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Association of Shock Index on Admission with Coronary Slow or No Reflow in Patients with Acute Myocardial Infarction "STEMI" undergoing Primary PCI

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ORIGINAL ARTICLE

Association of Shock Index on Admission with Coronary Slow or No Reflow in Patients with Acute Myocardial Infarction "STEMI" undergoing Primary PCI

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Abstract

Objectives: ***An unfavorable cardiovascular prognosis following the first percutaneous coronary intervention (PCI) is considerably impacted by the shock index (SI). In patients with acute myocardial infarction (AMI) ST-elevation myocardial infarction (STEMI) receiving the primary percutaneous coronary intervention.***

Aim: ***This research sought to assess the relationship between SI at admission and coronary slow/no-reflow.***

Methods: ***In the first 24 hours following the start of symptoms, 200 patients who were receiving primary PCI participated in this prospective observational trial. Patients were divided into two groups: those with sluggish or no flow (n= 74) and those with normal flow (n= 126). Results: there was a statistically higher elevated troponin level among the slow flow/ no flow group compared to the regular flow group, with no significant difference regarding ECG and angiography.***

Results: ***Mean SI was statistically higher among the slow flow/ no flow group. MBG demonstrates a lower score among the slow flow/ no flow group. Elevated creatine kinase-myocardial band (CKMB) and clinically relevant bleeding (CRB) were statistically higher in the slow flow/ no flow group. SI demonstrates a higher mean among TIMI 0. High SI, elevated C-reactive protein (CRP), and random blood sugar (RBS) can predict slow flow/ no flow. SI was a significant predictor for slow flow / no flow at cut-off 0.67 with 81% sensitivity, 80.9% specificity, 70.7% PPV, 86.4% NPV, and 80% total accuracy.***

Conclusions: ***High SI, elevated CRP, and RBS can predict slow flow or no flow. SI was a significant predictor of slow flow or no flow at a cut-off value of 0.67 67, with 81% sensitivity and 80.9% specificity.***

Keywords: Shock Index; Coronary Slow or No Reflow; Acute Myocardial Infarction; Primary PCI

1. Introduction

The short- and long-term mortality of patients with acute myocardial infarction (AMI) was considerably reduced by primary percutaneous coronary intervention (PCI) with stenting insertion and combination with dual antiplatelet medications and statins therapy. According to earlier research, however, the likelihood of coronary slow/no-reflow was as high as 20% to 30%. As a consequence, patients continued to have noteworthy myocardial reperfusion harm even after the infarct-related artery (IRA) was successfully opened. Slow or no reflow is frequently indicative of a microvascular blockage in the distal coronary artery and is thought to be a risk factor for unfavorable cardiovascular events. [1]

Primary PCI for AMI: CVIT expert consensus paper for 2018. 33(2), 178–203. Cardiovascular intervention and therapy. After completing primary PCI, patients with AMI frequently experience coronary delay or no reflow, and shock index (SI) is a significant risk factor for a poor cardiovascular prognosis. This study aims to determine if coronary slow/no-reflow in individuals with AMI after initial PCI correlates with SI. [2]

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The majority of our research interests and treatment plans center on the epicardial coronary arteries because complete coronary artery blockage was discovered in the early hours after transmural myocardial infarction. On the other hand, the coronary microvasculature receives less attention. When a coronary artery is blocked, the cardiac capillaries and arterioles undergo deleterious alterations. No reflow is a condition where blood flow to the ischemic tissue is still restricted even after the occlusion has been relieved. [3]

Structural no reflow: Microvessels in the necrotic myocardium area show (a) damage and loss of capillary integrity with endothelial enlargement and edema and (b) microvascular blockage when subjected to prolonged ischemia. The majority of structural no-reflow is irreversible. The degree and duration of ischemia determine the lesion's extent .[4] Functional no reflow: Microvasculature's patency is impaired by spasm, microthrombotic embolization, reperfusion damage, neutrophil and platelet buildup, and neurohumoral system activation. Functional no reflow may have varied degrees of reversibility. [5]

The complex and unresolved pathogenesis of the slow/no-reflow phenomenon has been the subject of several hypotheses, including distal microembolization of thrombus fragments, swelling of endothelial cells brought on by ischemic and reperfusion injury, and microvascular spasm. Numerous studies have been conducted in clinical settings to examine the predictors of the slow/no-reflow phenomenon. The findings indicated that thrombosis burden, reperfusion time, inflammatory factors, the ratio of stent size to vessel diameter, and pre-hospital treatment with tirofiban were all possibly related to slow/no-reflow and clinical prognosis in patients with AMI after emergent PCI.

