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Original article

Shock Index as a Predictor for Coronary Slow/No Reflow in Patients with Acute STEMI Undergoing Primary Percutaneous Coronary Intervention

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Abstract

Background: shock index (SI), defined as heart rate divided by systolic blood pressure (mmHg), was originally described by Allgöwer et al. in 1967. SI was developed as a simple tool for the detection of circulatory collapse in hemodynamically unstable patients. Elevated SI has also been shown as a risk factor of in-hospital mortality in patients undergoing PPCI. **Aim and objectives:** to evaluate the value of shock index as a predictor for angiographic slow/no reflow in patients with STEMI after primary PCI. **Subjects and methods:** This study was conducted at Cardiology department – Egypt Air Hospital. Eighty patients were admitted to coronary care unit (CCU) after diagnosis of STEMI and PCI was done within 24 hours from starting of symptoms. CT chest was routinely done to all patients before admission for reveal of COVID-19 virus infection. The study period started from July 2021 till March 2023. **Results:** there was a statistically significant difference between the study population regarding comparison of type of STEMI between cases with normal and cases with slow or no reflow, comparison of conventional 2D ECHO after PCI between cases with normal and cases with slow or no reflow, comparison of Shock index between cases with normal and cases with slow or no reflow and the roc curve. **Conclusion:** Based on our findings, shock index was significant predictor for slow/no coronary reflow in patients with STEMI after primary PCI and was found to be associated with in hospital morbidity and mortality.

Keywords: Shock; Coronary Slow/No Reflow; Acute STEMI; Percutaneous Coronary Intervention

Introduction

ST segment elevation myocardial infarction (STEMI) is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent electrocardiographic (ECG) ST elevation and subsequent release of biomarkers of myocardial necrosis. The great majority of these patients will show a typical rise of biomarkers of myocardial necrosis [(1)].

STEMI occurs from occlusion of one or more of the coronary arteries that supply the heart with blood. The cause of this abrupt disruption of blood flow is usually plaque rupture, erosion, fissuring or dissection of coronary arteries that results in an obstructing thrombus. The major risk factors for ST-elevation myocardial infarction are dyslipidemia, diabetes mellitus, hypertension, smoking, and family history of coronary artery disease **(2)**.

Slow flow and the no-reflow phenomenon are feared complications after PCI. In general terms, both phrases refer to impaired epicardial coronary flow and myocardial perfusion despite patency of the epicardial arteries during PCI. Slow flow and no-reflow usually manifest as a failure of the affected artery to opacify after angioplasty or stenting of the occluded segment during acute myocardial infarction (AMI) (no-reflow), or as a reduction in flow in the affected artery after PCI of the occluded segment (slow flow). No-reflow is associated with a worse prognosis and has been shown to be an independent predictor of death, MI, and impaired left ventricular function **(3)**.

In consideration of the phenomenon of slow/no reflow as a severe complication of catheterization laboratory and a pivotal indicator for clinical outcomes, the aim of this study is to explore whether Shock index effectively foresee angiographic slow/no reflow in patients with STEMI after primary PCI **(4)**.

Shock index is a marker assessing the hemodynamic state, which is calculated as heart rate (HR) divided by systolic blood pressure (SBP)with a normal range of 0.5 to 0.7 in healthy adults. patients with elevated SI, even with normal blood pressure and heart rate, should be paid more attention for the high risk of shock **(5)**.

The aim of this study was to evaluate the value of shock index as a predictor for angiographic slow/no reflow in patients with STEMI after primary PCI.

Patients and methods

This is a case-control clinical study that was conducted at Cardiology department – Egypt Air Hospital. Eighty patients were admitted to coronary care unit (CCU) after diagnosis of STEMI and PCI was done within 24 hours from starting of symptoms. CT chest was routinely done to all patients before admission for reveal of COVID-19 virus infection. The study period started from July 2021 till March 2023. The study design was approved by the ethical committee of Banha University Hospitals, Banha University {M.S.6.6.2021}. .

