Research Article

Clinical and Procedural Predictors of Myocardial Reperfusion in Primary Percutaneous Coronary Interventions

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Abstract
Background*:* Optimal myocardial perfusion in patients with ST elevation myocardial infarction (STEMI) who undergo primary percutaneous coronary intervention (PCI) is associated with better clinical outcomes, while suboptimal myocardial perfusion (No-reflow) associated with increased infarct size, an increased rate of left ventricular (LV) dysfunction, and higher mortality rates. The aim of the the study is to identify Clinical and Procedural Predictors of suboptimal myocardial reperfusion in Primary Percutaneous Coronary Intervention. **Methods**: A series of ١٠٠ patients with STEMI who underwent primary PCI within ١٢ h of symptom onset and also patients with ischemic symptoms continuing for ١٢-٢٤h were included. Patients in our study were classified into ٢ groups according to myocardial reperfusion; optimal myocardial reperfusion group with final myocardial blush grade (MBG)=٣, included ٧٣ patients and suboptimal myocardial reperfusion group with MBG≤ ٢, included ٢٧ patients. Suboptimal myocardial reperfusion. Clinical characteristics, electrocardiographic measures, laboratory investigations on admission, angiographic and procedural data were collected for each subject and were compared between both groups. **Results**: suboptimal reperfusion occurred in ٢٧٪ of patient study. Suboptimal reperfusion group compared with optimal reperfusion group had statistically significant fewer patients with history of angina prior to MI ٥ (١٨.٥٪) vs ٤٤ (٦٠.٣٪) , fewer patients with current aspirin intake until admission ٦ (٢٢.٢٪) vs ٣٨ (٥٢٪), increased blood glucose level on admission (٢٤٠±١٠١mg/dl vs ١٧١±٧٢mg/dl), increased TLC on admission (١٢.١±٣.٦ vs ١٠.٢±٣.٣) $١٠^{٣}$ /$mm^{٣}$, longer reperfusion time (٦.١±٢.٨ vs ٤.٣±٢.١ h), more patients with high thrombus burden ١٢ (٤٤.٤ %) vs ١٣ (١٧.٨ %), higher predilatation pressure (١٦.١±٢.٣ vs ١٤.٧ ± ١.٨ ATM), more repeated balloon inflation (>twice) during predilatation ٢٤ (٩٢.٣%) vs ٤٦ (٦٩.٧٪), (P <٠.٠٥ for all). repeated predilatation had odd ratio ٨.٢, high thrombus burden had odd ratio ٥, absence of angina prior to MI had odd ratio ٤.٣٨, TLC > ١٠ ($١٠^{٣}$/$mm^{٣}$) had odd ratio ٣.٤, blood glucose > ١٦٠ mg/dl had odd ratio ٣.٠, reperfusion time > ٤h had odd ratio ٢.٦٨, no current aspirin therapy had odd ratio ٢.٢٩ and predilatation pressure > ١٥ ATM had odd ratio ٢.٢٣. **Conclusion:** Based on the results of this study it can be concluded that repeated balloon inflation during predilatation, high thrombus burden, no history of angina prior to MI, high TLC, high blood glucose level, longer reperfusion time, no current aspirin intake before MI and high predilatation pressure were predictors for suboptimal reperfusion.

Keywords: No-reflow, STEMI and Myocardial blush

Introduction
Primary percutaneous coronary intervention (PPCI) is the preferred reperfusion strategy for treating acute ST-segment elevation myocardial infarction (STEMI). The main goals are to restore epicardial infarctrelated artery patency and achieve microvascular reperfusion as early as possible, thus limiting the extent of irreversibly injured (necrotic) myocar-dium(١). Adequate myocardial perfusion after primary PCI is associated with better clinical outcomes, whereas suboptimal myocardial perfusion associated with increased infarct size, an increased rate of left ventricular (LV) dysfunction and higher mortality rates(٢). Restoration of normal epicardial blood flow (TIMI grade ٣) does not mean optimal reperfusion at the microcirculation level. Myocardial blush grade (MBG) has been well validated as an angiographic technique to assess myocardial perfusion in patients with ST-elevation acute myocardial infarction (STEMI). MBG correlates with the more accurate method

of evaluating myocardial perfusion (magnetic resonance imaging) and enables the immediate evaluation of microvascular patency while in the catheterization laboratory[٢].

We aim to identify clinical and procedural predictors of suboptimal myocardial perfusion judged by MBG in patients with STEMI who undergo primary PCI.

Patient and Methods

The current study is prospective non randomized study included ١٠٠ patients with acute STEMI who were subjected to primary PCI ≤١٢ h duration from onset of chest pain or ١٢ -٢٤ h after onset of chest pain with signs or symptoms of continuing ischemia. Patients with severe left main or multivessel coronary artery disease who in need for urgent surgical revascularization and patients with saphenous vein grafts lesions were excluded.

All patients were subjected to the following:

١- Thorough history taking including history of risk factors for CAD as diabetes mellitus, systemic hypertension, smoking, dyslipidemia and family history of premature CAD, previous myocardial infarction, angina prior to MI and history of previous medication.

٢- Clinical examination on admission including Killip class.[٣]

٣- laboratory investigations on admission (blood glucose level, Total leucocytic count (TLC), blood urea nitrogen (BUN), serum creatinine, heamoglobin (Hb), and platelets).

٤- Twelve lead Surface ECG: For evidence and site of myocardial infarction and calculation of ST segment resolution (STR) as sum of ST elevation at ECG ٩٠ minutes after primary PCI / sum of ST elevation at initial ECG, STR was classified according to the Schröder classification as complete (٧٠٪ to ١٠٠٪), partial (٣٠٪ to ٦٩٪), or absent (<٣٠٪)[٤].

Interventional variables:

Primary PCI was done by experienced operators on call according to standard protocols. Unless contraindicated, all patients received; aspirin ٣٠٠ mg, nitroglycerin infusion, oxygen supplementation, intravenous bolus of ٥٠٠٠-١٠٠٠٠ units of heparin, clopidogrel (loaded with ٦٠٠ mg followed by ٧٥ mg per day) and Glycoprotein IIb/IIIa receptor antagonist infusion. Other anti-ischemic drugs like B-blockers, anti-arrhythmics, inotropics and vasopressors were given when indicated. Culprit vessels were identified by the operator according to leads involved at ECG. Selection of guiding catheter and guide wire, usage of catheter aspiration device, predilatation, maximum balloon inflation pressure, stenting for residual lesions and post dilatation were determined according to operator discretion.

