BENHA MEDICAL JOURNAL bmj
Print ISSN 1110-208X.
Online ISSN 2357-0016

Original article

No-Reflow after Stenting in Left Anterior Descending (LAD) versus Non-LAD Culprit in STEMI Patients, Angiographic Finding and Short Term Outcomes

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Abstract:

Background: No-reflow is a serious complication defined as thrombolysis in myocardial infarction (TIMI) flow grade of <3 with patent coronary artery and absence of dissection or spasm. This study aimed to investigate the angiographic results and clinical outcomes after no-reflow in the left anterior descending artery (LAD) versus non-LAD artery ST-elevation myocardial infarction. **Methods:** This study was a dual center, prospective observational study that was conducted in cardiology department of both Benha University Hospital and Mataria Teaching Hospital. This study included 100 STEMI patients who underwent primary PCI with TIMI flow <3 post stenting. Patients were divided into 2 groups: Group I: patients with LAD culprit (50 patient) & Group II: patients with non-LAD culprit (50 patient). Cases were subjected to: Detailed history, clinical examination, routine laboratory investigation and transthoracic Echocardiography, then were followed for in-hospital and 30-days major adverse cardiovascular events. **Results:** number of patients receiving nitrate and adrenaline were significantly higher in group I than group II . Patients of group I had statistically lower incidence of final TIMI flow 3. After 30 days follow up; patients of group I had statistically significant lower mean LVEF and higher incidence of heart failure than group II. No significant difference between two groups as regard to stroke, re-infarction and mortality. **Conclusion:** We detected that no-reflow in LAD group was more refractory in nature with less final TIMI flow and higher incidence of heart failure than non-LAD group.
Keywords: No-reflow, STEMI, LAD culprit, non-LAD culprit.

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Received:
Accepted:

Introduction

Primary percutaneous coronary intervention (PPCI) is the gold standard of treatment of ST segment elevation myocardial infarction (STEMI). However, there remain a small proportion of patients, who continue to exhibit overt impairment of myocardial reperfusion despite successful opening of infarct related epicardial artery (IRA). [(1)]

No-reflow is defined as thrombolysis in myocardial infarction (TIMI) flow grade of <3 with patent coronary artery with an absence of dissection or spasm. It is a serious complication and accounts for 11%−41% of cases of ST-elevation myocardial infarction (STEMI) during primary percutaneous coronary intervention (PCI) [(2)].

Nair et al. have reported a significant association of anterior wall myocardial infarction (AWMI) with the development of no-reflow [(3)]. Other studies have shown that left anterior descending artery (LAD)-related STEMI leads to significantly lower post-myocardial infarction (MI) left ventricular ejection fraction (LVEF) compared with non-LAD-related MI[(4)].

Numerous clinical and angiographic factors have been shown to be associated with no-reflow, including advanced age, a reperfusion time > 6 h, Killip Class ⩾ 3, long lesion length, high thrombus burden (grade ⩾ 3), a high admission glucose to estimated average glucose ratio and PRECISE-DAPT score. Moreover, there is evidence of a correlation between no-reflow and reduced left ventricular function, worse clinical outcome and higher mortality [(1)].

The purpose of this study was to investigate the angiographic results and short-term clinical outcomes after no-reflow in the left anterior descending artery (LAD) versus non-left anterior descending artery ST-elevation myocardial infarction.

Patients and methods

This study was a dual center, prospective observational study that was conducted in the CCU unit, cardiology department of both Benha University Hospital and Mataria Teaching Hospital within one year from March 2023 to March 2024.

An informed written consent was obtained from the patients. Every patient received an explanation of the purpose of the study and had a secret code number. The study was done after being approved by the Research Ethics Committee, Faculty of Medicine, Benha University and Mataria Teaching hospital.

Inclusion criteria were Patients aged ⩾18 years, patients diagnosed with STEMI which is defined as persistent ST-segment elevation at the J point in 2 contiguous leads with the following cutoff points: >0.1 mV in all leads other than leads V2 to V3.

For leads V2 to V3, the following cutoff points apply: ≥0.2 mV in men aged ≥40 years, ≥0.25 mV in men aged <40 years, or ≥0.15 mV in women [(5)]**.** Undergoing primary PCI, developed no-reflow (TIMI flow grade<3 post stenting).