However, SI has only sometimes been recorded in slow/no-reflow phenomena.[2] despite being a significant risk for major adverse cardiovascular events (MACEs) in patients with AMI. The slow/no-reflow phenomenon is a severe catheterization laboratory consequence and a crucial predictor of clinical outcomes. [6]

This study assessed the association between SI on admission and coronary slow/no-reflow in patients with AMI ST-elevation myocardial infarction "STEMI" receiving primary PCI during the first 24 hours of symptoms onset.

2. Patients and methods

This prospective observational study involved 200 patients of both sexes who underwent primary PCI within the first 24 hours of the onset of their symptoms and who met the clinical criteria for ST-elevation myocardial infarction (STEMI) [typical ischemic chest pain lasting longer than 30 minutes or other symptoms suggestive of ischemia with ST-segment elevation >1mm in at least two contiguous leads and possibly new LB on ECG combined with increased cardiac specific biomarkers].

El Hussein University Hospital and El-Mahalla Cardiac Center's Ethical Committee gave their clearance before the study. All participants provided written, voluntarily informed consent.

Patients who met the following criteria were excluded from the study: "NSTEMI" patients, patients with unstable angina, patients with "STEMI" who presented more than 24 hours after the onset of symptoms, patients with a history of recent surgery or trauma within the previous month, patients with hematologic diseases, patients with malignant tumors, patients with severe renal disorders, patients with acute or chronic inflammatory diseases, patients with febrile disorders, and patients who refused to participate in the study.

The patients were divided into two groups: 126 had normal flow, and 74 had sluggish or no flow.

Complete histories, clinical examinations (heart rate, blood pressure, respiratory rate, and temperature), cardiac auscultation of the neck veins, clinical manifestations (heart failure, pulmonary hypertension, and systemic hypertension), and laboratory tests (such as CK-MB, troponin, hs-CRP, serum creatinine, blood urea nitrogen, and glucose) were all performed on all subjects.

Standard twelve lead ECG: Focusing on the QRS complex, ST-segment, and T-wave alterations, this was examined to identify ischemia criteria in patients with coronary artery disease. STEMI was defined as ST-segment elevation with ST-segment elevation of 1 mm in the leads other than v2-v3 and measured at the J-point in at least two contiguous leads. In leads V2-V3, in the absence of left bundle branch block (LBBB), 2.5 mm is found in males 40 years, 2 mm in men 40 years, or 1.5 mm in women. [7]

Resting Transthoracic Echocardiography (TTE): In the emergency room, every patient had a bedside transthoracic two-dimensional (2D), M mode, and Doppler echocardiogram. In either the supine or left lateral postures, the patients were evaluated. The following formula was used to compute the ejection fraction (EF). EF is equal to 100% (EDV - ESV)/EDV. ESV stands for end-systolic volume, whereas EDV stands for end-diastolic volume.[8] The left ventricular ejection fraction (LVEF) can be semi-quantified via transthoracic echocardiography and the left ventricular wall motion score index (WMSI) calculation. LVEF calculation using Simpson's biplane approach.

Primary percutaneous coronary angioplasty: All patients were subjected first to diagnostic

coronary angiography followed by PPCI.

