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The SYNTAX Score and Angiographic "No-Reflow" in Patients with Acute Myocardial Infarction Undergoing Percutaneous Coronary Intervention

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ABSTRACT

Background: in patients with acute myocardial infarction, the immediate therapeutic goal is to establish patency of the infarct-related artery and to achieve optimal myocardial tissue reperfusion. The Synergy between percutaneous coronary intervention with TAXUS and cardiac surgery (SYNTAX) score (SS) quantifies the extent and complexity of angiographic coronary artery disease.

Patients and Methods: a total of 543 patients presenting with acute myocardial infarction undergoing primary PCI for the STEMI patients and early invasive strategy for the NSTEMI patients, both admitted within 24 hours from the symptoms onset, were analyzed. SS, thrombolysis in myocardial infarction (TIMI) flow grade score, and TIMI myocardial blush grade score (MBG) were determined in all patients. No-reflow was considered as the prescence of TIMI blood flow in the infarct related artery (IRA) ≤ 2 or TIMI grade 3 with myocardial blush grade (MBG) 0 or 1, at least 10 minutes after the end of the PCI procedure.

Results: no-reflow was observed in 26% of patients. The mean SS of the no-reflow group was higher than that of the TIMI III flow group . On multivariate logistic regression analysis a long target lesion (OR= 8.637, 95% C.I 1.975–37.768, p = 0.004) were found to be significantly associated with no-reflow and were the independent predictors of no-reflow phenomenon. The cutoff value of SS obtained by the receiver-operator characteristic curve analysis was 31 for the prediction of no-reflow .

Conclusion: the SS is a predictor of no-reflow in patients with acute myocardial infarction treated with percutaneous coronary intervention.

Keywords: acute myocardial infarction, percutaneous coronary intervention, No-reflow, SYNTAX score

INTRODUCTION

In patients with acute myocardial infarction, the successful restoration of epicardial culprit coronary artery patencydoes not always guarantee restoration of myocardial tissue-level perfusion & salvage of myocardium at risk of ischemia[(1)]. In a variable proportion of patients with acute myocardial infarction, however, microcirculatory impairment may persist after epicardial coronary artery recanalization following PCI and may attenuate its beneficial impact[(2)]. The phenomenon of myocardial no-reflow is defined as inadequate myocardial perfusion through a given segment of the coronary circulation without angiographic evidence of mechanical vessel obstruction[(3)].

Further, recent studies have revealed that distal embolizationof thrombusand/or plaque contents are one of the major causes ofno-reflow. It may be critically important, therefore, to be able to predict which lesions are high risk for myocardial no-reflow prior to beginning percutaneous coronary intervention (PCI)[(4)].

Accurate detection of 'no-reflow' is thus crucial because it is independently associated with low ventricular ejection fraction, adverse left ventricular remodeling, malignant arrhythmias, cardiac failure, as well as mortality both at short- and long-term follow-up[(5)].

The SYNTAX (synergy between percutaneous coronary intervention with TAXUS and cardiac surgery) score has been shown to be predictive of clinical outcome in different clinical settings in patients undergoing percutaneous coronary intervention (PCI)[(6)].

PATIENTS AND METHODS

Study population:

The study was conducted on 543 patients with acute myocardial infarction undergoing primary PCI for the STEMI patientsand early invasive strategy for the NSTEMI patients, both admitted within 24 hours from the symptoms onset to the cath lab. Of Al-Hussein University hospital during the period from 11/2015 to 11/2017.

The study was approved by the Ethics Board of Al-Azhar University and an informed written consent was taken from each participant in the study.

The study **inclusion criteria** were patients presenting with an evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia. Under these conditions the following criteria meets the diagnosis for myocardial infarction[(7)]: Detection of a rise and /or fall of cardiac biomarkers values (preferably cardiac troponin (cTn) with at least one of the following:

- Symptoms of ischemia.

