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ORIGINAL ARTICLE

Predictive value of the Novel CHA_2DS_2-VASc-HSF-TT Score for No-reflow in Patients Undergoing Primary and Post-thrombolysis Percutaneous Coronary Intervention

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Running Title: Novel Score for No-Reflow in STEMI patients undergoing PCI

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ABSTRACT

Background: During primary PCI, coronary no-reflow occurs when microvascular obstruction persists despite open epicardial circulation. The CHA_2DS_2-VASc-HSF score, used to assess thromboembolic risk in atrial fibrillation, correlates also with no-reflow risk. This study aims to evaluate the novel CHA_2DS_2-VASc-HSF-TT score's predictive value for no-reflow in STEMI patients undergoing primary PCI.

Methods: This study at Zagazig University Hospitals included 255 STEMI patients underwent PCI for STEMI from June 2024 till March 2025. The CHA_2DS_2-VASc-HSF-TT score was calculated, incorporating factors like thrombolysis usage and PCI delay and was correlated to no-reflow. In-hospital outcomes like mortality, heart failure and arrhythmias were assessed.

Results: 22.35% of patients experienced no-reflow. Statistically significant associations with no-reflow included heart failure ($P = 0.0159$), diabetes ($P = 0.0007$), hypertension ($P < 0.0001$) and delayed PCI (>6 hours) ($P < 0.0001$). The CHA_2DS_2-VASc-HSF-TT score had an AUC of 0.846, with 82.5% sensitivity, 79.3% specificity, and 80% accuracy. PCI delay >6 hours was a strong predictor for no-reflow (AUC 0.955).

Conclusion: The CHA2DS2-VASc-HSF-TT score is a strong predictor of no-reflow in STEMI patients (cutoff ≥5). Delayed PCI (>6 hours) is an important independent predictor, emphasizing the need for timely intervention or bridging thrombolysis to improve outcomes.

Keywords: CHA_2DS_2-VASc-HSF-TT score; No-reflow; STEMI; PCI.

Abbreviations:

CAD	coronary artery disease
DM	Diabetes Mellitus
HTN	hypertension
PCI	percutaneous coronary intervention
STEMI	ST-segment elevation myocardial infarction

INTRODUCTION

Acute myocardial infarction with ST-segment elevation (STEMI) is primarily caused by the rupture or erosion of an atherosclerotic plaque, leading to the occlusion of a coronary artery. The most effective treatment for this condition is primary percutaneous coronary intervention (PCI), which helps open the infarct-related occluded coronary artery promptly [1].

Coronary no-reflow refers to myocardial hypoperfusion caused by microvascular obstruction despite a patent epicardial circulation. This phenomenon, observed in the primary PCI era for STEMI, is a significant poor prognostic marker for outcomes such as left ventricular remodeling, infarct size, left ventricular ejection fraction, and long-term mortality [2].

After approximately 6 hours of acute myocardial infarction, myocardial necrosis occurs, leading to capillary bed edema, myocardial cell swelling, neutrophil plugging, and alterations in capillary integrity, which contribute to the no-reflow phenomenon. Preventing this condition is crucial for improving the long-term prognosis of such patients [3].

The factors within the CHA_2DS_2-VASc score are linked to atherosclerosis, vascular spasm, and microvascular dysfunction, all of which are associated with the risk of the no-reflow phenomenon [4]. A new scoring system, the CHA_2DS_2-VASc-HSF score, builds upon the CHA2DS2-VASc framework by incorporating additional risk factors: smoking status (S), hyperlipidemia (H), and a family history of coronary artery disease (F). This refined scoring system offers a more comprehensive risk assessment, while also revising the gender component by replacing female sex with male sex [5]. But, we believe that the current scores don't include PCI related aspects that may affect the potency of myocardial revascularization underpowering their predictive value. Thus, by adding PCI time delay and thrombolytic use, this study aimed to demonstrate the value of CHA_2DS_2-VASc-HSF-TT scores in predicting no reflow in patients with ST segment elevation myocardial infarction submitting for percutaneous coronary intervention.

PATIENTS AND METHODS

Study population:

In a cross-sectional fashion, 255 STEMI patients underwent primary, pharmaco-invasive or rescue PCI within 48 hours of admission at cardiology department, Zagazig University Hospitals from June 2024 till March 2025 were enrolled in the current study. The study received approval from the Zagazig University Institutional Review Board (IRB) (ZU-IRB #350/19-05-2024), and informed consent was obtained from each participant. Patients with non-STEMI, atrial fibrillation, severe liver or renal disease, malignancy, sepsis, hematological disorders, or any infectious or inflammatory conditions were excluded.