Experienced interventional cardiologist who was unaware of patient's clinical data carefully assessed a set of parameters for each angiogram:

١-**Thrombus burden**: It was classified according to a modified TIMI Thrombus Grade classification into five grades: Grade ٠: No angiographic characteristics of thrombus are present, Grade ١: Possible thrombus is present (reduced contrast density, haziness, or irregular lesion contour), Grade ٢: There is definite thrombus, with greatest dimensions ≤ ٠.٥ vessel diameter, Grade ٣: Definite thrombus, with greatest linear dimension > ٠.٥ but <٢ vessel diameters, Grade ٤: Definite thrombus, with the largest dimension ≥٢ but <٤ vessel diameters, Grade ٥: Definite thrombus, with the largest dimension ≥٤ vessel diameters. Then we reclassified thrombus burden into: **Absent** thrombus burden: includes thrombus TIMI grade ٠. **Low** thrombus burden: includes thrombus TIMI grade ١&٢. **Moderate** thrombus burden: includes thrombus TIMI grade ٣. **High** thrombus burden: includes thrombus TIMI grade ٤&٥.

٢- **Determination of TIMI flow grading and myocardial blush grading (MBG)** at basic angiography and at end of procedure .

TIMI flow grade Classification was defined as follow: TIMI flow ٠: no antegrade flow beyond the point of occlusion. TIMI flow ١: the contrast material passes beyond the area of obstruction but "hangs up" and fails to opacify the entire coronary artery. TIMI flow ٢: the contrast material passes across the obstruction and opacifies the coronary bed distal to the obstruction, but the rate of entry of contrast into the vessel distal to the obstruction and/or its rate of clearance from the distal bed is slower than its entry into and/or clearance from comparable areas not perfused by the culprit vessel. TIMI flow ٣: antegrade flow into the bed distal to the obstruction occurs as promptly as into the bed

proximal to the obstruction and clearance of contrast material from the involved bed is as rapid as from an uninvolved bed in the same vessel or the opposite artery. Myocardial blush was graded according to the dye density score of the myocardial territory subtended by the infarct related vessel proposed by van't Hof et al from grade ٠ to ٣ as follows: MBG=٠: No contrast density or abnormal persistence of contrast medium. MBG=١: Minimal myocardial blush or contrast density. MBG=٢: Moderate myocardial blush or contrast density but less than that obtained during angiography of a contralateral or ipsilateral non-infarct related coronary artery. MBG=٣: Normal myocardial blush or contrast density, comparable with that obtained during angiography of a contralateral or ipsilateral non-infarction related coronary artery[٥].

Reperfusion time: was defined as time from the onset of chest pain to the first balloon inflation.

Statistical analysis

The Statistical Package for Social Sciences (SPSS) software (version ٢٠) was used for data entry and analysis. Categorical variables were expressed as frequency tables and compared with chi square statistics test. While continuous variables were expressed as mean and standard deviation (SD) and were compared using t test. A probability level of P ≤ ٠.٠٥ was chosen to be significant. Multiple regression analysis was used to determine power of the predictors. Roc curve was used to determine accuracy and cut off value of continuous variables.

Results

Patients were divided into two groups according to final myocardial blush grade (MBG): Optimal reperfusion group: Patients who had final MBG= ٣, included ٧٣ patients (٧٣٪) and suboptimal reperfusion group Patients who had final MBG ≤٢, included ٢٧patients (٢٧٪).

Clinical characteristics:

There was no statistically significant difference between both groups regarding their age, male gender, current smoking, history of hyper-tension, family history of early CAD, or history of dyslipidemia, history of previous MI, history of current beta blockers, Angiotensin-converting enzyme inhibitors (ACEI) or statins therapies, Heart rate, blood pressure and Killip Class on admission. (p>٠.٠٥ for all). Suboptimal reperfusion group had statistically significant fewer patients with history of angina prior to MI ٥(١٨.٥)% vs ٤٤(٦٠.٣٪), fewer patients with current aspirin intake ٦(٢٢.٢٪) vs ٣٨(٥٢٪) as compared to optimal reperfusion group (P=٠.٠٠١ and P=٠.٠٠٨ respectively) (tab ١).

Table ١: Comparison between optimal and suboptimal reperfusion groups regarding their clinical data

	Optimal reperfusion (n=٧٣)	Suboptimal reperfusion (n= ٢٧)	P value
Age (years) **(mean ± SD)**	٥٤ ±١٠	٥٦ ±١٣	٠.٢٦٨
Male n (%)	٦٣ (٨٦.٣٪)	١٩ (٧٠.٤٪)	٠.٠٦٦
Smoker n (%)	٤٤ (٦٠.٣٪)	١٤ (٥١.٩٪)	٠.٤٤٩
HTN n (%)	٢٤ (٣٢.٩٪)	١٤ (٥١.٩٪)	٠.٠٨٣
Family history of CAD n (%)	١٢ (١٦.٤٪)	٩ (٣٣.٣٪)	٠.٠٦٦
Dyslipidemia n (%)	١٧ (٢٣.٣٪)	٧ (٢٥.٩٪)	٠.٧٨٤
DM n (%)	٢٢ (٣٠.١٪)	١٤ (٥١.٩٪)	٠.٠٥٧
Previous MI n (%)	٦ (٨.٢٪)	٣(١١.١٪)	٠.٦٥٤
Angina prior to MI n (%)	٤٤ (٦٠.٣ %)	٥(١٨.٥٪)	٠.٠٠١
Current therapies:			
ASA n (%)	٣٨(٥٢٪)	٦(٢٢.٢٪)	٠.٠٠٨
BB n (%)	١٦ (٢١.٩٪)	٦(٢٢.٢٪)	٠.٩٧٤
ACEI n (%) Statins	١٣ (١٦.٨٪)	٨ (٢٩.٦٪)	٠.١٩٨
n (%)	١٩ (٢٦ %)	٩ (٣٣.٣٪)	٠.٤٧٠

Laboratory data on admission:
Suboptimal reperfusion group had statistically significant increased mean blood glucose level on admission (٢٤٠±١٠١mg/dl vs ١٧١±٧٢mg/dl), increased mean TLC on admission (١٣.٥٤±٢.٦٤ vs ١٠.٢٤±٣٠) $١٠^{٣}$ /$mm^{٣}$ as compared to optimal reperfusion group P=٠.٠٠١, and P= ٠.٠٠١ respectively. While there is no statistically significant difference between both groups regarding BUN, serum creatinine, Hb and platelets on admission (P> ٠.٠٥ for all).(tab٢).