Pre-procedural medications: All patients were given a loading dosage of 600 mg of clopidogrel and 300 mg of aspirin. Unfractionated heparin was administered intravenously at a dosage of 70–100 U/kg. The usual vascular morphological characteristics of thrombus-laden or hazy filling defects and impairment of distal flow were used to identify the infarct-related lesion.[9] Coronary angiography defined angiographic coronary stenosis as a luminal diameter decrease of 50%. A given coronary segment was considered to have a complete coronary blockage if there was no antegrade flow of contrast material through it. [8]

Instead of the infarction-related artery, multivessel disease was defined as one or more lesions with >50% stenosis in one or more main epicardial coronary arteries or its major branches.[8] Pre-dilatation and post-dilatation of the balloon were done as needed if there was a need for them. According to the TIMI blood flow grade, reperfusion success was determined. If the flow was TIMI 3, reperfusion was deemed adequate. TIMI flow grade 2 during the surgery without signs of dissection, persistent stenosis, distal embolism, or vasospasm is characterized as angiographic slow/no-reflow. The agreement between two intervention doctors was used to calculate the TIMI flow for grades.[9] Myocardial blush grade (MBG), an angiographic indicator of myocardial perfusion, was evaluated. According to the definition, SI was determined as HR divided by systolic blood pressure (SBP).

Statistical analysis

IBM Inc., Chicago, Illinois, USA, used SPSS v26 to conduct the statistical study. The unpaired Student's t-test was used to compare quantitative data between the two groups. Quantitative variables were provided as mean and standard deviation (SD). The Chi-square test or Fisher's exact test was used to analyse qualitative variables reported as frequency and percentage (%). Statistical significance was defined as a two-tailed P value of 0.05.

3. Results

The two studied groups were matched regarding age, sex distribution and risk factors, no difference that is statistically significant (p value > 0.05) Table 1

Table 1. Sociodemographic data and risk factors among the studied groups

SOCIODEMOGRAPHIC DATA		TOTAL (N=200)	SLOW FLOW / NO FLOW (N=74)	NORMAL FLOW (N=126)	P VALUE
AGE (YEARS)		59.03±5.07	59.64±4.58	58.67±5.32	0.187
SEX	Female	121(60. %)	43 (58.1%)	78 (61.9%)	0.596
	Male	79 (39.5%)	31 (41.9%)	48 (38.1%)	
RISK FACTORS	HTN	68 (34.0%)	21 (28.4%)	47 (37.3%)	0.198
	DM	41 (20.5%)	18 (24.3%)	23 (18.3%)	0.305
	Smoking	62 (31.0%)	25 (33.8%)	37 (29.4%)	0.530
	Family history	19 (9.5%)	5 (6.8%)	14 (11.1%)	0.311
	BMI	26.72±3.15	26.50±2.96	26.85±3.26	0.442

Data are displayed as the mean, standard deviation, or frequency (percent). Body mass index; HTN: hypertension; DM: diabetes mellitus.

The slow flow/no flow group had statistically reduced mean SBP, DBP, and pulse, but there was no statistically significant difference in laboratory tests between the examined groups. In contrast, troponin levels were greater in the slow flow/no flow group than in the normal flow group. The slow flow/no flow group had statistically greater levels of elevated CKMB and CRB. Additionally, mean RBS levels were greater in the group with sluggish flow or no flow.

Table 2. Clinical information and lab tests for the groups under study

			TOTAL (N=200)	SLOW FLOW / NO FLOW (N=74)	NORMAL FLOW (N=126)	P VALUE
CLINICAL DATA	SBP		130.81±10.73	126.34±9.07	133.44±10.80	≤0.001*
	DBP		75.65±9.97	71.67±8.29	77.99±10.16	
	Pulse		83.96±8.91	88.16±9.23	81.50±7.74	
LABORATORY INVESTIGATIONS	Hb		13.05±1.26	13.01±1.19	13.08±1.30	0.706
	S.cr		0.98±0.19	1.02±0.19	0.96±0.18	0.061
	AST		19.56±4.40	18.67±4.30	20.07±4.39	0.029
	ALT		21.86±7.51	20.51±7.69	22.65±7.32	0.051
	TC		195.09±27.11	197.53±25.46	193.66±28.03	0.331
	TGS		140.76±22.21	139.77±21.56	141.34±22.65	0.63
	LDL		117.80±9.04	118.81±8.43	117.21±9.36	0.229
	HDL		48.35±7.27	47.47±7.29	48.86±7.23	0.192
	Troponin	Normal	68 (34.0%)	13 (17.6%)	55 (43.7%)	≤0.001*
		Elevated	132 (66.0%)	61 (82.4%)	71 (56.3%)	
MBG	0		9 (4.5%)	9 (12.1%)	0 (0%)	≤0.001*
	1		44 (22.0%)	44 (59.5%)	0 (0%)	
	2		51 (25.5%)	21 (28.4%)	30 (23.8%)	
	3		96 (48.0%)	0 (0%)	96 (76.2%)	
CKMB	Normal		43 (21.5%)	3 (4.1%)	40 (31.7%)	≤0.001*
	Elevated		157 (78.5%)	71 (95.9%)	86 (68.3%)	
CRP	Normal		82 (41.0%)	7 (9.5%)	75 (59.5%)	
	Elevated		118 (59.0%)	67 (90.5%)	51 (40.5%)	
	RBS		222.10±104.9	288.78±108.7	182.93±80.27	