Data collection:

All the studied patients underwent history taking including risk factors as hypertension, diabetes mellitus, smoking, dyslipidemia and family history of premature coronary artery disease (CAD). All patients underwent physical examination with emphasis on signs of heart failure. Patients were categorized using the Killip classification: class I (no heart failure signs) to class IV (cardiogenic shock) [6].

Cardiac evaluation included auscultation and palpation for signs of cardiomegaly or additional heart sounds. Electrocardiography was performed using a 12-lead ECG to identify STEMI. Laboratory investigations at admission included lipid profile, renal and liver function tests, complete blood count, blood glucose level and cardiac enzymes (CK-MB and troponin) measured initially and after 6 hours.

Scoring systems:

The original CHA_2DS_2-VASc score included: C was labeled 1 for congestive heart failure, H: 1 for hypertension, A_2: 2 for age ≥75y. old, D: 1 for DM, S_2: 2 for previous stroke or TIA, V: 1 for previous vascular event like MI, A: 1 for age 65-74y. old, Sc: 1 for female sex category. Each patient's CHA_2DS_2-VASc-HSF-TT score was calculated retrospectively after data collection, which expands upon the CHA_2DS_2-VASc score by including hyperlipidemia, smoking status, family history of CAD and sex risk factor was modified to male sex [4]. We modified the score by adding time delay to PCI was scored 1 if >6 hours, and thrombolytic therapy scored 1 if not administered. This score was evaluated for its predictive value in detecting no-reflow phenomena in STEMI patients. Transthoracic echocardiography was performed using the Vivid 7 GE Medical System with 2D, Doppler, and Tissue Doppler imaging in parasternal and apical views. It assessed valve morphology, left and right ventricular function, wall motion, and any complications of MI or rule-out other cardiac conditions like pericarditis or aortic stenosis. Coronary angiography and primary PCI were done within 48 hours of symptom onset, regardless of prior thrombolytic use, via radial or femoral access. TIMI flow grades were used to assess coronary perfusion, ranging from grade 0 (no perfusion) to grade 3 (complete perfusion) [7]**.** No-reflow was defined as a reduction in flow to TIMI grade 0 or 1 after stent deployment, not caused by vessel closure, spasm, or stenosis.

Statistical Analysis:

A version of SPSS 24.0 was used for the data analysis. For variables that followed a normal distribution, quantitative data was presented as mean ± standard deviation. For variables that did not follow a normal distribution, median and range were used. Frequencies and percentages were used to display the categorical data. For continuous variables, we used the Student-t-test and the Mann-Whitney U test to look for distribution-based differences; for categorical data, we used the Chi-square test. We used the Shapiro-Wilk test to see if things were normal. To maximize the balance between specificity and sensitivity, we constructed receiver operating characteristic (ROC) curves to assess diagnostic accuracy and determine the optimal cutoff values. In order to measure this, the AUC was calculated. Statistical significance was determined by a p-value that was less than 0.05 [8].

RESULTS

Fortunately, there was no missing data among our subjects. As described in **Table 1**, a comparison between cases with and without no-reflow showed no statistically significant difference in age (p = 0.8) or sex distribution (p = 0.8). Heart failure, DM, HTN and vascular events were more prevalent among cases with no reflow (P = 0.0159, 0.0007, <0.0001 and 0.0066 respectively). No significant differences were observed for stroke (p = 0.7801), family history of CAD (p = 0.9468), smoking (p = 0.7824), or dyslipidemia (p = 0.1475).

Cases with no-reflow had a statistically significant higher CHA_2DS_2-VASC-HSF-TT Score (median: 6 vs. 2, p < 0.0001) and a longer time from chest pain to ER presentation (170.44 ± 51.81 vs. 82.48 ± 58.27 minutes, p < 0.0001). Thrombolysis was absent in all no-reflow cases, while 19.7% of cases without no-reflow received thrombolysis (p = 0.0002). TIMI flow at presentation was statistically significantly lower in the no-reflow group (median: 1 vs. 3, p < 0.0001). Killip class distribution showed a significantly higher proportion of Killip II (p = 0.0009), Killip III (p = 0.0169), and Killip IV (36.84% vs. 11.62%, p < 0.0001) in the no-reflow group, while Killip I was more frequent in cases without no-reflow (p = 0.0546). Further details are listed in **Table 2**.

Cases with no-reflow had a significantly longer time delay to PCI (p < 0.0001). A significantly lower proportion of no-reflow cases underwent PCI within 3 hours (50.88% vs. 90.91%, p < 0.0001), while a significantly

higher proportion underwent PCI within 3–6 hours (42.11% vs. 8.08%, p < 0.0001) and beyond 6 hours (7.02% vs. 1.01%, p = 0.0082). Cases with no-reflow had a significantly lower incidence of diagonal artery involvement (8.77% vs. 28.28%, p = 0.0022) and a significantly higher incidence of LAD and LCX involvement (19.3% vs. 9.09%, p = 0.04, 0.0325 respectively). No significant differences were observed in OM (p = 0.4277), or RCA (p = 0.2076) involvement.