Table ٢: Comparison between optimal and suboptimal reperfusion groups regarding laboratory findings

	Optimal reperfusion (n= ٧٣)	*Suboptimal reperfusion (n= ٢٧)*	P value
Blood glucose level *(mg/dl)***(mean± SD)**	١٧١ ±٧٢	٢٤٠ ±١٠١	٠.٠٠١
TLC *($١٠^{٣}$/ $mm^{٣}$)* **(mean ± SD)**	١٠.٢٦ ±٣.٣٣	١٢.٠٨ ±٣.٦٥	٠.٠٢٠
BUN *(mg/dl)* **(mean ± SD)**	١٢.٧٣ ±٣.١	١٤.٣٧±٥.١٨	٠.٠٥٨
S. creatinine *(mg/dl)* **(mean ± SD)**	١ ±٠.٢٤	١.٠٥ ±٠.٤٧	٠.٤٨٤
Hb *(g/dl)* **(mean ± SD)**	١٤.٠٦ ±١.٤٧	١٣.٤٧ ±١.٧٨	٠.٠٩٩
Platelets *($١٠^{٣}$/ $mm^{٣}$)* **(mean ± SD)**	٢٥٥.٠٨±٦٣.٣٩	٢٦٠.٥١±٦٦.٩٧	٠.٧١٠

ECG findings:
There was no statistically significant difference between both groups regarding site of acute MI (P= ٠.٤٤٧). Suboptimal reperfusion group had statistically significant less ST segment resolution (STR), as it was complete STR in ٤ patients (١٤.٨٪) vs ٥٩ patients (٨٠.٨٪), partial STR in ٤ patients (١٤.٨٪) vs ١٢ patients (١٦.٤٪) and absent STR in ١٩ patients (٧٠.٤٪) vs ٢ patients (٢.٧٪) as compared to optimal reperfusion group (P=٠.٠٠١) tab ٣

Table ٣: Comparison between optimal and suboptimal reperfusion groups regarding ECG data

	Optimal reperfusion (n= ٧٣)	*Suboptimal reperfusion* (n=٢٧)	P value
Site of acute STEMI			
Anteroseptal n (%)	١٣ (١٧.٨ %)	٤ (١٤.٨ %)	
Anterior n (%)	١٦ (٢١.٩ %)	٩ (٣٣.٣ %)	
Extensive anterior n (%)	٨ (١١.٠ %)	٥ (١٨.٥ %)	٠.٤٧٧
Inferior n (%)	١٥ (٢٠.٥٪)	٥ (١٨.٥ %)	
Inferior + lateral n (%)	٩ (١٢.٣ %)	٣ (١١.١ %)	
Inferior + RV n (%)	١٢ (١٦.٤٪)	١ (٣. ٧ %)	
ST segment resolution			٠.٠٠١
Complete n (%)	٥٩ (٨٠.٨٪)	٤ (١٤.٨ %)	
Partial n (%)	١٢ (١٦.٤٪)	٤ (١٤.٨ %)	
Absent n (%)	٢ (٢.٧ %)	١٩ (٧٠.٤ %)	

Angiographic data: There was no statistically significant difference between both groups regarding infarction related artery, site of the occlusion at IRA, and type of the occlusion (P= ٠.١٩٦, P= ٠.٣٧٣ and P=٠.٣١١ respectively) tab ٤.

Table ٤: Comparison between patients of optimal and suboptimal reperfusion groups regarding IRA, site of the lesion and type of the occlusion

	Optimal reperfusion (n= ٧٣)	*Suboptimal reperfusion* (n=٢٧)	P value
Infarction related artery (IRA)			٠.١٩٦
LAD n (%)	٣٧ (٥٠.٧٪)	١٩ (٧٠.٤ %)	
LCX n (%)	٧ (٩.٦ %)	١ (٣.٧ %)	
RCA n (%)	٢٩ (٣٩.٧٪)	٧ (٢٥.٩٪)	
Target lesion location			٠.٣٧٣
Proximal n (%)	٣٤ (٤٦.٦٪)	١٤ (٥١.٩٪)	
Mid n (%)	٣٤ (٤٦.٦%)	١٣ (٤٨.١٪)	
Distal n (%)	٥ (٦.٨ %)	٠ (٠%)	
Type of occlusion			٠.٣١١
Total cut-off n (%)	٣٢ (٤٣.٨ %)	١٢(٤٤.٤٪)	
Total tapered n (%)	٢٨ (٣٨.٤٪)	١١(٤٠.٧٪)	
Subtotal concentric n (%)	١٢ (١٦.٤٪)	٢(٧.٤ %)	
Subtotal eccentric n (%)	١(١.٤٪)	٢(٧.٤٪)	

Suboptimal reperfusion group had statistically significant higher thrombus burden as ١٢ (٤٤.٤٪) vs ١٣(١٧.٨٪) had high thrombus burden, ٩(٣٣.٣٪) vs ١٩(٢٦٪) had moderate thrombus burden, ٥(١٨.٥٪) vs ٢٧ (٣٧٪) had low thrombus burden and ١(٣.٧٪) vs ١٤(١٩.٢%) had no thrombus detected angiogra-phically as compared to optimal reperfusion group **P = ٠.٠١١**. ***fig ١.***

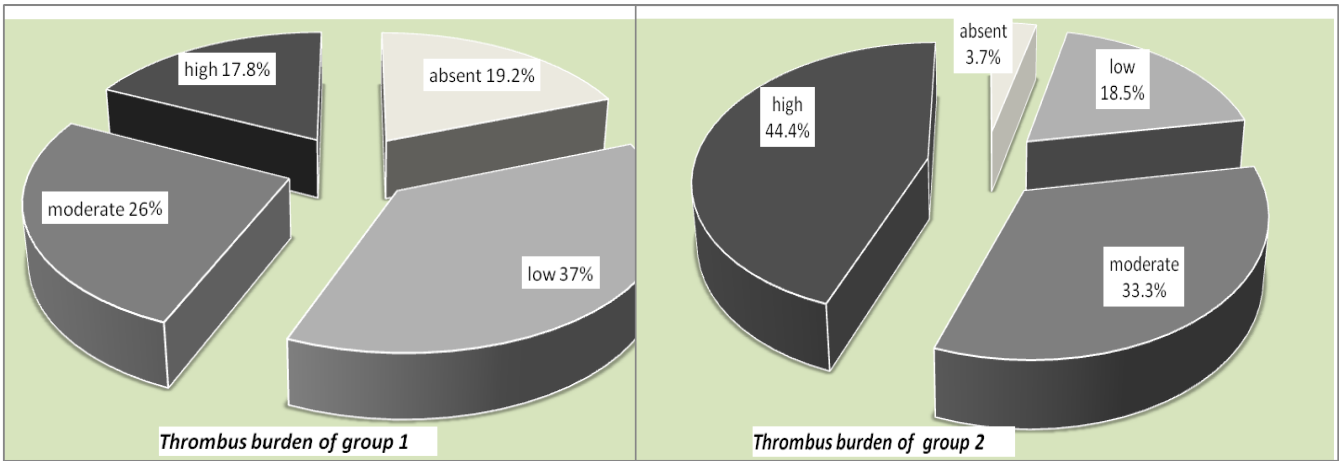


Fig. ١: Comparison between optimal and suboptimal reperfusion groups regarding thrombus burden