STEMI patients were divided into two groups according to coronary angiography during primary PCI: 1st group as a control group with normal flow during PCI and 2nd group as a study group with slow/ no reflow during PCI.

Inclusion criteria: All Patients presenting with Acute ST segment elevation Myocardial infarction (STEMI) or new onset left bundle branch block (LBBB) within 24 hours from starting of symptom that undergoing primary PCI as a revascularization treatment irrespective of age, gender, race and clinical severity.

Exclusion criteria: Patient with hematologic diseases and malignant tumors, or those with severe renal impairment (GFR< 29 ml/min), or hepatic dysfunction, or with STEMI onset ≥ 24 hours.

Ethical Approval: The purpose of the research was explained to all patients, informed consent was signed from all patients included in the study. Participants' rights to decline participation or to withdraw from the research once it had started and the patient's confidentiality was saved.

All patients were subjected to the following measures: Full history taking, clinical examination, well standardized 12-leads resting electrocardiogram (ECG), laboratory investigations, echocardiography and coronary angiography and 1ry PCI (Culprit lesion (IRA), TIMI flow and TIMI Thrombus Grade).

Morbidity among patients with STEMI was defined as post MI significant arrhythmias or heart failure or cardiogenic shock during the hospital stay.

Mortality was defined as in hospital death among study population.

Statistical analysis

Statistical analysis was done by SPSS v26 (IBM Inc., Chicago, IL, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing unpaired Student's t- test. Qualitative variables were presented as frequency and percentage (%) and were analyzed utilizing the Chi-square test or Fisher's exact test when appropriate. A two tailed P value < 0.05 was considered statistically significant.

Results

Patients with slow/ no coronary reflow had significantly higher incidences of hypertension and Covid-19 infection when compared to patients with normal coronary flow (p=0.042* and 0.004* respectively). Patients with slow/ no coronary reflow had significantly higher incidence of Killip classification III/IV when compared to patients with normal coronary flow (76% vs 12.7%; p<0.001*) **[Table, 1].**

No significant difference was reported between both groups regarding type of STEMI and infracted related artery **[Table, 2].**

Patients with slow/ no coronary reflow had significantly lower EF as well as higher EDD, ESD, and WMSI when compared to patients with normal coronary flow (p=<0.001*, <0.001*=0.001*, and <0.001* respectively) **[Table, 3].**

Patients with slow/ no coronary reflow had significantly higher incidences of congestive heart failure, cardiogenic shock, ventricular arrhythmia and death when compared to patients with normal coronary flow (p<0.001*, p<0.001*, p0.001*and 0.028* respectively) **[Table, 4].**

Patients with slow/ no coronary reflow had significantly higher shock index when compared to patients with normal coronary flow. Moreover, incidence of shock index above 0.66 was significantly higher in patients with slow/ no coronary reflow when compared to patients with normal coronary flow (p <0.001*) **[Table 5,6].**

ROC curve revealed that cut off value 0.6220 of shock index was a significant predictor for slow/ no reflow coronary flow with AUC 0.968 (p<0.001*) with 96.4% sensitivity, 92.3% specificity, and 96.3% accuracy **[Figure, 1].**

Table (1): comparison of demographic characteristics, medical history Killip classification between cases with normal flow and cases with slow/ no reflow

	Normal flow N=55(%)	**Slow /No reflow N=25(%)**	**Test of significance**
Age/ years	57.91±13.02	60.08±8.07	t=0.768 *p*=0.445
Sex			
Female	13(23.6)	2(8.0)	$\chi2$=2.76
Male	42(76.4)	23(92.0)	*p*=0.097
Hypertension			$\chi2$=4.16
	31(56.4)	20(80.0)	*p*=0.042*
Smoker	15(27.3)	9(36.0)	$\chi2$=0.623 *p*=0.430
Diabetes	29(52.7)	15(60)	$\chi2$=0.367 *p*=0.544
Dyslipidemia			
	18(32.7)	8(32.0)	$\chi2$=0.004 *p*=0.949
COVID-19			
-VE	54(98.2)	20(80.0)	$\chi2$=8.19
+VE	1(1.8)	5(20.0)	*p*=0.004*
Killip classification			
I/II	48(87.3)	6(24.0)	$\chi2$=31.37
III/IV	7(12.7)	19(76.0)	*p*<0.001*