The mean target vessel diameter was significantly larger in no-reflow cases (p < 0.0001). Door-to-balloon time was significantly prolonged in the no-reflow group (p < 0.0001). The number of stents used was significantly higher in no-reflow cases (p < 0.0001), and post-dilatation was more frequently performed (p = 0.0318). Pre-dilatation rates showed no significant difference (p = 0.1115). Procedural details are demonstrated in **Table 3**.

Postoperative in-hospital follow-up showed that the no-reflow group were more complicated with arrhythmia (p = 0.0201), poor EF (p < 0.0001), acute heart failure (p < 0.0001), and mortality (p < 0.0001), see **Table 4.**

There was significant positive association between the incidence of no reflow and heart failure (P = 0.01), diabetes mellitus (P < 0.0001), hypertension (P =0.011), Time from chest pain to ER admission (P = 0.001), LCX vessel affection (P = 0.036), LAD vessel affection (P = 0.011), door to balloon time (P = 0.014) and number of stents (P < 0.0001). While there was negative association between the incidence of no reflow and diagonal vessel affection (P = 0.002), thrombolysis (P < 0.0001), TIMI flow at presentation (P < 0.0001) and ejection fraction (P < 0.0001), as shown in **Table 5**.

ROC curve analysis assessing the association between the CHA_2DS_2-VASc-HSF-TT score and no-reflow revealed an area under the curve (AUC) of 0.846. A cutoff value of **≥ 5** demonstrated a sensitivity of 82.5% and a specificity of 79.3%, with a positive predictive value (PPV) of 53.41% and a negative predictive value (NPV) of 94.01%. The overall accuracy was 80%, with a kappa value of 0.5173. The association was statistically significant (P < 0.001), **Figure 1**.

After yielding the hoc analysis we found that time delay from chest pain to PCI > 6 hours was an independent predictor for no reflow with AUC of 0.955, 90% sensitivity, 90.44% specificity, 75% PPV, 98.07% NPV, and 93.73% accuracy, (P < 0.001), **Figure 2**.

Table 1: Comparison between cases represented with and without no-reflow regarding basal demographic and clinical data

	With No-reflow (N = 57)	Without No-reflow (N = 198)	P. Value
Age (Years)	55.88 ± 21.2	56.45 ± 20.41	0.8052
Sex			
•Female	22 (38.6%)	79 (39.9%)	0.8594
•Male	35 (61.4%)	119 (60.1%)	
Comorbidities			
•Heart Failure	33 (57.89%)	79 (39.9%)	0.0159*
•Diabetes Mellitus	36 (63.16%)	75 (37.88%)	0.0007*
•Hypertension	44 (77.19%)	91 (45.96%)	<0.0001*
•Stroke	32 (56.14%)	107 (54.04%)	0.7801
•Vascular Event	26 (45.61%)	53 (26.77%)	0.0066*
•Family History of CAD	13 (22.81%)	46 (23.23%)	0.9468

	With No-reflow (N = 57)	Without No-reflow (N = 198)	P. Value
•Smoking	21 (36.84%)	69 (34.85%)	0.7824
•Dyslipidemia	35 (61.4%)	100 (50.51%)	0.1475

Data presented as mean ± standard deviation or number and percentage. CAD, coronary artery disease; N, number.

Table 2: Comparison between cases represented with and without no-reflow regarding cardiological evaluations

	With No-reflow (N = 57)	Without No-reflow (N = 198)	P. Value
CHA_2DS_2-VASc- HSF- TT Score	6 (1 - 9)	2 (0 - 9)	<0.0001*
Time from chest pain to ER (minutes)	170.44 ± 51.81	82.48 ± 58.27	<0.0001*
Thrombolysis	0 (0%)	39 (19.7%)	0.0002*
TIMI Flow at Presentation	1 (0 - 2)	3 (1 - 3)	<0.0001*
Killip Class			
I	14 (24.56%)	76 (38.38%)	0.0546
II	9 (15.79%)	78 (39.39%)	0.0009*
III	13 (22.81%)	21 (10.61%)	0.0169*
IV	21 (36.84%)	23 (11.62%)	<0.0001*

Data presented as mean ± standard deviation or number and percentage. N, number; TIMI, Thrombolysis in Myocardial Infarction.