Exclusion criteria were patients with cardiogenic shock at the time of presentation, patients with valvular or congenital heart disease, patients with cardiomyopathy, myocarditis, pericarditis, contraindication to the use of antiplatelet or anticoagulant, complicated cases like coronary dissection, perforation or spasm and patients who refused to enroll in the study.

Grouping: Patients were selected and divided into two groups according to culprit vessel: Group I: patients with LAD culprit vessel (50 patient). Group II: patients with non-LAD culprit vessel (50 patient).

All studied cases were subjected to the following: Detailed history

taking, including [age, gender, and history of diabetes mellitus which is diagnosed by FBG ⩾126 mg/dl or 2h PPBG ⩾ 200 mg/dl or classic DM symptoms with RBG > 200 mg/dl or HbA1c >6.5% [16] , hypertension which is diagnosed by SBP ⩾140 mmHg and\or DBP ⩾90 mmHg [17], hyperlipidemia, smoking, and family history of premature CAD, previous CABG, previous ischemic stroke or TIA].

Full clinical examination, including [heart rate, systolic blood pressure, diastolic blood pressure, and signs of heart failure general & local examination and killip class]. **Routine laboratory investigations [**Complete blood count (CBC), total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides, and renal function tests].

12 leads resting ECG:

To confirm diagnosis of STEMI and its site and to exclude significant arrhythmia. STEMI diagnosis is defined when there is persistent ST-segment elevation at the J point in 2 contiguous leads or more with the following cutoff points: >0.1 mV in all leads other than leads V_2 to V_3. Moreover, for leads V_2 to V_3, the following cutoff points apply: ≥0.2 mV in men aged ≥40 years, ≥0.25 mV in men aged <40 years, or ≥0.15 mV in women. [5]

2D conventional Echocardiography:

Echo-Doppler exam was performed to all patients within 48 hours of admission and 30 days after discharge using Philips Epiq 7 machine with 5s-1 probe with simultaneous ECG signal. Patients were examined in left lateral decubitus using accessible window to parasternal long axis and apical views.

The following echo-Doppler parameters were obtained according to the American society of cardiology:

- 2-dimensional echocardiography to obtain ejection fraction (EF) using modified Simpson method (biplane method of discs), which is recommended by American society of echocardiography. This method requires the measurement of LVEF by tracing the endocardial border in both the apical four-chamber and two-chamber views in end-systole and end-diastole. These tracings eventually divide the LV cavity into a predetermined number of disks (usually 20) [6].
- Color flow mapping & continuous wave doppler to assess presence of MR jet and its severity.

Primary PCI intervention

The culprit vessel was identified, and reperfusion was achieved with standard PCI techniques. The following parameters were assessed: Number of vessels diseased, the affected culprit vessel, vessel size (diameter), and degree of thrombus burden according to TIMI thrombus grading: (Grade 0: no thrombus, Grade 1: Possible thrombus, Grade 2: the thrombus' greatest dimension is <1/2 vessel diameter, Grade 3: Greatest dimension >1/2 to <2 vessel diameters, Grade 4: Greatest dimension >2 vessel diameters, Grade 5: total vessel occlusion due to thrombus) [7] .

We recorded medication used as heparin, glycoprotein (GP) IIb/IIIa inhibitor & intracoronary medication, like nitrate, adenosine or adrenaline. Also evaluated number of implanted stents, occurrence of pre or post dilatation & aspiration and final TIMI flow by measuring the coronary artery clearance of radiographic dye (TIMI 0 refers to absence of any antegrade flow beyond occlusion, TIMI 1 is a faint antegrade flow with incomplete filling of distal coronary bed while TIMI 2 flow is delayed or sluggish antegrade flow with complete filling of distal

DOI: DOI: 10.21608/bmfj.2025.335923.2251.

territory and TIMI 3 is the normal flow) [8].

Follow up after 30 days:

All included patients were followed up for in-hospital and 30-days Major adverse cardiovascular events including: All-cause mortality, re-infarction, repeat revascularization, cerebrovascular accidents and heart failure.