*statistically significant t: Student t test, $\chi2$: Chi-Square test

Table (2): comparison of type of STEMI between cases with normal and cases with slow or no reflow

Type of STEMI	Normal flow N=55(%)	Slow /No reflow N=25(%)	Test of significance
Anterior STEMI			
	32(58.2)	18(72.0)	$\chi2$=1.40 *p*=0.237
Lateral STEMI			
	2(3.6)	0	$\chi2^{FET}$=0.932 *p*=1.0
Inferior STEMI			
	21(38.2)	7(28.0)	$\chi2$=0.783 *p*=0.454

$\chi2$:Chi-Square test; FET: Fischer exact test; *statistically significant

Table (3): comparison of conventional 2D ECHO after PCI (within 12h from admission) between cases with normal and cases with slow or no reflow

Conventionnal 2D ECHO after PCI	Normal flow N=55(%)	Slow /No reflow N=25(%)	Test of significance
EF%	49.18±4.75	42.72±5.44	t=5.39 *p*<0.001*
EDD (cm)	46.80±3.14	50.44±3.35	t=4.71 *p*<0.001*
ESD (cm)	30.80±5.82	35.84±6.73	t=3.42 *p*=0.001*
WMSI	1.37±0.181	1.725±0.148	t=5.25 *p*<0.001

$\chi2$:Chi-Square test , FET: Fischer exact test *statistically significant please write all the abbreviation mentioned in the table here

Table (4): comparison of in hospital out come between cases with normal and cases with slow or no reflow

In hospital out come	Normal flow N=55(%)	Slow /No reflow N=25(%)	Test of significance
Congestive heart failure	7(12.7)	14(56.0)	$\chi2$=16.63 *p*<0.001*
Cardiogenic shock	2(3.6)	15(60.0)	$\chi2$=32.63 *p*<0.001*
Ventricular arrhythmia	5(9.1)	12(48.0)	$\chi2$=15.55 *p*<0.001*
Death	0(0.0)	3(12.0)	$\chi2^{FET}$=6.86 *p*=0.028*

$\chi2$:Chi-Square test; FET: Fischer exact test; *statistically significant

Table (4): comparison of Shock index between cases with normal and cases with slow or no reflow

	Normal flow N=55(%)	Slow /No reflow N=25(%)	Test of significance
Shock index			
<0.66	53(96.4)	2(8.0)	$\chi2^{FET}$=62.47
≥0.66	2(3.6)	23(92.0)	*p*<0.001*
Mean±SD	0.539±0.075	0.839±0.114	

FET: Fischer exact test; *statistically significant

Table (5): binary logistic regression in prediction of slow or no reflow among studied cases

	B	*p* value	AOR (95%CI)
Hypertension			
-ve	1.05	0.078	2.84(0.891-9.07)
+ve			
COVID-19			
-VE	2.49	0.029*	12.11(1.28-17.89)
+VE			
Killip classification			
I/II (r)	2.35	0.002*	10.47(2.42-45.41)
III/IV			
TIMI Thrombus Grade			
<3 (r)	3.86	0.001*	47.54(4.95-55.64)
≥3			
Shock index	20.56	<0.001*	84.5(25.4-96.5)

Overall % predicted=95%; AOR: Adjusted odds ratio; *statistically significant *p*<0.05

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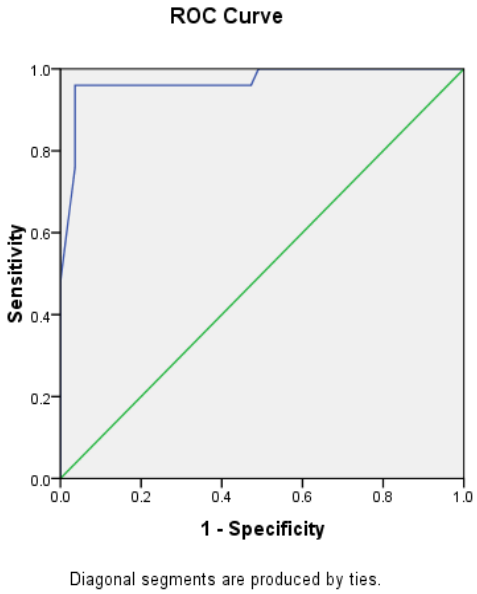

Figure (1): ROC Curve of shock index in differentiating between no-reflow and normal flow cases.