Procedural data:

Suboptimal reperfusion group had statistically significant longer reperfusion time (٦.١±٢.٨ vs ٤.٣±٢.١ h), increased predilatation maximum balloon inflation pressure (١٧±٢ vs ١٣±٢ ATM), more repeated balloon inflations (> twice) during predilatation ٢٣(٨٨.٥٪) vs ٣٠ (٤٨.٤٪) as compared to optimal reperfusion group P=٠.٠٠١, P=٠.٠٠١ and P=٠.٠٢ respectively). While there was no statistically significant difference between both groups regarding predilatation, length of balloon, size of balloon, stent length, stent size, maximum deployment pressure and post-dilatation (P > ٠.٠٥ for all). tab ٥ .

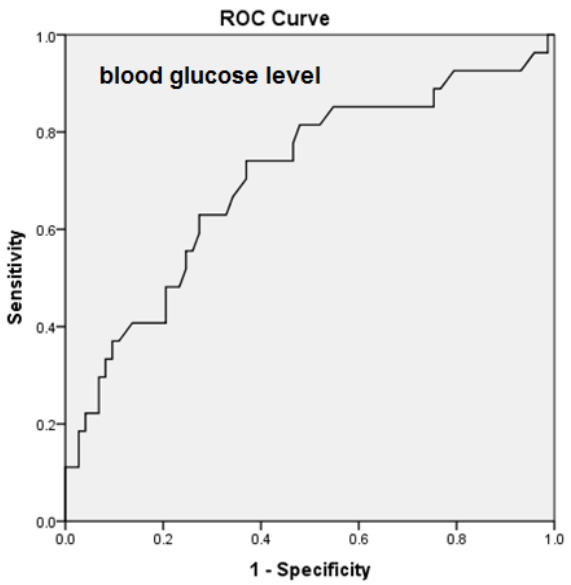
Table ٥: Comparison between optimal and suboptimal reperfusion groups regarding procedural data

	Optimal reperfusion (n= ٧٣)	*Suboptimal reperfusion (n= ٢٧)*	P value
Reperfusion time (hours)	٤.٣ ±٢.١	٦.١ ±٢.٨	٠.٠٠١
Predilatation	٦٥ (٨٩٪)	٢٦ (٩٦.٦٪)	٠.٢٤٢
Length of balloon (mm)	٢٠ ± ٢	٢٠ ± ٢	٠.٢٢٧
Size of balloon (mm)	٢.٤ ± ٠.٥	٢.٨ ± ١.٩	٠.١٥٣
Maximum pressure (ATM)	١٤.٧ ± ١.٨	١٦.١ ± ٢.٣	٠.٠٠٢
Repeated dilatation (> twice)	٤٦ (٦٩.٧%)	٢٤ (٩٢.٣ %)	٠.٠٢٢
Stenting	٦٨ (٩٣.٢٪)	٢٣ (٨٥.٢٪	٠.٢١٧
Length of stents (mm)	٢٩ ± ٩	٣٠ ±١١	٠.٥٣٢
Size of stents (mm)	٣.٢ ±٠.٤	٣.٢ ±٠.٣	٠.٧٢٣
Maximum deployment pressure (ATM)	١٥.٨ ± ١.٨	١٥.٨±١.٩	٠.٩١٤
Post-dilatation	١٩ (٢٨.٤٪)	١١ (٤٧.٨٪)	٠.٠٨٧

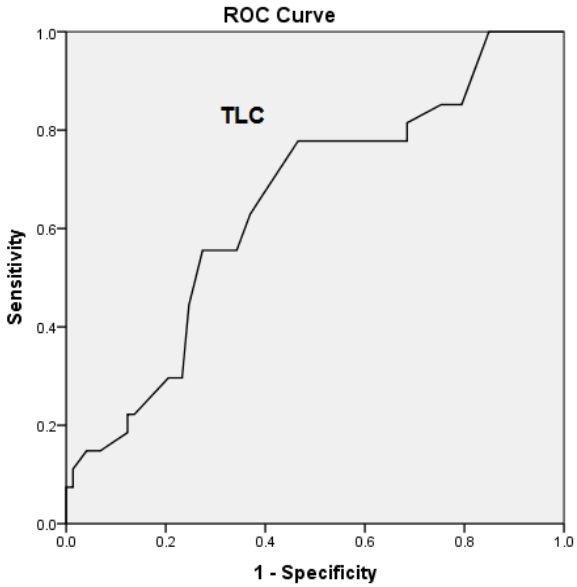
Blood glucose level on admission had ٧٠٪ accuracy, TLC on admission had ٦٥٪ accuracy, maximum predilatation pressure had ٦٩٪ accuracy and reperfusion time had ٧٠ % accuracy as a predictors for suboptimal reperfusion. fig٢.

Fig ٢:

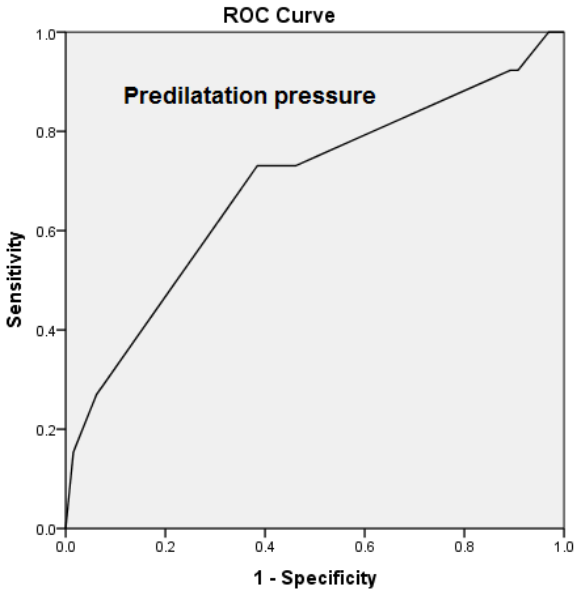
Roc curve of blood glucose level



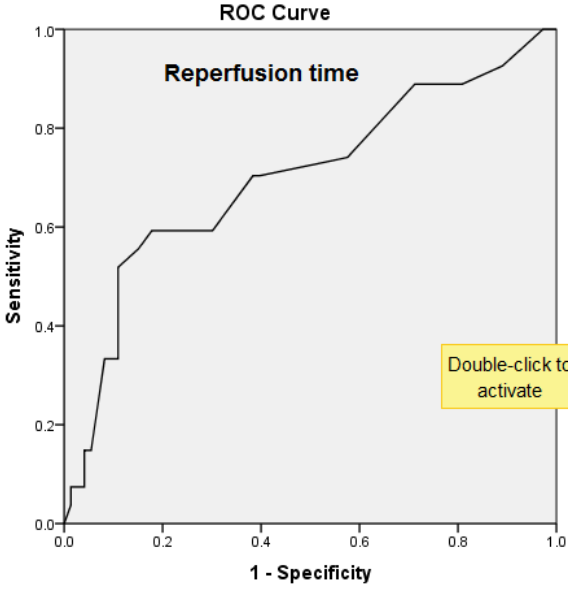
Roc curve of TLC



Roc curve of reperfusion time



Roc curve of Predilatation pressure



Cut off values for continuous variables :