Approval code: MS 37-4-2023

Statistical analysis:

Statistical analysis was done by SPSS v28 (IBM Inc., Armonk, NY, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing unpaired Student's t- test. Qualitative variables were presented as frequency and percentage (%) and were analyzed utilizing the Chi-square test or Fisher's exact test when appropriate. A two tailed P value < 0.05 was considered statistically significant

Results

There was no statistically significant difference between two groups as regard to age or gender. In addition, no differences were observed between two groups as regard to the risk factors such as DM, HTN, smoking, family history of premature, history of previous IHD or**. (Table 1)**

The mean systolic blood pressures were statistically significant lower and mean heart rate was statistically higher in patients of group I than those of group II (113.8 ± 14.97 mmHg vs. 120.7 ± 14.43 mmHg & 85.06 ± 11.36 beat/min vs. 74.42± 9.51 beat/min; respectively) with p-value <0.05. Patients of group I had lower incidence of Killip class I and higher incidence of class II and III than those of group II [8 patients (16%) vs. 29 patients (58%) & 23 patients (46%) vs. 15 patients (30%) and 19 patients (38%) vs. 6 patients (12%); respectively] with significant p-value at <0.001. **(Table 2**).

Regarding the laboratory investigation, the mean WBC count was statistically significant higher in patients of group I (10.83±3.57 vs. 9.19±3.28 with p value= 0.019). Also, the mean total cholesterol and LDL levels were significantly higher in group I (214.12±21.44 vs. 194.4±16.84 and 123.84±17.65 vs. 106.9±11.73; respectively with p value < 0.001). No significant difference between the 2 groups regarding mean hemoglobin level & platelet count and mean serum creatinine (p value >0.05).

Regarding baseline Echocardiography, the mean LVEF by modified Simpson's method was statistically significant lower in patients of group I (35.76 ± 7.77% vs. 45.48 ± 7.27%). p-value<0.001. but no statistically significant difference was detected between both groups as regards degree of MR. **(Figure 1)**

Regarding angiographic data, there was no significant difference between the two studied groups regarding mean vessel size, number of diseased vessels and degree of thrombus burden**.** The number of patients of group I that received intracoronary nitrate and adrenaline were statistically significantly higher than those of group II {40 patients (80.0%) vs. 24 (48.0%) & 26 (52.0%) vs. 8 (16.0%); respectively, p-value< 0.001. However, there is no significant difference between the 2 groups regarding use of GP IIb/IIIa and intracoronary adenosine. **(Table 3).** Patients of group I had statistically significant lower incidence of final TIMI flow 3 as compared to those of group II {29 (58.0%) vs. 44 (88%), p value= 0.003). **(Figure 2)**

Regarding procedural technique, there were no significant differences among the studied groups regarding number of implanted stents, aspiration, predilation

{30 (60.0%) vs. 37 (74.0%) and post dilatation {10 (20.0%) vs. 6 (12.0%), p value>0.05.

Regarding 30 days follow-up, mean LVEF was statistically significant lower in patients of group I (34.06 ± 7.34% vs. 44.21 ± 8.58%, p-value<0.00)**. (Table 4).** Patients of group I had statistically higher incidence of heart failure {6 patients (12%) vs. 1 patient (2%), p value =0.049, but no significant difference between two groups as regard to incidence of stroke, re-infarction, revascularization and mortality. (**Table 4**).

Table (1): Demographic data and risk factors among the studied groups:

	Groups		**Test of sig.**	**P-Value**
	LAD group(I) (N=50)	**Non-LAD group (II) (N= 50)**		
Age (years)				
Mean± SD	57.44±10.43	57.34±10.15		
Min – Max	28 – 75	37 – 83	0.049	0.961[(a)]
Sex				
Male	38 (76.0%)	41 (82.0%)		
Female	12 (24.0%)	9 (18.0%)	0.461	0.542[(b)]
Family history	8 (16.0%)	14 (28.0%)		
Smoking index	26 (52.0%)	26 (52.0%)		
Diabetes mellitus	21 (42.0%)	19 (38.0%)		
Hypertension	31 (62.0%)	23 (46.0%)		
Previous IHD	9 (18.0%)	12 (24.0%)		
Previous CABG	0 (0%)	0 (0%)		
Previous stroke	4 (8.0%)	5 (10.0%)		