Data are displayed as the mean, standard deviation, or frequency (percent). Diastolic blood pressure is

sometimes referred to as DBP. Haemoglobin, or Aspartate aminotransferase (AST), alanine transaminase (ALT), serum creatinine (S.cr), total cholesterol (TC), Triglycerides, low-density lipoprotein, high-density lipoprotein, and random blood sugar are all abbreviations for the same thing. The statistical significance level is P0.05.

No statistically significant difference was observed among slow flow / no flow and normal flow groups regarding ECG and angiography results. MBG demonstrates lower score among slow flow / no flow group. Mean SI was statistically higher among slow flow / no flow group, p value equal or less than 0.05.

Table 3. lists the examined groups' ECG, angiography, EF%, and shock index.

		TOTAL (N=200)	SLOW FLOW / NO FLOW (N=74)	NORMAL FLOW (N=126)	P VALUE
ECG	Anterior MI	52 (26.0%)	21 (28.4%)	31 (24.6%)	0.72
	Inferior MI	53 (26.5%)	16 (21.6%)	37 (29.4%)	
	Anterolateral	40 (20.0%)	14 (18.9%)	26 (20.6%)	
	MI	28 (14.0%)	11(14.9%)	17 (13.5%)	
	Inferolateral MI	27 (13.5%)	12 (16.2%)	15 (11.9%)	
	Antroseptal MI				
ANGIOGRAPHY	LAD	118	46 (62.2%)	72 (57.1%)	0.554
	RCA	(59.0%)	16 (21.6%)	36 (28.6%)	
	LCX	52 (26.0%)	12 (16.2%)	18 (14.3%)	
		30 (15.0%)			
EF%		48.10±9.38	44.37±9.29	50.28±8.75	≤0.001*
SHOCK INDEX	Mean ± SD	0.646±0.08	0.703±0.08	0.613±0.06	≤0.001*
	≥0.67	82 (41.0%)	58 (78.4%)	24 (19.0%)	
	<0.67	118	16 (21.6%)	102	
		(59.0%)		(81.0%)	

The presentation of data is in frequency (%) or meanSD. RCA stands for the right coronary artery, LCX for the left circumflex artery, and EF stands for ejection fraction. The statistical significance level is *P<0.05.

TIMI 0 was presented in 2% of patients, TIMI 1 was presented in 16.5%, TIMI 2 was presented in 18.5% and TIMI 3 was presented in 63%. There was statistically significant difference between (TIMI 0, TIMI 1, TIMI 2), and TIMI 3 have p-values ≤ 0.001.

Table 4. shows how TIMI and shock index are related

TIMI	THE STUDIED GROUP (N=200) (N%)	MEAN SHOCK INDEX	P VALUE
TIMI 0	4 (2.0%)	0.72±0.002 a	P≤0.001*
TIMI 1	33 (16.5%)	0.73±0.04 b	
TIMI 2	37 (18.5%)	0.67±0.08 c	
TIMI 3	126 (63.0%)	0.61±0.06 abc	

Data are presented as frequency (percentage) or mean±SD. abc: are letters of significance. * P<0.05 is statistically significant.

The following variables were independent predictors of sluggish flow or no flow following multivariate logistic regression analysis and controlling for confounding variables., SI ≥0.67 (OR=9.41), Elevated CRP (OR=8.36) and RBS >222 (OR=5.81). So, high shock index, elevated CRP and RBS can predict slow flow / no flow group.