Suboptimal reperfusion group had statistically significant more patients with blood glucose level on admission > ١٦٠ mg/dl n= ٢٠(٧٤.١٪) vs n=٢٩(٣٩.٧٪) compared with optimal reperfusion group, P= ٠.٠٠٢. This cut point had sensitivity and specificity =٧٤% & ٦٢٪ respectively. Also Suboptimal reperfusion group had statistically significant more patients with TLC > ١٠ (10^3/mm^3) n= ٢١(٧٧.٨ %) vs n=٣٤ (٤٦.٦%) compared with optimal reperfusion group, P= ٠.٠٠٥. This cut point had sensitivity and specificity = ٦٣% & ٦٣٪ respectively. Also suboptimal reperfusion group had statistically significant more patients with reperfusion time > ٤ hours n= ١٩(٧٠.٤٪) vs ٢٩(٣٩.٧٪) compared with optimal reperfusion group, P= ٠.٠٠٦. This cut point had sensitivity and specificity =٧٠% & ٦١٪ respectively and suboptimal reperfusion group had statistically significant more patients with maximum predilatation pressure >١٥ ATM n=١٩ (٧٣.١٪) vs n= ٣٠ (٤٦.٢٪) compared with optimal reperfusion group, P= ٠.٠٢٠. This cut point had sensitivity and specificity = ٧٣% & ٦٢٪ respectively. Table (٦)

Table ٦: Cut off values for blood glucose level, TLC, reperfusion time and predilatation pressure

	Sensitivity	Specificity	Optimal reperfusion (n=٧٣)	Suboptimal reperfusion (n=٢٧)	P value
Blood glucose >١٦٠	٧٤%	٦٢%	٢٩ (٣٩.٧٪)	٢٠ (٧٤.١٪)	٠.٠٠٢
TLC on admission > ١٠	٦٣%	٦٣%	٣٤ (٤٦.٦ %)	٢١ (٧٧.٨٪)	٠.٠٠٥
Reperfusion time > ٤ h	٧٠٪	٦١٪	٢٩ (٣٩.٧٪)	١٩ (٧٠.٤٪)	٠.٠٠٦
Predilatation pressure>١٥ *ATM*	٧٣٪	٦٢٪	٣٠ (٤٦.٢٪)	١٩ (٧٣.١٪)	٠.٠٢٠

The predictors of suboptimal reperfusion were arranged according to their power which was determined according to its odd ratio (OR) as follow: Repeated balloon inflation during predilatation (OR= ٨.٢), high thrombus burden at basic angiography (OR= ٥.٠١), no history of angina prior to MI (OR= ٤.٣٨), TLC on admission > ١٠ ($١٠^{٣}/mm^{٣}$) (OR=٣.٤١), Blood glucose level on admission > ١٦٠ mg/ dl (OR= ٣.٠٤), longer reperfusion time (>٤h) (OR= ٢.٦٨), no current ASA therapy prior to MI (OR=٢.٢٩) and lastly balloon inflation at high pressure during predilatation (OR= ٢.٢٣). Tab ٧

Table ٧: Predictors of suboptimal reperfusion according to its odd ratio :

	Predictors	Odd ratio
١	**Repeated balloon inflation during predilatation**	٨.٢٠
٢	**High thrombus burden**	٥.٠١
٣	**No history of angina prior to MI**	٤.٣٨
٤	**TLC on admission > ١٠ *($١٠^{٣}/mm^{٣}$)***	٣.٤١
٥	**Blood glucose level on admission > ١٦٠ mg/ dl**	٣.٠٤
٦	**Longer reperfusion time (٤h)**	٢.٦٨
٧	**No current ASA therapy**	٢.٢٩
٨	**Predilatation at pressure >١٥ ATM**	٢.٢٣

Aspiration device was used in ١٢ patients according to operator's discretion, ١٠ (٨٣٪) of them had final MBG = ٣ (١٤٪ of optimal reperfusion group) and ٢ (١٧٪) had final MBG ≤ ٢ (٧.٤٪ of suboptimal reperfusion group).

Relation between final MBG and final TIMI flow grade: TIMI grade ٣ was achieved in ٨٤ patients, ١١ patients of them (١٤٪) had suboptimal reperfusion (MBG≤٢) and ٧٣ patients had optimal reperfusion (MBG = ٣). Agreement between final TIMI flow and final MBG was ٨٩٪.

		Final MBG	
		٣	≤ ٢
Final TIMI	***III***	٧٣	١١
	≤ II	٠	١٦

Short term Clinical outcome :

In hospital mortality and major cardiac events (MACE): Suboptimal reperfusion group compared with optimal reperfusion group had statistically significant higher in-hospital total MACE (death, re-infarction, HF and stroke) ١٠(٣٧٪) vs ٦(٨.٢٪), P=٠.٠٠٣.

Duration of hospital stay: Suboptimal reperfusion group com-pared with optimal reperfusion group had statistically significant longer duration of hospital stay (٨±٩ vs ٥ ±٢ days), P=٠.٠٢. table ٨

Table ٨: Comparison between optimal and suboptimal groups regarding short term clinical outcome

	Optimal reperfusion (n=٧٣)	Suboptimal reperfusion (n=٢٧)	P value
Total MACE			٠.٠٠١
Mortality	١ (١.٤ %)	٣ (١١.١ %)	
HF and shock	٤ (٥.٥ %)	٥ (١٨.٥ %)	
Reinfarction	٠ (١.٤ %)	٢ (٧.٤ %)	
Stroke	١ (١.٤ %)	٠ (٠ %)	

Echocardiographic data:

Echocardiography was avail-able in ٩٩ patients, as one patient died before echocardiography was done. Suboptimal reperfusion group had statistically significant lower mean Ejection fraction (٤٥±٥ vs ٥٥±٧٪) and higher wall motion score index (١.٣±٠.٢ vs ١.٢±٠.٢) com-pared with optimal reperfusion group, P= ٠.٠٠١ and P = ٠.٠٠٤ respectively.