SELECTED CASE

Patient No (5): male patient, 53 years old, hypertensive, diabetic, dyslipidemic, non-smoker presented by retrosternal compressing chest pain of 6 hours' duration. **Physical examination revealed that his** blood pressure was 100/60 mmHg, heart rate was 90bpm regular sinus rhythm with intact peripheral pulsations, shock index was 0.9. **On auscultation there was no detectable murmur or additional heart sound.** ECG: NSR with pathological Q and ST elevation in V1 through V6.

ECG Findings:

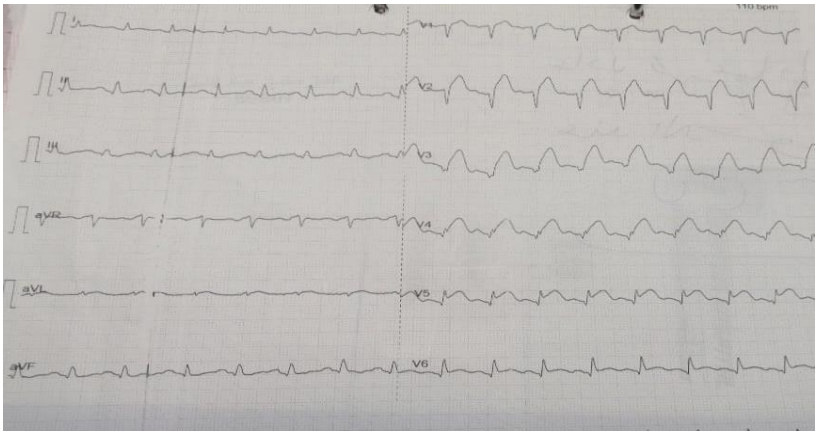

Figure (2): 12 Leads standard surface ECG showing Anterior STEMI Echocardiographic Findings