Table 5. Analysis of multivariate logistic regression for independent determinants of sluggish flow or no flow

INDEPENDENT PREDICTORS	P VALUE	OR	95% CI
SHOCK INDEX ≥0.67	≤0.001	9.41	3.9-22.54
ELEVATED CRP	≤0.001	8.36	2.9-23.7
RBS >222	≤0.001	5.81	2.4-13.9

The best cut-off value considering SI in prediction of low flow / no flow was 0.67with 81% sensitivity, 80.9% specificity, 70.7% PPV, 86.4% NPV and 80 % total accuracy as observed in. first Figure 1

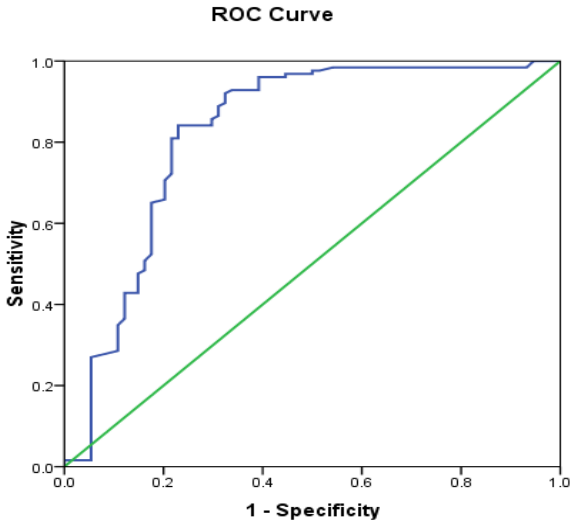

Figure 1. ROC curve for shock index-based slow flow prediction

Case (1):

Male patient, 58 years old, not diabetic, not hypertensive, not smoker, and not known to be ischemic heart disease before. He was presented with retrosternal compressing chest pain 9 hours duration. Blood pressure was 136/75 mmHg, heart rate 82 bpm. Shock index: 0.60. Laboratory findings: Hb: 11, S. creatinine: 1.2 mg/dl, Tpoponin : +ve , CK-MB: +ve , CRP: +ve and RBS:110.

ECG: 12-lead ECG: sinus rhythm with ST segment elevation in lead v1-v6 with pathological Q wave in v1, v2. Figure 1

Echocardiography: LVEF 49%, RSWMA inform of hypokinetic of basal, mid and apical septal and no mechanical complications. Figure 1(B)

Medication: 300 mg aspocid P.O, 600 mg clopidogrel.

Coronary angiography presented in figure 1 (C, D).

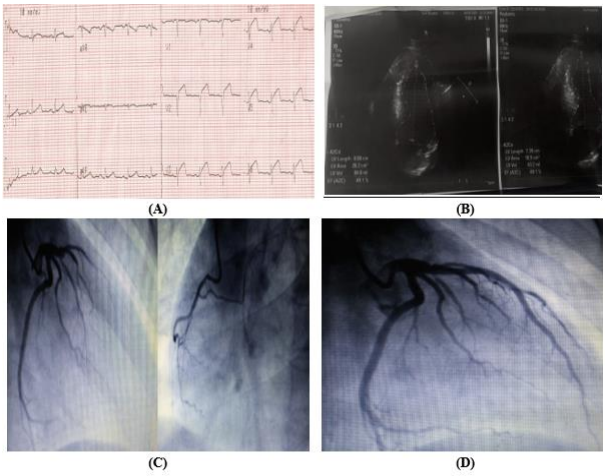

Figure 2. (A) ECG, (B) Echocardiographic images, (C) Coronary angiography revealed proximal LAD total occlusion, with normal dominant LCX and small non dominant and (D) Primary PCI to LAD with two DES with TIMI flow III , MBG: II

Case (2):

Male patient, 56 years old, hypertensive, not known diabetic, heavy smoker, dyslipidaemia and not known to be ischemic heart disease before. He was presented with retrosternal compressing chest pain 9 hours duration. Blood pressure was 133/89 mmHg, heart rate 95 bpm. Shock index :0.71. Laboratory findings: Hb:11.3, S. creat:0.9, Troponin; +ve , CK-MB: +ve , CRP : +ve and RBS:320.