LAD: left anterior descending artery, **N:** number**, (a):** Independent-Sample T Test, **(b):** Chi-Square Test, **IHD:** Ischemic heart disease, **CABG:** coronary artery bypass graft, *****: Statistically significant at p ≤ 0.05, ******: Highly statistically significant at p < 0.001, **P:** P-value between groups

Table (2): Clinical examination among the studied groups:

	Groups		**Test of sig.**	**P-Value**
	LAD group (I) (N=50)	**Non-LAD group (II) (N= 50)**		
Systolic blood pressure(mmHg)				
Mean± SD	113.8±14.97	120.7±14.43		
Min – Max	90 – 150	90 – 150	-2.347	**0.021*[(a)]**
Diastolic blood pressure (mmHg)				
Mean± SD	70.1±11.32	72.9±10.59		
Min – Max	55 - 90	60 – 90	-1.277	0.205[(a)]
Pulse (bpm)				
Mean± SD	85.06±11.36	74.42±9.51		
Min – Max	60 - 110	55 – 100	5.079	**<0.001**[(a)]**
Killip class				
I	8 (16%)	29 (58%)		
II	23 (46%)	15 (30%)	20.363	**<0.001**[(b)]**
III	19 (38%)	6 (12%)		

LAD: left anterior descending artery, **N:** number**, (a):** Independent-Sample T Test**,** *: Statistically significant at p ≤ 0.05, **: Highly statistically significant at p < 0.001, **P:** P-value between group

DOI: DOI: 10.21608/bmfj.2025.335923.2251.

Table (3): Procedural medication among the studied groups:

	Groups		**Test of sig.**	
	LAD group(I) (N=50)	**Non-LAD group (II) (N= 50)**		**P-Value**
Heparin dose				
Mean	10000IU	10000 IU		
Min - Max	10000 - 10000 IU	10000 – 10000 IU		
GP IIb/IIIa inhibitor use				
No	22 (44.0%)	16 (32.0%)		
Yes	28 (56.0%)	34 (68.0%)	1.528	0.216(b)
IC nitrates				
No	10 (20.0%)	26 (52.0%)		
Yes	40 (80.0%)	24 (48.0%)	11.111	**<0.001**(b)**
IC adenosine				
No	42 (84.0%)	41 (82.0%)		
Yes	8 (16.0%)	9 (18.0%)	0.071	0.790(b)
IC adrenaline				
No	24 (48.0%)	42 (84.0%)		
Yes	26 (52.0%)	8 (16.0%)	14.439	**<0.001**(b)**

LAD: left anterior descending artery, **N:** number**, GP:** glycoprotein, **TIMI:** Thrombolysis in Myocardial Infarction, **(b):** Chi-Square Test, *: Statistically significant at $p \leq 0.05$, **: Highly statistically significant at $p < 0.001$, **P:** P-value between groups

Table (4): 30-day follow-up outcomes among the studied groups:

	Groups			**Test of sig.**	**P-Value**
	LAD group(I) (N=50)	**Non-LAD group (II) (N= 50)**			
LVEF after 30 days)					
Mean± SD	34.06 ± 7.34	44.21 ± 8.58			
Min - Max	20 - 50	25 – 60		-6.249	**<0.001**(a)**
Moderate to severe MR after (30 days)					
No	31 (62.0%)	33 (66.0%)			
Yes	19 (38.0%)	17 (34.0%)		0.174	0.677(b)
Mortality					
No	48 (96.0%)	49 (98.0%)			
Yes	2 (4.0%)	1 (2.0%)	0.344	0.558(b)	
Re-infarction					
No	47 (94.0%)	48 (96.0%)			
Yes	3 (6.0%)	2 (4.0%)	0.211	0.646(b)	
Stroke					
No	49 (98.0%)	49 (98.0%)			
Yes	1 (2.0%)	1 (2.0%)	0.001	1.00(b)	
Heart failure					
No	44 (88%)	49 (98%)			
Yes	6 (12%)	1 (2%)	3.840	**0.049*(b)**	
Revascularization					
No	48 (96.0%)	48 (96.0%)			
Yes	2 (4.0%)	2 (4.0%)	0.001	1.00(b)	