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- New or presumed new significant ST-segment-T wave (ST-T) changes or new left bundle branch block(LBBB).
- Development of pathological Q waves in the ECG.
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Identification ofan intracoronary thrombus by angiography.

Acute myocardial infarction patients included:

1- STEMI (ST segment elevation myocardial infarction).

2- NSTEMI (non- ST segment elevation myocardial infarction).

The **exclusion criteria** were patients with a prior CABG surgery, patients with prior PCI and stenting and patients with poor quality coronary angiograms.

All enrolled patients underwent the following: Full history taking.

Clinical examination, 12 lead electrocardiography, serial cardiac biomarkers, renal functions, weight and height measurements, Echo-cardiographic study at the hospital stay.

A written informed consent was obtained from all the patients enrolled in the study and was approved from the local Ethical Committee.

Angiographic procedure

PCI was done to treat the de novo culprit lesions with significant stenosis in the native coronary artery. The culprit lesion was identified on the basis of E.C.G, transthoracic echocardiography and coronary angiography.theSYNTAX score was calculated according to The SYNTAX score algorithm**[8].**

Angiographic criterion was used for the diagnosis of no reflow. Coronary angiography was performed according to the standard criteria. Offline analysis of digital angiograms was performed in the core laboratory using automated edge detection.

The initial and postprocedural blood flow in the infarct-related artery was gradedaccording to the Thrombolysis in Myocardial Infarction (TIMI) flow grading system[9]and TIMI myocardial blush grade (MBG) score[10]. The diagnosis of no reflow required the following criteria[11]:

- Angiographic evidence of reopening of occluded coronary arteryand successful stent placement with no evidence of flow-limitingresidualstenosis (≥ 50%), dissection, spasm, or apparent thrombus and
- Angiographic documentation of a TIMI flow grade≤ 2or TIMI grade 3 with MBG 0 and 1, atleast 10 minutes after the end of PCI procedure.

Angiographic criterion of 25% residual stenosis was adopted as a definition of successful PCI and the endpoint of the interventional procedure.

Statistical study:

All statistical studies were carried out using the statistical package for social sciences (18.0 for Windows; SPSS Inc., Chicago, Illinois, USA).

Quantitative variables were expressed as mean±SD. Qualitative data were expressed as counts and percentages. The Student t-test and the $\chi 2$ -test were used to compare quantitative and qualitative values, respectively. Multivariable logistic regression analysis was performed to identify independent predictors of no-reflow phenomenon. The receiving operator characteristic (ROC) curve was used to detect optimal cutoff values of Syntax score for predicting no-reflow. A P value less than 0.05 was considered statistically significant.

RESULTS

Five hundred forty three patients with acute myocardial infarction who underwent primary PCI for the STEMI patients (362) and early invasive strategy for the NSTEMI patients (181), both admitted within 24 hours from the symptoms onset were included in the study. Patients were divided into two groups according to the TIMI flow post PCI; the TIMI III flow group (group I) included 402 patients (74%) and no-reflow group (group II) that included 141 patients (26%).

Baseline clinical characteristics

- There were no significant differences between the two groups in terms on gender , DM , HTN, Dyslipedimia, smoking status, BMI, the family history of CAD, the hemodynamic profile and the types ofmyocardial infarction.Patients in the No-reflow group were older, had higher prevalence of PVD, higher peak CKMB levels, higher RBS levels on admission, higher level of killip class (III / IV), and they tended to have more worse LV contractility (P<0.001, 0.018, <0.001, <0.001, <0.001, and <0.001, respectively, Table 1).