Table ٩: Comparison between optimal and suboptimal groups regarding details of Echocardiography

	Optimal reperfusion (n=٧٣)	Suboptimal reperfusion (n= ٢٦)	P value
LV EF % (mean ± SD)	٥٥.٤ ±٧.٦	٤٥.٧ ±٥.٤	٠.٠٠١
WMSI (mean ± SD)	١.٢٣ ±٠.٢١	١.٣٧ ±٠.١٩	٠.٠٠٤

Case ١ (From group ١):

MR MAR ٤٣Ys old, smoker, no other risk factors of CAD. He is known to have IHD with previous PCI to LAD ٢ years ago, he had history of angina prior to MI and current aspirin therapy. Examination on admission: sinus bradycardia with HR ٥٠bpm, BP ٧٠/٥٠, killip class ٤. ECG showed Acute Inferior MI with lateral Ischemia (ST elevation at leads II, III, and AVF). Basic coronary Angiography Revealed patent LAD stent and cut off totally occluded proximal RCA, mild thrombus bur-den. ١ry PCI for RCA with reperfusion time ٩٠ minutes, flow restoration was achieved by passage of non-inflated balloon without predilatation, followed by deployment of the stent from ٠ up to ١٨ ATM. Successful restoration of flow [achieved TIMI III flow, Myocardial Blush grade ٣]. post-procedure HR ٨٥bpm, BP ١١٠/ ٧٠ mmHg, STR =١٠٠٪, no in-hospital MACE and LVEF was ٦٠٪. [Fig ٢].

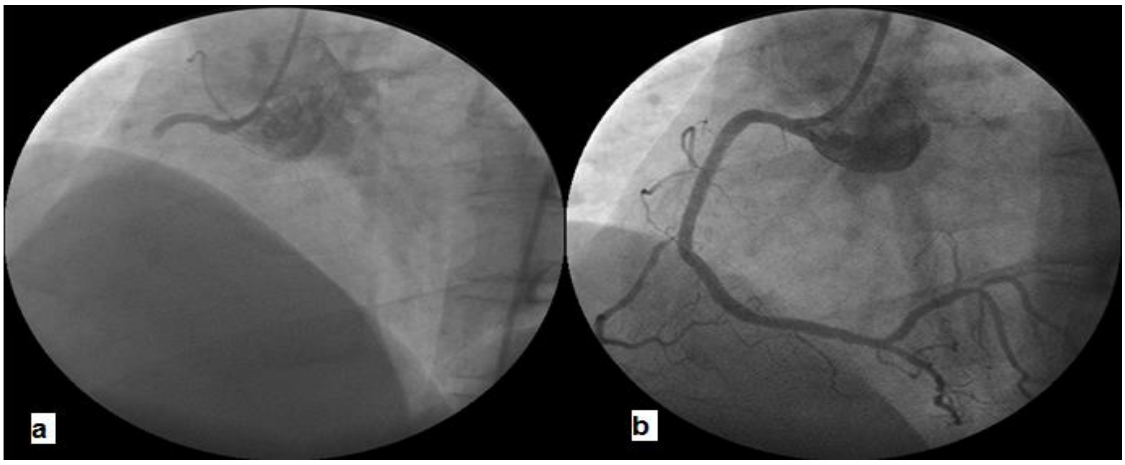


Fig. (٢): Coronary angiogram of patient from Group I, a- RCA has proximal cut off total occlusion. b- RCA shows final TIMI ٣ flow and final MBG ٣.

Case ٢ (From group٢):
Mr. MGM ٥٩ years old, he was not known as ischemic patient, no history of angina prior to MI, no current aspirin therapy. Examination on admission revealed HR ٧٠ bpm & BP= ١١٠/٧٠ mmHg, Killip Class ١. ECG showed acute anterior MI (ST elevation at V١-V٦). BS and TLC on admission = ١٨٩mg/dl and ١١ ١٠^{٣}/ul respectively. Coronary Angiography Revealed mid LAD tapered total occlusion. ١^{ry} PCI for LAD with repeated balloon inflation during predilatation from ٠ up to ١٨ ATM, stent was deployed up to ٢٠ ATM. after stent TIMI grade ١ achieved so intracoronary isoptin was gives with partial improvement, final TIMI=II and final MBG = ٠.STR =٣٠٪, in-hospital Heart failure, LVEF was ٤٠٪ [Fig. ٣].

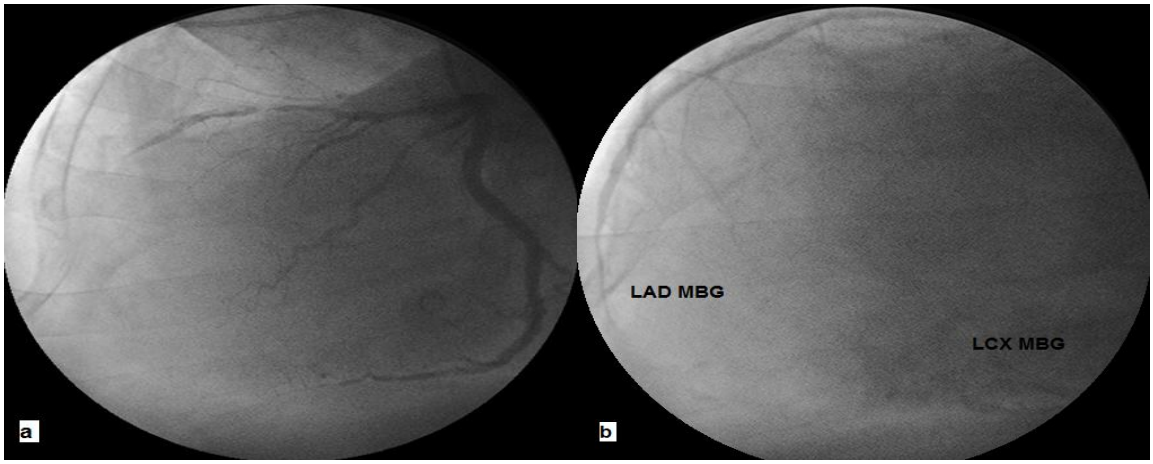


Figure (٣) Coronary angiogram of patient from Group ٢,
a- LAD has mid total occlusion. b- LAD shows final TIMI II flow and final MBG ٠

Discussion

Suboptimal myocardial reperfusion during primary PCI for STEMI patients is considered to be a negative hallmark predicting the occurence of worse clinical outcome. Therefore, attention has shifted from epicardial artery patency to the status of the coronary microvascular and myocardial reperfusion[٦]. Our study revealed that incidence of suboptimal myocardial perfusion was ٢٧٪. And predictors for suboptimal reperfusion arranged according to their odd ratio were repeated balloon inflation during predilatation, high thrombus burden, no history of angina prior to MI, high TLC, high blood glucose level, longer reperfusion time, no current aspirin intake before MI and high predilatation pressure.