LAD: left anterior descending artery, **N:** number**, LAD:** left anterior descending artery, **N:** number**, LVEF:** ventricular ejection fraction, **MR:** Mitral regurgitation, **(b):** Chi-Square Test, *: Statistically significant at $p \leq 0.05$, **: Highly statistically significant at $p < 0.001$, **P:** P-value between group

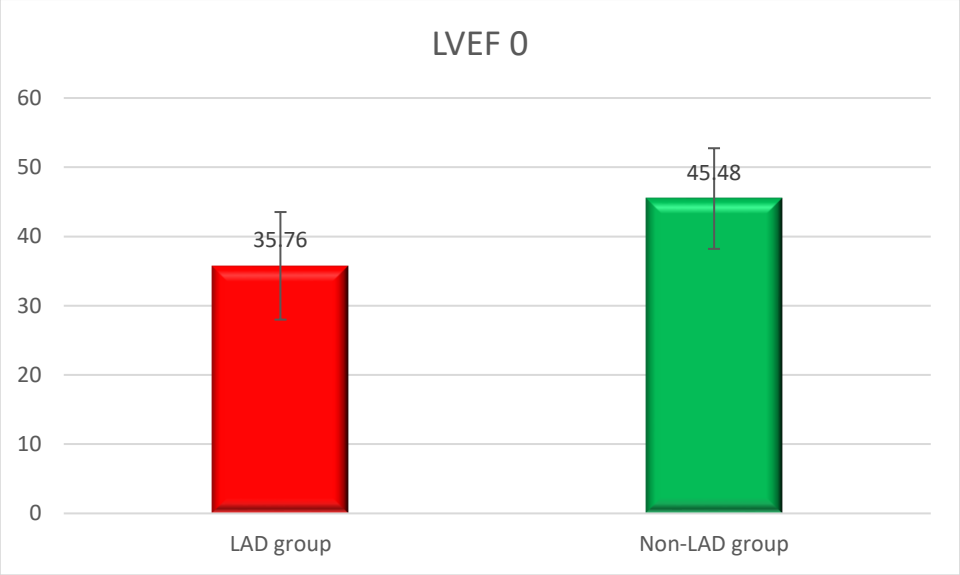

Figure (1): Comparison between the studied groups as regards bassline LVEF

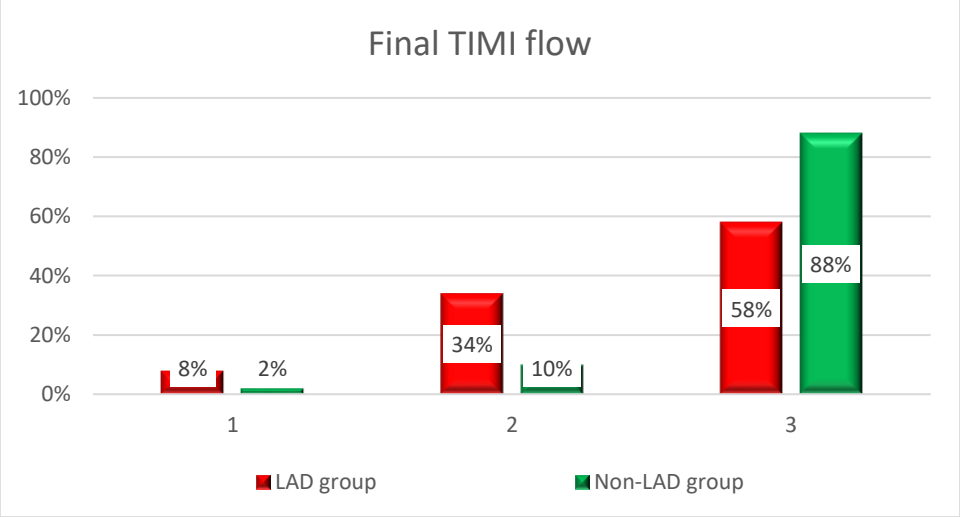

Figure (2): Final TIMI flow distribution among the studied groups

Discussion

No-reflow is defined as thrombolysis in myocardial infarction (TIMI) flow grade of <3 with patent coronary artery with an absence of dissection or spasm. It is a serious complication and accounts for 11%–41% of cases of ST-elevation myocardial infarction (STEMI) during primary percutaneous coronary intervention (PCI) [(2)].