Table (1): Comparison between demographic and clinical data in group I &II

	Group I (reflow)	Group II (no-reflow)	P value
Age	55.19 ± 10.88	63.19 ± 9.6	**<0.001**
Male gender	351 (87.3%)	132 (93.6%)	0.236
Female gender	51 (12.6%)	9(6.3%)	
Smoker	243 (60.4%)	89 (63.1%)	0.213
DM	162(40.29%)	57 (40.4%)	0.988
HTN	132 (32.8%)	48 (34%)	0.880
Dyslipedimia	249 (61.9%)	93 (65.9%)	0.624
Family history of CAD	45 (11.1%)	9(6.3%)	0.343
BMI	27.45 ± 3.48	28.39 ± 2.97	0.125
Peripheral vascular disease	32 (8%)	41(29.16%	**0.018**
SBP	127.74 ± 25.31	129.11 ± 33.20	0.770
DBP	78.82 ± 11.42	78.43 ± 15.6	0.854
HR	81 ± 7.2	76 ± 10.1	0.683
Killip class			
I	246(61.1%)	36 (25.5%)	
II	126 (31.3%)	78 (55.3%)	**< 0.001**
III	27 (6.7%)	24 (17%)	
IV	3 (0.74%)	3 (2.1%)	
AWMI	150 (37.3%)	48(34%)	0.836
IWMI	52 (12.9%)	24 (17%)	0.309
IWMI + RVMI	64 (15.9%)	20 (14.1%)	0.862
Lateral STEMI	2 (0.49%)	2 (1.4%)	0.772
NSTEMI	134 33.3%)	47 (33.28%)	0.988
Peak CKMB	234.71± 136.21	403.00 ± 144.15	**<0.001**
Creatinine Clearance	96.42±17.42	93.25±10.37	0.970
RBS	144.2±70.3	275.0±76.9	**<0.001**
Total cholesterol	217.74±50.05	219.53±51.30	0.836
EF	53.1±6.0	45.7±7.2	**<0.001**

DM, Diabetes mellitus; HTN, hypertension; CAD ,coronary artery disease; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; AWMI, anterior wall myocardial infarction; IWMI, inferior wall myocardial infarction; RVMI, right ventricular myocardial infarction; STEMI, ST segment elevation myocardial infarction; RBS, random blood sugar; EF, ejection fraction.

Angiographic & PCI Data

- There was a significant difference between patients within both groups for their SYNTAX score denoting higher predicted risks and higher anatomical complexity for No-reflow patients (22.2±5.8 vs 37.4±3.8 for TIMI III & no-reflow patients respectively with a p value <0.001).
- STEMI patients who experienced no-reflow had a longer symptom to balloon time (7.8±2.4 h for noreflow vs 3.4±1.6h for TIMI III group with a p value <0.001) Unlike for the NSTEMI patients who experienced no-reflow who had a median time to revascularization of 19±2.7h which is Comparable to that on TIMI III flow group 16±1.8h with a p value of 0.074 (table 2).
- There were no significant differences between the two groups regarding the number of diseased vessels, infarct-related artery, the target lesion locations, reference vessel diameter, stent length, stent diameter, rate of use of thrombus aspiration device. No-reflow was more common in patients who had a low (≤1) initial TIMI flow (p value <0.001) and a low initial TMP grade (≤1) (p value <0.001), also for patients with high thrombus burden (P < 0.001), and those had longer lesions > 20mm (P value <0.001) (Table 3).

Table (2): No-reflow & acute myocardial infarction (STEMI & NSTEMI): Symptom to balloon time.
STEMI, ST segment elevation myocardial infarction; NSTEMI, non- ST segment elevation myocardial infarction

	Median symptom to balloon time		P-Value
	TIMI III	No-reflow	
STEMI	3.4±1.6h	7.8±2.4	**<0.001**
NSTEMI	16±1.8	19±2.7	0.074

Table (3): Angiographic Data in the TIMI III & No-reflow groups.