Our study revealed that the incidence of MBG ≤٢ was ٢٧٪ and in spite of TIMI grade ٣ was achieved in ٨٤ patients, ١١ patients of them (١٤٪) had suboptimal reperfusion (MBG≤٢). In agreement with our results are those of Marlos et al., ٢٠١٢, who evaluated ٩٩ primary PCI procedures, they found that TIMI ٣ flow was restored at procedure's end in ٩١ patients however MBG=٣ was achieved in only ٦٩ patients (٦٩.٧٪) and incidence of MBG ≤٢ was ٣٠.٣٪.[٢] Gjin et al., ٢٠١٠ studied ١٢١٣ patients with STEMI and they found that ٣٤ % patients with TIMI flow grade ٣ at the end of primary PCI had MPG ≤٢[٧].

Prodromal angina pectoris (AP) has a cardioprotective effect by the mechanism of ischemic preconditioning**,** which delays infarct progression during the early hours after the onset of AMI and extends the window of time for reperfusion therapy[٨]. Prodromal AP had been associated with smaller infarct size, improved left ventricular function and favorable short- and long-term prognoses after AMI[٩,١٠]. Our study revealed that suboptimal reperfusion group compared with optimal reperfusion group had statistically significant fewer patients with history of angina prior to MI ٥(١٨.٥٪) vs ٤٤(٦٠.٣٪), (P=٠.٠٠١). In agreement with our results are those of Koichi et al., ٢٠١١ found that pre-infarction angina occurred ٢٤ to ٧٢ hours before infarction was the most strongly associated with reductions in the rates of microvascular obstruction (MVO) and myocardial hemorrhage assessed by cardiac MRI (p=٠.٠٢١),[١١]. Also ZHANG et al., ٢٠١٢ found that prodromal angina (PA) associated with higher rates of overall procedural and of complete ST-segment resolution at ٩٠ minutes after the procedure (P=٠.٠٠١)[١٢].

Platelet reactivity on admission, as assessed by the Platelet Function Analyzer–١٠٠ is associated with the prevalence of no-reflow and adverse remodeling[١٣]. Platelets may be implicated in no-reflow through several mechanisms: micro-vascular obstruction by platelet aggregates and release of platelet-derived vasoactive and chemotactic mediators. patients with angio-graphic no-reflow had higher levels of TXA٢ compared with patients without angiographic no-reflow, independently of the Abciximab treatment[١٤]. Aspirin induces the irreversible inhibition of platelet cyclo-oxygenase ١, blocks thromboxane A٢ production, and is a mainstay of antiplatelet therapy, several reports in the thrombolytic era have already suggested the correlation between previous aspirin treatment and smaller infarct[١٥]. Our study revealed that suboptimal reperfusion group com-pared with optimal reperfusion group had statistically significant fewer patients with current aspirin therapy ٦ (٢٢.٢٪) vs ٣٨ (٥٢٪), (P=٠.٠٠٨).

Acute hyperglycemia is a predictor of short-term mortality after AMI, but diabetes is still an important determinant of long-term outcomes in patients with AMI[١٦, ١٧]. Stress hyperglycemia in a setting of AMI increases the risk of malignant ventricular tachyarrhythmias as well as in-hospital mortality[١٨].

Acute hyperglycemia causes several unfavorable effects that contribute to the poor outcomes of patients with AMI: Oxidative stress, inflamemation, apoptosis, endothelial dysfunction, hypercoagulability, platelet hyperactivity, impaired ischemic preconditioning, and impaired microcirculation which directly damage the ischemic myocardium and may cause the no-reflow phenomenon during reperfusion[١٩]. Our result revealed that suboptimal reperfusion group compared with optimal reperfusion group had statistically significant increased blood glucose level on admission (٢٤٠±١٠١ mg/dl vs ١٧١±٧٢ mg/dl) P=٠.٠٠١ and more patient with blood glucose level > ١٦٠ mg/dl ٢٠(٧٤.١) vs ٢٩(٣٩.٧), P= ٠.٠٠٢. In agreement with our results are those of Iwakura

et al., ٢٠٠٣, who found that the no-reflow phenomenon was found on myocardial contrast echo (MCE) in ٣٣.٦٪ of the patients, and associated with higher mean glucose level on admission than that of patients who did not exhibit this phenomenon (٢٠٩±٧٩ vs. ١٥٩±٥٦ mg/dl, P =٠.٠٠٠١). and the no-reflow phenomenon was more often observed in the patients with hyperglycemia (defined as BS >١٦٠ mg/dl) ٥٢٪ vs. ١٤٪ compared to patients without hyperglycemia, (P =٠.٠٠٠١). They also found that There was no difference in glycosylated hemoglobin or in the incidence of diabetes mellitus between the two subsets[٢٠].

The baseline leucocytic count had been shown to independently predict short-term and long-term risk for ischemic events and death in patients presenting with ACS, including STEMI[٢١]. The suggested pathologic mechanisms involving neutronnphils were microvascular plugging, spasm, endothelial swelling, and inflammatory response by pro-inflammatory cytokines release, all of which eventually lead to impairment of microvascular perfusion during acute myocardial ischemia[٢٢]. Our study revealed that suboptimal reperfusion group compared to optimal reperfusion group had statistically significant increased TLC on admission (١٢.١± ٣.٦ vs ١٠.٢±٣.٣) $١٠^{٣}$ /$mm^{٣}$, P= ٠.٠٢٠. and suboptimal reperfusion group had more patients with TLC >١٠ $١٠^{٣}$ /$mm^{٣}$ on admission ٢١(٧٧.٨٪) vs ٣٤ (٤٦.٦), P= ٠.٠٠٥. In agreement with our results are those of Matteo et al., ٢٠٠٦, who studied ٢٣٨ consecutive acute STEMI patients treated with successful primary PCI. They found that MBG ٢–٣ was related to lower total WBC, monocytes and neutrophils (P=٠.٠٠١),[٢٣].