It is thought to be caused by a combination of ischemic endothelial injury that obstructs the capillary lumen, neutrophil accumulation, reactive oxygen species and distal embolization of atherothrombotic debris [(9)].

In our study, LAD group patients had lower systolic blood pressure, higher HR and Killip class than those in non-LAD group. (P value >0.05). In line with our study Sa Couto D., et al 2023 [(12)] They detected that anterior STEMI group presented with higher Killip class than non-anterior STEMI group. Also, Khan et

DOI: 10.21608/bmfj.2025.335923.2251

al., 2022 [10] showed higher heart rate and Killip class in LAD group than non-LAD group (20% vs. 14%, p value =0.046).

In the current study, patients in LAD group had higher WBCs count and LDL/cholesterol than non-LAD group. (p value =0.019& <0.001). In agreement with our study Paul & Biswas 2020 [13] showed that LAD lesions were associated with higher WBCs count than non-LAD lesions (p value =0.0089) and Bodde M et al., 2109 [14] who demonstrated that higher LDL levels on admission were independently associated with greater infarct size presented with anterior STEMI. (p value<0.008)

In our study, baseline LVEF was statistically significant lower in LAD group (p value< 0.001). This was concordant to Entezarjou A et al., 2108 [15] who showed that the incidence of LVEF <30% was statistically significant higher in LAD vs. LCx vs. RCA culprit vessels. (8.2% vs. 2.7% vs. 1.9%, p value <0.001).

We found that no-reflow in LAD group was more refractory in nature and needed more aggressive treatment than non-LAD group. As number of patients received IC nitrate and IC adrenaline were statistically significant higher in LAD group (p value <0.001). In addition, the final TIMI flow 3 was statistically significant lower in LAD group than non-LAD group. This was consistent with Khan et al., 2022[10] who revealed that final TIMI II flow in LAD culprit group was 24.8% vs. 11.3% in non-LAD culprit group, final TIMI III flow in LAD culprit group was 74.4% vs. 87.5% in non-LAD culprit group (p value = 0.017 & 0.024; respectively). Also, showed that the incidence of LVEF <30 at 30 days was 24.8% in LAD group vs. 3.8% in non-LAD group, while the incidence of LVEF >40% was 16.5% in LAD group vs. 52.5% in non-LAD group (p value < 0.001).

Regarding 30 days follow up in our study, patients in LAD group had statistically significant higher incidence of heart failure (p value =0.049), while there was no significant difference between two groups regarding mortality, stroke or revascularization. This was concordant with Betric et al., 2020 [11] who detected that LAD culprit vessel in patients with_ out of hospital cardiac arrest_ had statistically significant higher incidence of congestive heart failure and cardiogenic shock than those with non-LAD culprit vessel (18.1% vs. 5.2% & 8.4% vs. 3.3%; respectively with p value< 0.0001). Also**,** Entezarjou et al.,2018 [15] revealed that the incidence of both 30 days and 1 year risk of heart failure was significantly higher in LAD culprit group compared to LCX and RCA culprit groups {4.2% vs. 2.2% vs. 1.4% & 10% vs. 5.9% vs. 4.3%; respectively (p value < 0.001)}.

The limitations of the current study were the relatively small sample size, and the short follow-up duration.

Therefore, a larger cohort with longer follow-up are recommended to validate our findings.

Conclusion

We can conclude that no-reflow in LAD group was more refractory in nature and needed more aggressive treatment than non-LAD group and was associated with more LVEF impairment and higher rate of heart failure incidence.

Sources of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution

Authors contributed equally to the study**.**

Conflicts of interest

No conflicts of interest

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To cite this article: Mahmoud S. Abd-Elmoneum, Khaled E. Elrabbat, Osama A. Ahmed, Hager I. Allam. No-Reflow after Stenting in Left Anterior Descending (LAD) versus Non-LAD Culprit in STEMI Patients, Angiographic Finding and Short-Term Outcomes. BMFJ XXX, DOI: 10.21608/bmfj.2025.335923.2251.