Table 3: Comparison between cases represented with and without no-reflow regarding PCI related data

	With No-reflow (N = 57)	Without No-reflow (N = 198)	P. Value
Time from chest pain to PCI:	274.79 ± 57.78	172.93 ± 63.01	<0.0001*
< 3 hours	29 (50.88%)	180 (90.91%)	<0.0001*
3-6 hours	24 (42.11%)	16 (8.08%)	<0.0001*
> 6 hours	4 (7.02%)	2 (1.01%)	0.0082*
Target Vessel:			
Diagonal	5 (8.77%)	34 (17.17%)	0.0022*
LAD	14 (24.56%)	54 (27.27%)	0.04*
LCX	11 (19.3%)	18 (9.09%)	0.0325*
OM	6 (10.53%)	29 (14.65%)	0.4277
RCA	11 (19.3%)	28 (14.14%)	0.2076
Target vessel Diameter (mm):	3.94 ± 0.75	3.16 ± 1.2	<0.0001*
Door to Balloon Time: (minutes)	104.35 ± 10.96	90.45 ± 12.95	<0.0001*
Predilatation:	12 (21.05%)	25 (12.63%)	0.1115
Number of Stents:	2.14 ± 0.35	0.62 ± 0.76	<0.0001*
Post Dilatation:	8 (14.04%)	11 (5.56%)	0.0318*

Data presented as mean ± standard deviation or number and percentage. N, number; PCI, percutaneous coronary intervention.

Table 4: Comparison between cases represented with and without no reflow regarding post- operative follow up data

	With No-reflow (N = 57)	**Without No-reflow (N = 198)**	**P. Value**
Arrhythmia	14 (24.56%)	24 (12.12%)	0.0201* [X]
Ejection fraction (%)	44.23 ± 7.28	49.11 ± 5.3	<0.0001* [MWU]
Acute HF	19 (33.33%)	17 (8.59%)	<0.0001* [X]
Death	15 (26.32%)	8 (4.04%)	<0.0001* [X]

Data presented as mean ± standard deviation or number and percentage. HF, heart failure; N, number.

Table 5: Multivariable regression analysis between no-reflow status and variable parameters

	Unstandardized Coefficients		**OR**	**Test value**	**P. Value**	**95.0% Confidence Interval for B**	
	B	**Std. Error**				**Lower Bound**	**Upper Bound**
(Constant)	1.648	0.403				0.854	2.442
Age	-0.001	0.001	0.999	-0.981	0.327	-0.002	0.001
Male sex	-0.007	0.024	0.993	-0.291	0.771	-0.053	0.04
Heart Failure	0.132	0.051	1.1411	2.585	0.01*	0.031	0.232
Diabetes Mellitus	0.182	0.051	1.1996	3.567	<0.0001*	0.082	0.283
Hypertension	0.063	0.024	1.065	2.574	0.011*	0.015	0.111
Stroke	-0.005	0.023	0.995	-0.211	0.833	-0.051	0.041
Vascular Event	0.014	0.027	1.0141	0.51	0.61	-0.039	0.066
Family History of CAD	0.01	0.028	1.0101	0.359	0.72	-0.045	0.065
Smoking	0.001	0.024	1.001	0.061	0.951	-0.047	0.05
Dyslipidemia	0.012	0.025	1.0121	0.499	0.618	-0.036	0.061
CHA_2DS_2-VASc-HSF-TT Score	-0.009	0.011	0.991	-0.762	0.447	-0.031	0.014
Time Chest Pain to ER	0.002	0	1.002	3.482	0.001*	0.001	0.002
Thrombolysis	-0.322	0.046	0.7247	-7.01	<0.0001*	-0.412	-0.231
TIMI Flow at Presentation	-0.096	0.016	0.9085	-6.023	<0.0001*	-0.127	-0.064
Killip Class	0.01	0.013	1.0101	0.768	0.443	-0.015	0.035
Target vessel							
• Diagonal	-0.239	0.078	1.27	-3.069	0.002*	-0.085	0.392
• LAD	-0.034	0.042	1.0346	-0.81	0.011*	0.036	0.116
• LCX	0.111	0.053	1.1174	2.104	0.036*	0.7	0.214
• OM	-0.171	0.067	1.1865	-2.564	0.011*	0.04	0.303
Vessel diameter	-0.046	0.042	0.955	-1.085	0.279	-0.13	0.038
Door to Balloon Time	0.003	0.001	1.003	2.476	0.014*	0.001	0.005
Pre-dilatation	0.035	0.034	1.0356	1.028	0.305	-0.032	0.103
Number of Stents	0.093	0.017	1.0975	5.466	<0.0001*	0.059	0.126
Post Dilatation	0.041	0.049	1.0419	0.834	0.405	-0.056	0.138
Arrhythmia	0.059	0.034	1.0608	1.719	0.087	-0.009	0.126
Ejection fraction	-0.04	0.006	0.9608	-6.583	<0.0001*	-0.051	-0.028
Acute HF	0.059	0.037	1.0608	1.608	0.109	-0.013	0.132
Death	0.06	0.044	1.0618	1.341	0.181	-0.028	0.147

CAD, coronary artery disease; HF, heart failure; TIMI, Thrombolysis in Myocardial Infarction.