It is well established that prolonged ischemia leads to edema of distal capillary beds, swelling of myocardial cells, neutrophil plugging and alterations of capillary integrity. Furthermore, delayed reperfusion can result in an older, more organized intracoronary thrombus, which may increase the risk of distal embolization during primary PCI and reduce the likelihood of achieving myocardial perfusion[٢٤]. Our result revealed that suboptimal reperfusion group compared to optimal reperfusion group had statistically significant longer reperfusion time (٦.١±٢.٨ vs ٤.٣±٢.١ h), P=٠.٠٠١, and more patients with reperfusion time >٤h ١٩(٧٠.٤) vs ٢٩ (٣٩.٧), P= ٠.٠٠٦. In agreement with our results are those of Gjin et al., ٢٠١٠, who studied ١٢١٣ patients with STEMI. They found that suboptimal reperfusion group compared to optimal perfusion group had statistically significant longer reperfusion time (٥ vs ٣.٨ hours), P=٠.٠٠١[٧]. Also in agreement with our results are of Cevat et al., ٢٠٠٨, who studied ٣٨٢ consecutive acute STEMI patients treated with primary PCI, they found that group of suboptimal reperfusion had statistically significant more patients with reperfusion time (≥٤h) (٤٨.٤٪) compared with optimal reperfusion group (٢٦.٣٪), P= ٠.٠٠٤[٢٦]

STEMI is characterized by thrombus formation which lead to occlusion of the infarct related artery (IRA). Angiographic evidence of thrombus in the IRA is associated with poor in-hospital outcomes, increased infarct size and higher long term mortality.[٢٥] While attempting to open the occlusion during PCI, it is more likely to dislodge the thrombus down-stream causing distal embolization and microvascular obstruction and contribute to "no reflow phenomenon"[٢٥]. our study revealed that Suboptimal reperfusion group compared with optimal reperfusion group had statistically significant higher thrombus burden. In agreement with our results are those of Cevat et al., ٢٠٠٨ studied ٣٨٢ consecutive STEMI patients treated with primary PCI, they classified angiographic thrombus burden into mild, moderate and high. They found that no-reflow group had statistically significant higher thrombus burden, P = ٠.٠١[٢٦]. Also in agreement with our results are those of Xiaofan et al., ٢٠١١, who analyzed clinical, angiographic, and IVUS data from ٣٦٤ patients enrolled in the randomized HORIZONS-AMI trial. They found that no-reflow group had statistically significant more angiographic thrombus compared with reflow group, P=٠.٠٤٣[٢٧]. while in dis-agreement with our results are those of Marlos et al., ٢٠١٢, who retrospectively evaluated ٩٩ STEMI pati-ents treated with primary PCI procedures, They found that there was no statistically significant difference between reflow and no-reflow groups as regard to presence of angiographic thrombus, P=٠.٧٤. This controversy can be explained by the fact that Marlos et al., ٢٠١٢ compared between the two groups according to the

presence of angiographic thrombus but not according to burden of the thrombus[١].

Macro-embolisation occurred in ١٤٪ of patients with STEMI treated with primary PCI and associated with worse TIMI flow in the infarct-related artery, worse myocardial reperfusion, and less complete ST-segment resolution, larger infarct size, worse left ventricular function and higher mortality. Also distal micro-embolisation with primary PCI may contribute to poor myocardial reperfusion[٢٥]. This has stimulated great interest in attempts to remove thrombus, prevent distal micro-embolisation. TAPAS study indicated that thrombus aspiration during primary PCI is associated with an improvement in survival at ١ year[٢٥]. In our study Aspiration device was used during the procedure of ١٢ patients according to operator's discretion, ١٠ patients (٨٣٪) of them had optimal reperfusion (MBG = ٣) and ٢ patients (١٧٪) had suboptimsl reper-fusion (MBG ≤ ٢). In agreement to our result are those of Tone et al., ٢٠٠٨ who randomized ١٠٧١ STEMI patients to aspiration thrombectomy followed by stenting versus stenting alone. They found that final MBG ٠ or ١ occurred in ١٧.١٪ in the thrombus-aspiration group and in ٢٦.٣٪ in the conventional-PCI group (P<٠.٠٠١) and complete STR was significantly higher with aspiration thromb-ectomy ٥٦.٦٪ vs ٤٤.٢٪ in the conventional-PCI group, (P<٠.٠٠١),[٢٨]. Sardella et al., ٢٠٠٩, and Dudek et al., ٢٠١٠ found that aspiration during primary PCI associated with more final MBG and STR[٢٩,٣٠]. Also, Diana et al., ٢٠١١, who conducted meta-analysis included ١٧ trials evaluated catheter aspiration devices. They found that Catheter aspiration devices also significantly increased the achievement of ST-segment resolution and MBG-٣, while reducing the risk of distal embolization and no reflow[٣١].

It has been hypothesized that predilatation of unstable plaques in the context of unstable angina or myocardial infarction may facilitate embolisation of cholesterol and necrotic debris, also local activation of platelets and coagulation factors, liberation of clot-bound-thrombin due to mechanical disruption of thrombus which increase the likelihood of suboptimal reper-fusion[٣٢]. Also, predilatation can activate leuko-cytes and oxidative stress, aggravating the inflammatory response and myocardial reper-fusion injury.[٢١] Our study revealed that suboptimal reperfusion group compared to optimal reperfusion group had statistically significant higher predilatation maximum balloon inflation pressure (١٦.١±٢.٣ vs ١٤.٧±١.٨ ATM), more repeated balloon inflation (> twice) during predilatation ٢٤ (٩٢.٣%) vs ٤٦(٦٩.٧%) and more patient with predilatation pressure > ١٥ ATM, P = ٠.٠٠٢, P=٠.٠٢٢ and P=٠.٠٢. respectively. In agreement with our results are those of Marlos et al., ٢٠١٢, who found that suboptimal reperfusion group compared to optimal reperfusion group had statistically significant increased maximum dilation pressure (١٥.٥±٣.٩ vs ١٣.٥±٤.٢ ATM), P= ٠.٠٣٩.[٢]

Study limitations: Small number of patient who underwent aspiration due to availability and cost, and usage of one type only of aspiration device (diver C).

Conclusion

Based on the results of this study it can be concluded that repeated balloon inflation during predilatation, high thrombus burden, no history of angina prior to MI, high TLC, high blood glucose level, longer reperfusion time, no current aspirin therapy and high predilatation pressure were predictors for suboptimal reperfusion.

Clinical implications Prevention rather than treatment should be the way forward as treatment after no-reflow (suboptimal reper-fusion) is established is unlikely to succeed so an efficient emergency system that guarantees the reduction in reperfusion time, careful history focused on angina prior to MI, aspirin therapy as well as the evaluation of laboratory data such as glucose level and TLC, and thrombus burden at basic angiography to determine patients at a risk for suboptimal reperfusion to take a measures to prevent its incidence. Lastly during primary PCI if possible avoidance of repeated predilatation and also avoidance of predilatation high pressure

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