	TIMI III	No-reflow	P value
IRA **LAD** **LCX** **Ramus** **RCA**	225 (55.9%) 33 (8.3%) 3 (0.7%) 144 (35.8%)	72 (51.1%) 9 (6.4%) 0 60 (42.6%)	
Initial TIMIflow **0/1** **2/3**	 189 (47%) 213 (53%)	 129 (91.5%) 12 (8.5%)	 **<0.001** **<0.001**
Initial TMP Grade **0/1** **2/3**	 225 (56%) 177 (44%)	 138 (97.9%) 3 (2.1%)	 **<0.001** **<0.001**
Target lesionlocation **Proximal** **Mid** **Distal**	 216 (53.7%) 162 (40.3%) 24 (6%)	 93 (66%) 42 (29.8%) 6 (4.3)	 0.346
Total occlusion	246 (61.2%)	102 (72.3%)	0.059
High thrombus burden	60 (15%)	93 (66%)	**<0.001**
Reference vessel diameter	3.204±0.377	3.123±0.270	0.341
Number of diseaded vessels **1** **2** **≥3**	 101 (25%) 153 (38%) 148 (37%)	 28 (20%) 48 (33%) 64 (45%)	
Thrombus aspiration alone	3 (0.74%)	3 (2.1%)	0.436
Stenting after thrombus aspiration	123 (30.5%)	84 (59.5%)	0.029
Direct stenting alone	123 (30.5%)	18 (12.7%)	0.016
Pre-dilatation	159 (39.5%)	66 (46.8%)	0.385
Post-dilatation	123 (30.5%)	63 (44.6%)	0.080
Target lesion length **>20mm** **<20mm**	 132 (32.8%) 267 (66.4%)	 114 (80.8%) 27 (19.1%)	 **<0.001**
Stent Length	3.2±0.364	3.13±0.25	0.341
Stent diameter	22.11±5.0	20.17±5.9	0.122

IRA, infarct-related artery; TIMI; thrombolysis in myocardial infarction, TMP; TIMI myocardial perfusion grade

A multivariable logistic regression model was built to identify the independent predictors of no-reflow. The SYNTAX score (odds ratio = 1.833, 95% confidence interval, 1.139 – 2.951 , P = 0.013), the time to reperfusion > 6 hours for STEMI patients (OR =13.844, 95% C.I. 3.214-59.636, P =0.003), age > 60 years (OR = 8.884 , 95% C.I.2.145 – 36.800, P= 0.003), low initial TIMI flow (≤ 1) (OR = 20.861 , 95% C.I.1.739 – 250.290, p= 0.017, a long target lesion (OR = 8.637, 95% C.I1.975 – 37.768, p = 0.004) were found to be significantly associated with no-reflow and were the independent predictors of no-reflow phenomenon ((Table 4)). ROC curve analysisidentified syntax score greater than 31 as the best cut off value predictive ofno-reflow phenomenon with sensitivity of 95.1%and specificity of 94.5% and area under ROC curve 97.3% , p value <0.001.((Figure 1)).

Table (4): Independent predictors of No-reflow

	Odds ratio	95% C.I.		P value
		Lower	Upper	
Age	8.884	2.145	36.800	**0.003**
Time to reperfusion	13.844	3.214	59.636	**<0.001**
Target lesion length	8.637	1.975	37.768	**0.004**
Initial TIMIflow	20.861	1.739	250.290	**0.017**
Initial TMPG	0.851	0.036	20.116	0.920
Thrombus burden	3.262	0.769	13.831	0.109
Killip class III / IV	1.468	0.210	10.249	0.698
EF %	2.476	0.191	32.087	0.488
PVD	1.870	0.540	3.212	0.755