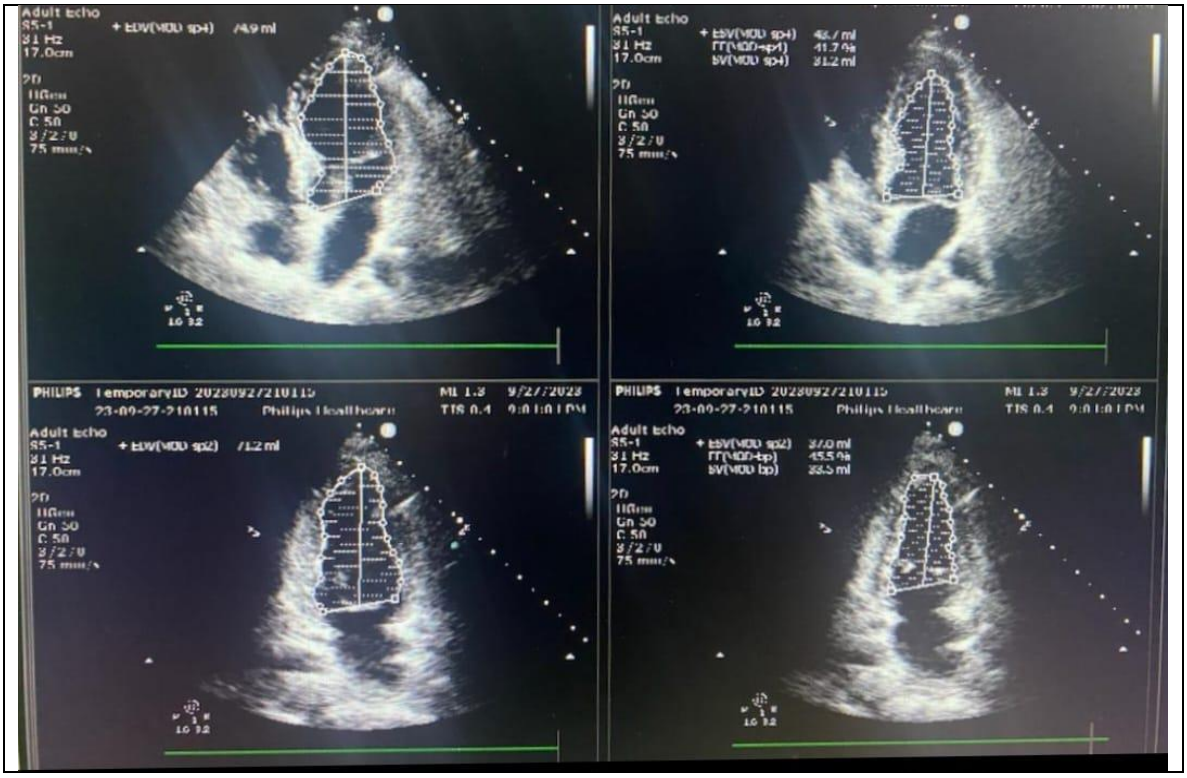

Figure (3): LV EF by Simpson method=40%, WMSI 1.5

Coronary angiography and primary PCI:

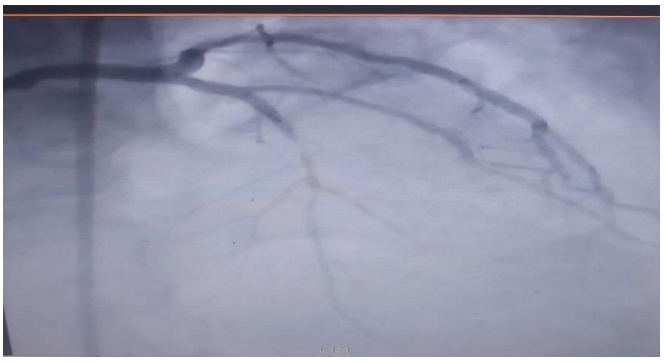

Figure **(4)** RAO cranial view shows mid total occlusion in LAD

Figure (5) RAO cranial view shows no reflow after PCI to mid LAD

Culprit: LAD, **Number of diseased vessels**: 1 (LAD),**Complication:** No Reflow, **TIMI Flow**:**0,Shock index**:0.9, **In hospital MACE**: Congestive heart failure and cardiogenic shock**.**

Discussion

Myocardial perfusion should be restored as soon as possible in patients with acute ST-elevation myocardial infarction (STEMI) [(6)]. Primary percutaneous coronary intervention (PPCI) to achieve a resumption of optimal blood flow is the preferred method of reperfusion, which significantly prevents further necrosis of the myocardium and improves the quality of life of patients with acute myocardial infarction (AMI) [(7)].

Patients with slow/ no coronary reflow in our study had significantly higher frequency of hypertension when compared to patients with normal coronary flow.

In accordance with our finding, other studies [(8,9)] showed the frequency of hypertension was found to be higher in the no-reflow group.

In the current study, patients with slow/ no coronary reflow had significantly increased heart rate when compared to patients with normal coronary flow.

In agreement with our study, another study [(8)] found that heart rates were higher in the no-reflow group, however no study explains the direct correlation between heart rate and no reflow. It was proposed that HF may bridge the association between heart rate and no reflow [(10)].

According to the present study, patients with slow/ no coronary reflow had significantly higher incidence of Killip classification III/IV when compared with patients with normal coronary flow.

In accordance with our study, a previous study [(8)] found that the Killip class were higher in the no-reflow group. Moreover, [(12)] results showed that subjects with Killip class ≥2 had 2.82 fold the risk for no reflow. Killip class ≥2 suggests that evidence of HF has been found [(11)].