ECG: 12-lead ECG: sinus rhythm with ST elevation in lead v1-v6 with pathological Q wave in v1 –v3. Figure 2(A)

Echocardiography: LVEF 32 %, RSWMA inform of hypokinesia of basal, mid and apical septal wall and anterior wall. Figure 2(B)

Medication: Aspirin 300 mg PO, in form of chewable tablets and a loading dose of 600 mg clopidogrel PO were given.

Coronary angiography presented in Figure 2 (C, D).

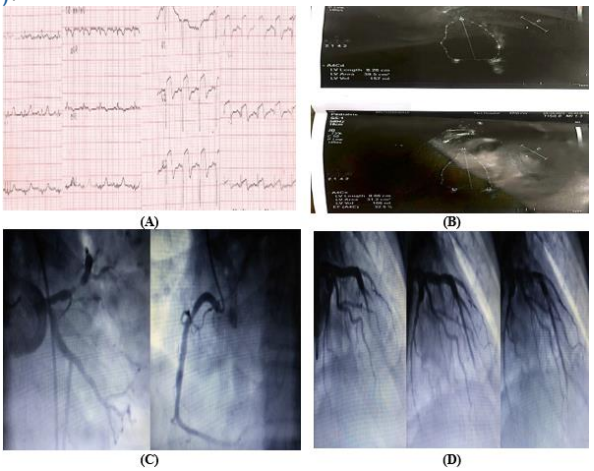

Figure 3. (A) ECG, (B) Echocardiographic images, (C) Coronary angiography revealed proximal subtotal LAD occlusion, LCX: normal

non dominant artery with normal dominant RCAand (D) Primary PCI to LAD with two DES with TIMI flow II , MBG: I

4. Discussion

Because of the complex clinical circumstances surrounding STEMI, early risk assessment, immediate therapeutic treatment, and swift diagnosis are required. As a kind of reperfusion therapy, primary percutaneous coronary intervention (PCI) is used to treat the majority of STEMI patients. Despite the low prevalence of major adverse cardiovascular events (MACE) with contemporary primary PCI, patients with STEMI nevertheless have a dismal prognosis. [10]

The current study's results showed no discernible difference in either group's age or sexual orientation (P = 0.596).

In line with our findings, Wang et al. [11] conducted a retrospective research involving 374 individuals who received emergency angiography regularly. There were two groups of patients. The participants were split into two groups: those who experienced no hospital issues (n=197) and those who did (n=60).

Regarding risk variables (HTN, DM, smoking, and family history), the study demonstrated no statistically significant differences between the analyzed groups.

In line with our findings, Abe et al. [12] We conducted a retrospective analysis involving about 680 individuals who had PCI for revascularization treatment. To examine the prognostic consequences of admission SI on long-term prognoses in patients with AMI who were discharged after PCI, participants were split into two groups: group A, consisting of approximately 504 patients with SI0.006, and group B, consisting of patients with SI0.006.

The findings revealed no statistically significant differences between the study groups in terms of the risk variables (HTN, DM, smoking, and family history) (p >0.005).

Parallel to our results, Hwang et al. [13] analyzed cardiac magnetic resonance imaging (CMR) STEMI and treated it with the first PCI. To examine the relationship between SI and myocardial damage in STEMI patients having primary PCI (PCI), the 306 research participants were split into SI>0.7 (n = 88) and SI0.7 (n = 218) groups. The findings demonstrated that there was no statistically significant difference in terms of risk variables (HTN and DM) between the two groups (p > 0.005).

However, Wang et al.[11] highlighted that Risk variables (HTN, DM, smoking, and family history) were substantially higher in the group with in-hospital problems than in the group with no in-hospital difficulties. This contradiction between both studies can be justified by the difference between the criteria of both study groups (p < 0.005).

In the present study, mean SBP, DBP, and pulse were statistically lower in the slow flow / no flow group.