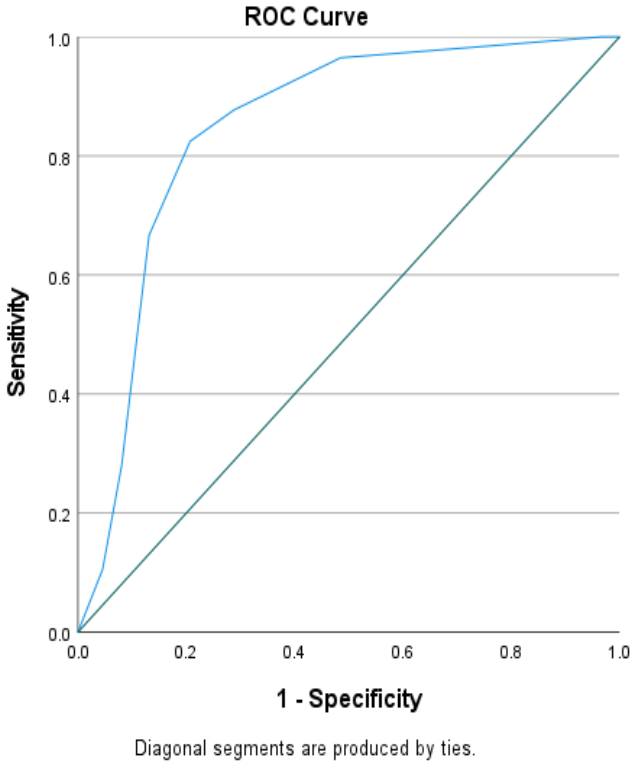

Figure 1: ROC curve analysis of association between CHA_2DS_2-VASc-HSF-TT Score and no reflow

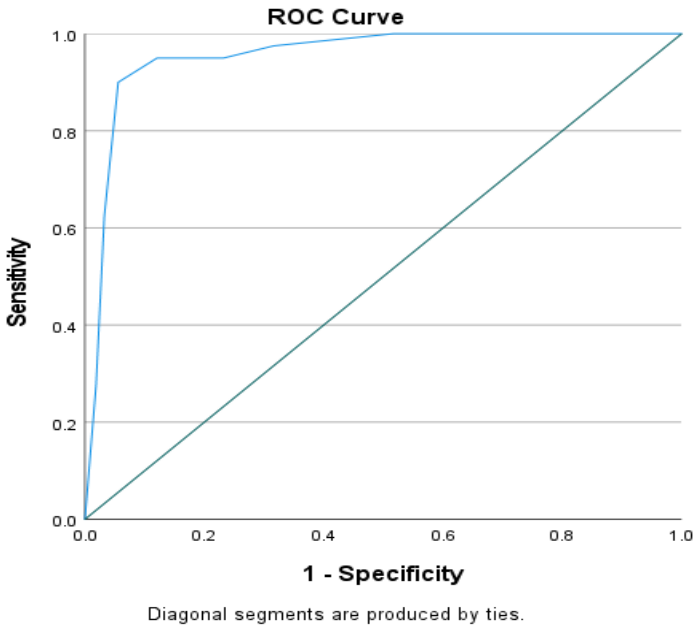

Figure 2: ROC curve analysis of association between no reflow and time from chest pain to PCI.

DISCUSSION

As we learnt from SWEDEHEART trial, 30-day all-cause and in-hospital cardiovascular mortality was higher among STEMI patients, those without standard modifiable risk factors. Thus, Identifying and early managing the modifiable risk factors for STEMI is an essential concern [9]. Our study findings showed significantly higher rates of heart failure, diabetes, hypertension, and prior vascular events in no-reflow cases compared to non-no-reflow, while other variables showed no significant differences. These findings align with Mansour et al [10], who reported a significant association between diabetes and no-reflow ($p = 0.023$). Similarly, Dönmez et al [11] found a higher prevalence of hypertension ($p = 0.001$), congestive heart failure (20.6% vs. 10.9%; $p = 0.006$), and prior stroke ($p < 0.0001$) in no-reflow patients.

Zhao et al [12] further confirmed that diabetes contributes to microvascular dysfunction and atherosclerosis, predisposing patients to no-reflow post-PCI, explained by microvascular dysfunction. Heart failure elevates left ventricular end-diastolic pressure, impairing perfusion; diabetes causes endothelial damage and prothrombotic states [13]**;** hypertension induces vascular remodeling and stiffness [14]**;** and prior vascular events reflect systemic inflammation and thrombotic risk. These factors cumulatively lead to microvascular obstruction despite successful epicardial recanalization [15].