Patients with slow/ no coronary reflow had significantly lower EF when compared to patients with normal coronary flow.

Supporting our finding, other researchers[(10)] stated that low LVEF was proven to be associated with no reflow. Moreover, Bayramoğlu et al. [(12)] reported that EF ≤40 was a predictor of no-reflow. So we suggest that low EF reflects poor prognosis of HF which might contribute to the development of no reflow.

In the current study, patients with slow/ no coronary reflow had significantly higher incidences of congestive heart failure and cardiogenic shock when compared to patients with normal coronary flow.

In agreement with our study, Wang et al., [(4)] found that the incidence rate of heart failure with Killip's grade ≥2 in the no-reflow group was significantly higher than that in the reflow group.

Patients with slow/ no coronary reflowing the present study had significantly higher incidences of ventricular arrhythmia when compared to patients with normal coronary flow.

In accordance with our finding, Ashraf et al. [(13)] study that included 3255 patients reported that no-reflow phenomenon was associated with significantly higher incidences of cerebrovascular accident (1.5% vs. 0%; $p < 0.001$), ventricular arrhythmia (2.5% vs. 0%; $p < 0.007$) and cardiogenic shock (3.8% vs. 1.2%; $p = 0.011$).

Patients with slow/ no coronary reflow in our study had significantly higher incidences of death when compared to patients with normal coronary flow (p= 0.028*).

Also, another study reported that no-reflow phenomenon was associated with significantly higher in-hospitality mortality (6.8% vs. 2.9%; $p = 0.01$) [13].

Covid-19 status in the present study was significant predictor for slow/ no reflow coronary flow .

In accordance with our finding**,** Güler et al. [14] study that included 126 patients reported that no-reflow phenomenon was numerically higher in COVID-19 patients who underwent PPCI due to STEMI compared to the non-COVID group.

According to our study, **Killip classification**, was significant predictor for slow/ no reflow coronary flow (AOR (95%CI) = 10.47; p=0.002*).

In agreement with our study, the predictors of no-reflow in Bayramoğlu et al.[12] study included Killip class ≥3. In addition, in a study by Wang et al., [4], high Killip class (≥3) was demonstrated to be associated with the development of no-reflow.

In line with our study, Celik et al. [15] found that high TIMI thrombus grade was significant predictor for slow/ no reflow in patients with STEMI after primary PCI.

The multivariate analysis in Ashraf et al. [13] study showed low pre-procedure TIMI flow grade [2.04, 1.3–3.21, $p = 0.002$] was an independent predictor of no-reflow. Furthermore, Fajar et al. [10] meta-analysis reported that initial TIMI flow ≤1 had the greater impact on no reflow (OR95%CI = 3.83, $p < 0.0001$).

Patients with slow/ no coronary reflow had significantly higher shock index when compared to patients with normal coronary flow. Moreover, incidence of shock index above 0.66 was significantly higher in patients with slow/ no coronary reflow when compared to patients with normal coronary flow (p <0.001*).

This comes in agreement with Mazhar et al.,[4] study, they found that patients with slow/no reflow had higher ratio of SI≥0.66, compared with the normal reflow.

ROC curve in our study revealed that cut off value 0.6220 of shock index was a significant predictor for slow/ no reflow coronary flow with AUC 0.968 with 96.4% sensitivity, 92.3% specificity, and 96.3% accuracy. In addition, we found that binary logistic regression analysis revealed that shock index was significant predictor for slow/ no reflow coronary flow (AOR (95%CI) = 84.5; p<0.001*).

Similarly, Mazhar et al.,[4] showed that SI is an independent predictor of slow/no-reflow after emergency coronary angiography in patients with acute myocardial infarction (AMI), and further demonstrated that slow/no reflow in patients with AMI following primary PCI was more likely associated with SI ≥ 0.66.

Death in the present study was associated with significantly higher shock index (p=0.001*).

Conclusion

Based on our findings, shock index was significant predictor for slow/no coronary reflow in patients with STEMI after primary PCI and was found to be associated with in hospital morbidity and mortality.

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