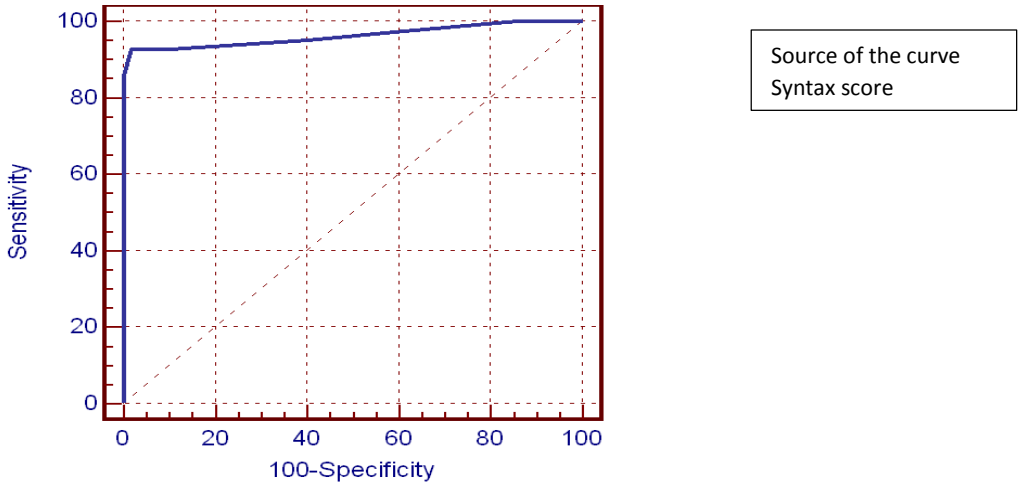

Figure (1): The ROC curve analysis to show the best cutoff value of the SYNTAX score that identifies No-reflow.

DISCUSSION

The present study evaluated the ability of the SS to predict no-reflow phenomenon in patients with acute myocardial infarctiontreated with percutaneous coronary intervention.

The main findings of the present study were as follows. (a) no-reflow phenomenon occurred in 26% of patients with STEMI treated with PPCI and NSTEMI patients treated with early invasive strategy. (b) Patients withno-reflow were older, had higher prevalence of PVD, higher peak CKMB levels, higher RBS levels on admission, higher level of killip class (III / IV), and they tended to have more worse LV contractility, had a longer symptom to balloon time for STEMI patient,.No-reflow was more common in patients who had a low (≤1) initial TIMI flow and a low initial TMP grade (≤1), also for patients with high thrombus burden, and those who had longer lesions > 20mm (c) The multivariable logistic regression model identified The **SYNTAX score**, the time to reperfusion > 6 hours for STEMI patients , age > 60 years, **low initial TIMI flow (≤ 1)**, a **long target lesion** as independent predictors of no-reflow. (d) ROC curve analysis revealed that SS greater than 31 had a sensitivity of 95.1%, specificity of 94.9%, and area under the ROC curve of 97.3 % for predicting no-reflow.

In our study, the incidence of no-reflow was 31%, although the incidence of no-reflow during PCI has ranged from 12% to 25% in some studies that have used the criterion of TIMI flow grade ≤ 2 [12-13]. In some studies, the incidence has been 29% using the TIMI myocardial perfusion grade [21], and 34% to 39% using myocardial contrast echocardiography [14-15].

We found that the high SxS group compared to the low SxS had more no reflow (37.4 ± 3.8 Vs 22.2 ± 5.8 with P value < 0.001),patients with a high SxS had a more complex anatomy of coronary arteries including multivessel disease, diffiuse disease, bifurcation lesions, chronic total occlusion and left main disease. These features could make the procedure of pPCI more difficult and complicated. Therefore, myocardial no-refow could be expected in patients with complex coronary anatomy. **Magro *et al.*[16]** found similar results in their study. They examined 669 patients admitted with STEMI and found that post-PCI no-reflow rate of patients with high SxS (SxS > 16) was significantly high.This was in concordance with **Sahin *et al.*[17]** and **Kammler *et al.*[18]** who all found that high SxS group had more no-reflow.

Iwakura *et al.*[19] found no-reflow phenomenon in 79 of 199 patients (39.6%) using myocardial contrast echocardiography 15 minutes after percutaneous coronary intervention and reported a statistically significant higher agein the no reflow group compared to the optimal flow group (64 years vs. 58 years respectively, p-value= 0.003).