Our study findings also revealed significantly higher CHA_2DS_2-VASc-HSF-TT scores, longer chest pain-to-ER times, absence of thrombolysis, lower TIMI flow grades, and worse Killip class in no-reflow cases. These are in agreement with Ipek et al [16], who associated higher CHA_2DS_2-VASc scores with no-reflow in STEMI. Zhang et al [4] also found CHA_2DS_2-VASc-HSF scores predictive of no-reflow. Tasar et al [17] identified pain-to-balloon time >4 hours and lower pre-PCI TIMI flow as significant predictors. Likewise, d'Entremont et al [2] reported higher no-reflow incidence with higher Killip class. Absence of thrombolysis may increase thrombus burden and distal embolization [18]**.** Lower TIMI flow reflects severe coronary occlusion, and poor Killip class reflects systemic instability, both contributing to impaired reperfusion [19].

Contrary to our findings, Fajar et al [20] reported a paradoxical inverse relation between smoking and no-reflow. Also, d'Entremont et al [2] found no significant difference in age between no-reflow and non-no-reflow groups. In our study, no-reflow patients had significantly longer chest pain-to-PCI times, with fewer procedures within 3 hours and more delays beyond 3–6 and 6 hours, highlighting delayed intervention as a key factor in no-reflow. Our study findings align with Khalfallah et al [3]**,** who identified ischemia times exceeding 6 hours as an independent predictor of no-reflow, showing a significant negative correlation between ischemia time and TIMI flow post-PCI. Delayed PCI likely worsened microvascular injury through prolonged ischemia, endothelial dysfunction, and capillary edema [21]**.** Longer ischemic times also promote neutrophil infiltration, thrombus propagation, and microvascular plugging, impairing reperfusion even after epicardial flow restoration [22]**.** Additionally, prolonged ischemia exacerbates myocardial stiffness and left ventricular dysfunction [23], reinforcing the importance of timely reperfusion. Our study also showed no-reflow cases had more LCX and LAD involvement and larger vessel diameters, with longer door-to-balloon times, more stent use, and post-dilatation, but no significant differences in OM, or RCA involvement. These findings are consistent with Tasar et al [17]**,** who identified a reference vessel diameter ≥3.5 mm as a significant predictor of no-reflow in STEMI patients. Pantea-Roșan et al [24] observed an association between multiple stent use and post-dilatation with increased no-reflow risk, although they found lower LCX involvement in no-reflow cases compared to reflow. Ohshima

et al [25] noted plaque ruptures were more evenly distributed in LCX, while LAD and RCA ruptures were mostly proximal. Fajar et al [20] highlighted that smaller vessel diameters are associated with higher no-reflow incidence, though differences in study populations or measurement techniques may explain discrepancies. Our study found no-reflow occurred in 22.35% of cases and was linked to worse outcomes, including higher arrhythmia rates, lower ejection fraction, more acute heart failure, and significantly increased mortality. This aligns with Ndrepepa et al [26]**,** who reported lower left ventricular ejection fraction in the no-reflow group at 6 months post-PCI (47.7% ± 13.1% vs. 54.2% ± 13.9%, $p < 0.001$). Tasar et al [17] found heart failure was significantly more common in no-reflow patients (32.1% vs. 8.7%, $p < 0.001$). d'Entremont et al [2] also observed higher cardiovascular death rates in no-reflow patients at 1 year (15.8% vs. 6.3%, adjusted HR 1.70, $p = 0.01$). No-reflow results in persistent microvascular obstruction, limiting the myocardial perfusion despite epicardial recanalization leading to larger infarcts and more ventricular dysfunction [27]**.** This ischemic injury increases arrhythmia risk, while impaired contractility and fluid overload contribute to acute heart failure [28]**.** Severe myocardial damage and hemodynamic deterioration elevate mortality risk due to fatal arrhythmias and pump failure [29]**,** explaining worse outcomes in no-reflow cases. No-reflow in our study was positively associated with heart failure, diabetes, hypertension, delayed presentation, LCX and LAD involvement, longer door-to-balloon times, and more stents, while negatively associated with diagonal artery involvement, thrombolysis, better TIMI flow, and preserved ejection fraction. Our findings are consistent with Fajar et al [20]**,** who reported delayed reperfusion increases no-reflow risk and that higher left ventricular ejection fraction correlates with better outcomes and lower no-reflow incidence. Our study found the CHA_2DS_2-VASc-HSF-TT score predicted no-reflow well (AUC 0.846), but chest pain-to-PCI time >6 hours was an even stronger predictor (AUC 0.955), emphasizing the critical role of delayed reperfusion. This is in agreement with Zhang et al [4]**,** who found that a CHA_2DS_2-VASc-HSF score ≥4 predicted no-reflow with an AUC of 0.755. Abd El-Kader et al [5] reported higher CHA_2DS_2-VASc-HSF scores in no-reflow patients ($p < 0.001$), with an AUC of 0.868, sensitivity of 93.75%, and specificity of 66.67%. Thus, we believe that the CHA_2DS_2-VASc-HSF-TT score can be used in the emergency department for rapid triage and identifying patients at high risk of no-reflow, so we can arrange its management measures and medications.

