LVEF as the LVEF tends to decrease with increased values of SI. There was a significant positive correlation between SI and serum troponin indicating that the increase in SI was accompanied by more myocardial damage as assessed by the elevated levels of serum troponin. There was a significant positive correlation between SI & SYNTAX score showing an increase in coronary arteries lesion severity accompanying the increase in SI values. Table 3 (r = 0.317, p = 0.008).

4. DISCUSSION

The concept of shock index (SI), defined as the ratio of heart rate to systolic blood pressure, has been coined by Allgower and Burri. It has been originally used to evaluate the degree of hypovolemia in hemorrhagic and infectious shock states. Then SI has been widely used for predicting outcomes in other critically ill patients, for example, those with severe sepsis and pulmonary embolism [14].

Recently several studies have revealed that high SI is a risk factor for acute myocardial infarction (AMI) patients, particularly for the ST elevated myocardial infarction patients. Huang and his colleagues explored that patients with SI ≥0.7 had a 2.2-fold increased risk of 7-day all-cause mortality and 1.9-fold increased risk of 30-day all-cause mortality. Another study has demonstrated that admission SI ≥0.66 were identified as an independent predictor of major adverse cardiac events (MACEs) with a cumulative hazard ratio for 5-year MACEs of 2.14. Elevated SI has also been shown as a risk factor of in-hospital mortality in patients undergoing primary percutaneous coronary intervention (PCI) [15].

Studies showed that SI was strongly associated with in-hospital mortality in patients with ACS following primary PCI, and SI ≥ 0.66 representing a cutoff value for clinical prediction was demonstrated in several studies [16].

Our results were in agreement with study of Shangguan et al. who studied the shock index (SI) and modified shock index (MSI) as predictors for 7-day outcomes for a number of 160 STEMI patients as they reported that there was high significant difference regarding SBP, DBP and heart rate [17].

Furthermore, Abe et al. have retrospectively studied 680 patients with acute myocardial infarction who received PCI and compared their admission shock index to the long term outcomes. Their work revealed that there was high significant difference among the studied groups regarding SBP, DBP and heart rate [18].

In the study of Reinstadler et al., there were 791 patients with STEMI treated with PPCI. Their work aimed to establish a relation between the admission shock index and the degree of myocardial damage as assessed by the cardiac magnetic resonance imaging CMR and the clinical outcomes. They found that patients with elevated admission shock index (n=321 [40.6%]) had a significantly larger area-at-risk (37.6 [27.8–50.4] % of left ventricular volume [LV] vs. 34.3 [24.5–46.0] % LV, P=0.02), larger infarct size (19.5 [10.7–28.0] % LV vs. 14.9 [7.7–22.3] % LV, P<0.001). Also, elevated admission shock index was associated with increased rates of MACEs at 1 year [19].

Primary treatment for patients with acute STEMI is fibrinolysis or primary PCI . The use of primary PCI has improved the outcome of STEMI patients significantly. Ischemic heart disease is the most common contributor to left ventricular dysfunction. The extent of left ventricular (LV) function varies considerably among patients with extensive coronary disease, and clinical and angiographic factors associated with LV impairment are poorly characterized. Specifically, whether clinical, demographic and angiographic characteristics differ among patients and are predictive of LV ejection fraction has not been determined [20].

Findings of our results were in line with study of Reinstadler et al. as they reported that there was no significant difference between the studied groups regarding stent implantation [19].

Our results were in line with study of Abreu et al. as they included 1234 patients with STEMI admitted to their center or referred to them for emergent PCI. They divided the patients into two groups based on the modified shock index MSI at presentation and retrospectively followed them for 6 months. They reported that there was a significant difference among their studied groups regarding serum troponin level [21].

However, Abe et al. reported that an admission SI <0.66 was found in 504 patients (normal SI group), whereas 176 patients had an admission SI >0.66 (elevated SI group). At admission, patients in the elevated SI group had significantly

lower hemoglobin (Hb) levels and estimated glomerular filtration rate (eGFR), significantly higher brain natriuretic peptide (BNP) levels than those in the normal SI group [18]. Their results can be attributed to the higher age in their study groups (mean age 67.2 year S.D. ± 12.4). Nevertheless, anemic patients tend to have reflex tachycardia which can lead to higher values of SI [18].

According to, Shangguan et al. demonstrated that demonstrated that SI of 0.7 or greater is a useful predictor for 7-day outcomes in the patients with STEMI [17]. This result was also concomitant with the work of Huang et al. who included 7187 patients with STEMI, in which SI of 0.7 or greater indicated greater 7- and 30-day all-cause mortality and MACE [13]. Bilkova et al. have retrospectively studied 644 STEMI patients among whom 92% had PPCI and 7% had rescue PCI. Their results showed that SI of 0.8 or more is strong independent predictor of short-term and/or long-term outcome in patients with STEMI [8].

In contrast with our study, findings of Shangguan et al., as they reported that elevated SI, defined as SI≥0.7, and elevated modified shock index, defined as MSI ≥ 1.4, were both significantly associated with higher rates of in-hospital mortality. Their results may be explained by the older age of the subjects in the study and the inclusion of patients with higher Killip classes (III & IV). Although they only calculated the time from the onset of symptoms till admission without referring to the time of PPCI, their patients took longer time (median = 5 hours) [17].

Regarding Huang et al., those who presented with elevated admission SI (>0.7) had greater incidence of short-term cardiovascular events compared with those with normal admission SI (<0.7) in patients with STEMI. Second, after multivariate adjustment, elevated admission SI (>0.7) was still an independent risk factor predicting the short-term outcomes. Third, the prognostic discriminatory capacity of admission SI is moderate for 7-day all-cause mortality but limited for 30-day all-cause mortality. Their study provided a simple indicator for predicting the short-term, especially for acute phase outcomes in patients with STEMI [13].

5. CONCLUSIONS

Calculating Shock index at presentation of patient with STEMI is a useful and quick tool to predict the severity of the underling coronary artery disease and correlate with the SYNTAX score. Shock index at presentation is a good indicator of the hemodynamics and the extent of myocardial damage in STEMI patient as assessed by elevated serum troponin level and reduced LVEF. Shock index at presentation is a quick bedside tool with a good prediction value for the in-hospital outcomes in STEMI patient undergoing PPCI.

6. LIMITATIONS OF STUDY

Our findings are based on observations in a relatively small number of subjects. A prospective study in a larger patient population is required to validate the relation between the shock index, severity of the coronary artery disease and the outcomes in STEMI patient.

The study was held during the hospitalization period of the patients, a longer period of follow up is recommended to establish the relation of the shock index at presentation and the outcomes in STEMI patient.

CONSENT AND ETHICAL APPROVAL

This study was carried out on 68 patients from October 2019 till October 2020 at the cardiovascular department, Tanta University Hospital after approval from Ethical Committee and obtaining informed written consent.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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