Also, **Cafri *et al.*[20]** and **Oduncu *et al.*[21]** showed a significantly higher age in the no reflow group compared to the optimal flow group Furthermore, delayed reperfusion can result in an older, more organized intracoronary thrombus which may increase the risk of distal embolization during pPCI and reduce the likelihood of achieving TIMI flow grade 3 after the procedure. Our study showed a statically significant **longer pain to door time** in the no reflow group compared to the optimal flow group (7.8±2.4 vs. 3.4±1.6, p-value <.001). This goes in concordance with **McNamara *et al.*[22]** and **Ndrepepa *et al.*[23],** where they all showed that the time from onset ofchest pain to the emergency room arrival was significantly higher in the no reflow group compared to the optimal flow group.

Delayed reperfusion (a long duration from onset to reperfusion) is related to no-reflow. Our study showed that patients with a long duration before reperfusion (> 6 h) had a significantly greater **thrombus burden** and an increase in the no-reflow rate than patients with a short duration of reperfusion.

This is in concordance with **Yip *et al.*[24]**who demonstrated that in patients with AMI who had a high thrombus burden, the rate of no-reflow was lower than in those with reperfusion in less than 4 h. This indicates the possible correlation of a thrombus burden with the duration of reperfusion. **Tanaka *et al.*[25]** used IVUS to examine plaque burden and identified that a higher lipid content in the inner plaque core and the width of the external elastic membrane were independent markers for the no-reflow phenomenon.

Systolic function of the left ventricle after AMI is one of the most important predictors of long-term outcomes [26]. In our study, patients with high SxS had lower **EF** because more no-reflow developed in patients with high SxS, inadequate reperfusion occurred in the myocardium despite patent IRA.

Our study revealed that Patients with lesions that were **longer than 20 mm**were more likely to develop no-reflow after primary PCI than those with lesions that were shorter than 20 mm in size. Large vessels are able to contain large amounts of plaque lipid or thrombi. The larger the lesioned vessels, the slower the flow velocity. The longer the target lesion, the larger the amount of thrombus and plaque burden. This would explain the high risk for slow/no-reflow that was observed in these patients after primary PCI[27].

This goes with what **Kirma*et al.*[28]** reportedin a series of 382 consecutive patients with AMI who underwent primary PCI who showed that advanced age (> 60 years), delayed reperfusion (≥4 h), **low (≤1) TIMI flow prior to PCI**, cut-off type total occlusion, high thrombus burden according to baseline angiography, the presence of a long target lesion (>13.5 mm) and large vessel diameter all correlated with no-reflow.

Among the multiple strategies postulated to prevent and treat this phenomenon, **direct stenting without predilation** have demonstrated a net clinical benefit. We found a statistically significant higher proportion of patientswho were subjected to direct stenting in the optimalflow group compared to the no reflow group (60.8%vs. 38.8% respectively with p-value <0.001). Wefound no significant difference between no reflowgroup and optimal flow group as regards the useof **thrombus aspiration** as an adjunctive to pPCI. Probably, a better selection of patients before theprocedure and an earlier implementation of thepresent or other promising strategies, as the combination of thrombus aspiration and intracoronary infusion of IIb-IIIa **Stone *et al.*[29]** might represent a greater benefit.

Limitation of this study:

The present study has several limitations. Firstly, part of the study was retrospective performedat a single center which could have led to bias andmodified the outcomes. The diagnosis of no-reflowwas made considering only the epicardial flow and that we didn't analyze the microvascular function and no-reflow using myocardial contrast echocardiography or nuclear scintigraphy.

Despite these considerations, we think that these results are convincing and highly significant, and should be confirmedby prospective studies.

CONCLUSIONS

Syntax score (Sxs) is an independent predictor of no reflow phenomenonand thus can be used to stratify AMI patients into low or high risk for angiographic no-reflow.

Predictors of no reflow after primary PCI using univariable analysis showed 9 variables (age, time to reperfusion,target lesion length, initial TIMI flow , initial TMPG , thrombus burden , Killip class III / IV , EF , PVD.).

The confirmation of these findingsin prospective studies might allow the implementation of strategies to prevent this phenomenonand eventually improve the long term clinicaloutcomes.

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