Limitations

Single-center, cross-sectional design limited the generalizability of the study. Detailed angiographic thrombus burden or microvascular resistance indices weren't included. Retrospective assessment of time delays could introduce recall bias. This can be considered in larger multicenter studies to generalize the predictive value of the novel score.

CONCLUSION

CHA_2DS_2-VASc-HSF-TT score is a strong predictor of no-reflow in STEMI patients undergoing primary or post-thrombolysis PCI, with a cutoff ≥5 providing high sensitivity (82.5%) and specificity (79.3%). Prolonged ischemia time (>6 hours to PCI) emerged as a strong independent predictor of no-reflow (AUC 0.955), highlighting the critical importance of minimizing delays in revascularization. We also advise to give thrombolytic therapy as abridge to PCI if expected time delay to intervention. No-reflow was associated with worse clinical outcomes, including higher mortality, arrhythmias, acute heart failure, and reduced ejection fraction, emphasizing the need for early risk stratification using this enhanced scoring system and timely intervention to improve prognosis in high-risk STEMI patients.

Financial Disclosure: None
Conflict of Interest: None

REFERENCES

1. Kumar D, Ahmed I, Bardooli F, Saghir T, Sial J A, Khan K A, et al. Techniques to treat slow-flow/no-reflow during primary percutaneous coronary intervention. Cardiovascular Revascularization Medicine. 2023; 47, 1-4.

2. d'Entremont M A, Alazzoni A, Dzavik V, Sharma V, Overgaard C B, Lemaire-Paquette, et al. No-reflow after primary percutaneous coronary intervention in patients with ST-elevation myocardial infarction: an angiographic core laboratory analysis of the TOTAL Trial. EuroIntervention, 2023;19(5), e394-e401.

3. Khalfallah M, Allaithy A, Maria D A. Impact of the total ischemia time on no-reflow phenomenon in patients with ST elevation myocardial infarction undergoing primary percutaneous coronary intervention. Anatolian Journal of Cardiology. 2022; 26(5), 382.

4. Zhang Q Y, Ma S M, Sun J Y. New CHA_2DS_2-VASc-HSF score predicts the no-reflow phenomenon after primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction. BMC Cardiovascular Disorders. 2020; 20, 1-9.

5. Abd El-Kader M, Hamed A M B, Rasheed H K, Farag S I. Prediction of angiographic (TIMI GRADE) blood flow using the novel CHA_2DS_2-VASC-HSF score in patients with STEMI. IJCS. 2024; 6(2), 114-23.

6. Vicent L, Velásquez-Rodríguez J, Valero-Masa M J, Díez-Delhoyo F, González-Saldívar H, Bruña V, et al. Predictors of high Killip class after ST segment elevation myocardial infarction in the era of primary reperfusion. International Journal of Cardiology. 2017; 248, 46-50.

7. Sukmawan R, Hoetama E, Suridanda Danny S, Giantini A, Listiyaningsih E, Gilang Rejeki V, et al. Increase in the risk of clopidogrel resistance and consequent TIMI flow impairment by DNA hypomethylation of CYP2C19 gene in STEMI patients undergoing primary percutaneous coronary intervention (PPCI). Pharmacology research & perspectives. 2021; 9(2), e00738.

8. Pallant Julie. SPSS survival manual: a step by step guide to data analysis using SPSS for Windows, Open University Press. 2007.

9. Figtree G A, Vernon S T, Hadziosmanovic N, Sundström J, Alfredsson J, Arnott C, et al. Mortality in STEMI patients without standard modifiable risk factors: a sex-disaggregated analysis of SWEDEHEART registry data. Lancet. 2021; 20;397(10279):1085-1094. doi: 10.1016/S0140-6736(21)00272-5.

10. Mansour H, Elabd A, Ghazy M, Sameer W, Abdelhamid I, Tamara A. Clinical and Laboratory Predictors of No-reflow during Primary Percutaneous Coronary Intervention. The Egyptian Journal of Hospital Medicine. 2021; 83(1), 1022-9.