Parallel to our results, Wang et al. [14] highlighted SBP, DBP, and heart rate were statistically lower in the slow flow / no flow group (p < 0.005). Also, Abe et al. [12] highlighted that SBP, DBP, and heart rate were statistically lower in patients with SI ≥ 0.006 (p < 0.005). Moreover, Hwang et al.,13 highlighted that SBP, DBP, and heart rate were statistically lower in the SI≤ 0.7 (p less than 0.005) group.

The current study discovered no statistically significant difference between the tested groups regarding mean HB, liver enzymes, and lipid profile. At the same time, the slow-flow group had a statistically higher elevated troponin level than the no-flow group.

According to our findings, Wang et al. [14] noted that there was no statistically significant difference in HB levels between the two groups but that the slow-flow group had statistically greater raised troponin levels than the no-flow group. According to our findings, Abe et al.,[12] In terms of HB level, it was noted that there was no statistically significant difference between the two groups (P > 0.001). According to Hwang et al.[15], the mean HB in the SI 0.7 group was not significantly different from the other investigated groups (P > 0.005).

The current investigation discovered that most patients had affected LAD, representing 62.2% as compared to 57.1%, while RCA was affected in 21.6% versus 28.6% and LCX in 16.2% versus 14.3% among slow flow / no flow and normal flow groups, respectively.

By our results, Abe et al. [12] highlighted that most patients had affected LAD, representing 44.8% as compared to 49.4%, while RCA affected 39.1% versus 36.9% and LCX affected 13.5% versus 12.5% between patients with a SI < 0.006 and those with a SI ≥0.006.

The current investigation discovered that the slow flow/no flow group's mean SI was significantly greater (p ≤0.05).

In line with our findings, Wang et al. [11] emphasized the statistical significance of SI high among in-hospital complications (P <0.001). Also, it highlighted that mean SI was statistically higher among the slow flow / no flow group (p ≤ 0.05).

Also, Abe et al.[12] highlighted that SI was statistically higher among patients with SI ≥ 0.006 (p ≤ 0.05). Moreover, Hwang et al.[13] highlighted that the mean SI was statistically higher among the SI≤ 0.7 group. Additionally, Zhang et al.15 examined three major outcomes for this analysis: in-hospital mortality, short-term negative outcomes, and long-term negative outcomes using a systematic review and meta-analysis. The

findings indicated that high SI may have a significant predictive value for the outcomes of AMI patients and may increase in-hospital mortality as well as short- and long-term bad outcomes.

The current study discovered that increased CKMB and CRB was statistically higher in the slow flow / no flow group, accounting for 95.9% and 90.5%, respectively, versus 68.3% and 40.5% among the normal flow group.

Parallel to our results, Wang et al. [14] highlighted that CRB was statistically higher in slow flow / in comparison to the typical flow group, no flow group (P = 0.045).

Furthermore, Hwang et al. [13] highlighted that CKMB was statistically higher in slow flow / compared to the typical, no-flow group. It was discovered that SI was relevant in the current investigation. Good predictor for slow flow / no flow. The area under the curve was 0.828, with a 95% CI from 0.76 to 0.89. The best cut-off value considering SI in predicting low flow / no flow was 0.67 with 81% sensitivity, 80.9% specificity, 70.7% PPV, 86.4% NPV, and 80 % total accuracy.

In line with our findings, Wang et al.[14] It was noted that ROC curve research showed that SI=0.66 had an area under the curve of 0.672 and a sensitivity of 76% and specificity of 59% for predicting slow/no-reflow phenomena. According to our study, Hwang et al. [13] emphasized that the optimal cut-off of SI for predicting big MI was 0.7 on the receiver operating characteristic curve (ROC, sensitivity 61%, specificity 74%, area under the curve [AUC] 0.73), which was defined as ≥18.7% of the median infarct size determined by CMR., P <0.01).

Limitations include the fact that the study only involved one center, so results may vary elsewhere, a limited sample size, which may have made some group comparisons ineffective at identifying significant differences for particular variables, and a lack of a follow-up period.

5. *Conclusion*

High SI, elevated CRP, and RBS can predict slow flow / no flow. SI was a significantly good predictor for slow flow / no flow at a cut-off value of 0.67 67with 80.9% specific and 81.1% sensitive.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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