11. Dönmez E, Özcan S, Şahin İ, Okuyan E. Predictors of No-reflow Phenomenon Development in Patients Presenting with ST Segment Elevated Myocardial Infarction and Treated with Primary Percutaneous Coronary Intervention. 2023.

12. Zhao S R, Huang R, Liu F, Li Y, Gong Y, Xing J. Study on Correlation between Type 2 Diabetes and No-Reflow after PCI. Disease markers. 2022; 2022(1), 7319277.

13. Dhananjayan R, Koundinya K S, Malati T, Kutala V K. Endothelial dysfunction in type 2 diabetes mellitus. Indian Journal of Clinical Biochemistry. 2016; 31, 372-9.

14. Humphrey J D. Mechanisms of vascular remodeling in hypertension. American journal of hypertension. 2021; 34(5), 432-41.

15. Aldujeli A. Coronary Microvascular Dysfunction in Patients with ST-Elevation Myocardial Infarction: Diagnostics, and Outcomes Lithuanian University of Health Sciences (Lithuania)]. 2024.

16. Ipek G, Onuk T, Karatas M B, Gungor B, Osken A, Keskin M, et al. CHA_2DS_2-VASc score is a predictor of no-reflow in patients with ST-segment elevation myocardial infarction who underwent primary percutaneous intervention. Angiology. 2016; 67(9), 840-45.

17. Tasar O, Karabay A K, Oduncu V, Kirma C. Predictors and outcomes of no-reflow phenomenon in patients with acute ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Coronary artery disease. 2019; 30(4), 270-76.

18. Alexiou S, Patoulias D, Theodoropoulos K C, Didagelos M, Nasoufidou A, Samaras A, et al. Intracoronary thrombolysis in ST-segment elevation myocardial infarction patients undergoing primary percutaneous coronary intervention: an updated meta-analysis of randomized controlled trials. Cardiovascular Drugs and Therapy. 2024; 38(2), 335-46.

19. Sahsah A A M. E.-S., Abo_Elazm T H, Atyeia A I, Assaf T H, Darwish M S. Shock Index as a predictor for Coronary Slow/No Reflow in Patients with Acute STEMI Undergoing Primary Percutaneous Coronary Intervention. Benha Medical Journal. 2025; 42(2), 48-57.

20. Fajar J K, Heriansyah T, Rohman M S. The predictors of no reflow phenomenon after percutaneous coronary intervention in patients with ST elevation myocardial infarction: A meta-analysis. Indian heart journal. 2018; 70, S406-S418.

21. Kanthallu S N, Sathyamurthy I. Coronary slow flow/no-reflow: revisited. Journal of Indian College of Cardiology. 2022; 12(1), 8-13.

22. Zhao Y, Tan M, Yin Y, Zhang J, Song Y, Li H, et al. Comprehensive macro and micro views on immune cells in ischemic heart disease. Cell proliferation. 2024; 57(12), e13725.

23. Singh A, Ashraf S, Irfan H, Venjhraj F, Verma A, Shaukat A, et al. Heart failure and microvascular dysfunction: an in-depth review of mechanisms, diagnostic strategies, and innovative therapies. Annals of Medicine and Surgery. 2025; 87(2), 616-26.

24. Pantea-Roșan L R, Pantea V A, Bungau S, Tit D M, Behl T, Vesa C M, et al. No-reflow after PPCI—a predictor of short-term outcomes in STEMI patients. Journal of Clinical Medicine. 2020; 9(9), 2956.

25. Ohshima K, Ikeda S, Kadota H, Yamane K, Izumi N, Kawazoe H, et al. Cavity volume of ruptured plaque is an independent predictor for angiographic no-reflow phenomenon during primary angioplasty in patients with ST-segment elevation myocardial infarction. Journal of cardiology. 2011; 57(1), 36-43.

26. Ndrepepa G, Tiroch K, Keta D, Fusaro M, Seyfarth M, Pache J r, et al. Predictive factors and impact of no reflow after primary percutaneous coronary intervention in patients with acute myocardial infarction. Circulation: Cardiovascular Interventions. 2010; 3(1), 27-33.

27. Annibali G, Scrocca I, Aranzulla T C, Meliga E, Maiellaro F, Musumeci G. "No-reflow" phenomenon: a contemporary review. Journal of Clinical Medicine. 2022; 11(8), 2233.

28. Severino P, D'Amato A, Pucci M, Infusino F, Birtolo L I, Mariani M V et al. Ischemic heart disease and heart failure: role of coronary ion channels. International journal of molecular sciences. 2020; 21(9), 3167.

29. Fishbein G A, Fishbein M C, Wang J, Buja L M. Myocardial ischemia and its complications. In Cardiovascular pathology, Elsevier. 2022; 407-445

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