Comparison of angiographic results and clinical outcomes of no-reflow after stenting in left anterior descending (LAD) versus non-LAD culprit STEMI

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Abstract

Objectives: No-reflow is a complication that frequently occurs after stenting during primary percutaneous coronary intervention. In this study, we focused on angiographic results and clinical outcomes after no-reflow in the left anterior descending (LAD) artery versus non–left anterior descending artery ST-elevation myocardial infarction (STEMI).

Methods: In this prospective study, a total of 201 patients who had developed no-reflow during primary percutaneous coronary intervention were enrolled. The patients were divided into left anterior descending artery culprit and non-left anterior descending artery culprit groups. The primary endpoints were final thrombolysis in myocardial infarction flow, corrected thrombolysis in myocardial infarction frame count and final myocardial blush grade. Secondary endpoints were major adverse cardiovascular events in-hospital and at 1 month.

Results: Out of the 201 patients, 60.19% had culprit left anterior descending artery. Pulse rate, baseline systolic and diastolic blood pressure, single-vessel disease, left ventricular ejection fraction $<30\%$, baseline thrombolysis in myocardial infarction I flow and final thrombolysis in myocardial infarction II flow (24.8% vs 11.3%, $p=.017$), and thrombolysis in myocardial infarction frame count (28.17 ± 11.86 vs 24.38 ± 9.05, $p=.016$) were significantly higher in the left anterior descending artery group. In contrast, baseline Killip Class I, three-vessel disease, baseline thrombolysis in myocardial infarction II flow, final thrombolysis in myocardial infarction III flow (74.4% vs 87.5%, $p=.024$) and left ventricular ejection fraction $>40\%$ were significantly greater in the non–left anterior descending artery group. However, for both in-hospital and at 30 days, overall major adverse cardiovascular event was similar in the two groups. The demographics, clinical and medication profiles and the routes used to treat no-reflow were all comparable in both groups.

Conclusions: No-reflow in left anterior descending artery ST-elevation myocardial infarction is associated with lower final thrombolysis in myocardial infarction III flow, higher thrombolysis in myocardial infarction frame count and relatively lower Grade III myocardial blush than non-left anterior descending artery ST-elevation myocardial infarction with subsequent lower left ventricular ejection fraction and a higher frequency of in-hospital heart failure and hospitalisation due to heart failure.

Keywords

No-reflow, primary PCI, stenting, LAD-culprit STEMI

Introduction

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.[1] 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] 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.[3–6] Moreover, there is evidence of a correlation between no-reflow and reduced

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left ventricular function, worse clinical outcome and higher mortality.[7]

Of particular importance, Nair et al. have reported a significant association of anterior wall myocardial infarction (AWMI) with the development of no-reflow (71.4% vs 43.2%, $p \leqslant .001$).[8] 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.[9] Several studies have been performed to address the predictors and use of medication for treatment. In the main, adenosine, verapamil, nitroprusside or nicardipine have resulted in improved coronary flow and better prognoses.[10–12] However, literature related to immediate treatment outcomes in terms of TIMI flow, final corrected TIMI frame count (cTFC) and myocardial blush grade (MBG), and lately overall clinical outcomes comparing LAD versus non-LAD STEMI is sparse. Therefore, we aimed to conduct this study of our population where the proportion of young patients with STEMI is on the rise, and the majority of cases were found to have LAD territory STEMI, as has been reported recently.[13] In this study, we focused on a comparison of angiographic results and clinical outcomes after no-reflow in the LAD versus non-LAD STEMI irrespective of the age.

Method

This was a prospective study conducted in a tertiary care cardiac hospital in an urban area in Pakistan from December 2020 to May 2021. Out of all STEMI patients ($n = 1862$) presented to the National Institute of Cardiovascular Diseases (NICVD), 201 were enrolled in the study, having fulfilled the inclusion criteria. The inclusion criteria for the study were patients aged $\geqslant 18$ years, who had been diagnosed with STEMI, were undergoing primary PCI and had developed TIMI flow grade $\leqslant 2$ post stenting. Patients with cardiogenic shock at the time of presentation, valvular or congenital heart disease, cardiomyopathy, myocarditis, pericarditis, contraindication to the use of antiplatelet or anticoagulation and those who did not give their consent to participate in the study were excluded.

The recruited patients were divided into two groups: LAD-culprit STEMI and non-LAD culprit STEMI. Data pertaining to sex, age, body mass index (BMI), hypertension, diabetes mellitus, smoking, prior coronary artery diseases (CADs), a family history of premature CAD, dyslipidaemia, haemodynamics and Killip Class on presentation were recorded after written and informed consent had been given in the emergency room. LVEF was assessed via echocardiogram on discharge and during the 30-day follow-up. Angiographic parameters, including vessel size (diameter), heparin dose, use of glycoprotein (GP) IIb/IIIa inhibitor (as indicated by American College of Cardiology and American Heart Association (ACC-AHA) guidelines), intracoronary use of medication, like nitrate, adenosine or adrenaline, and drug delivery route, such as proximal via guide catheter or distal via a device, were documented and compared.

Improvement in TIMI flow grade, reduction in cTFC (the number of cine-frames needed for contrast to get to the distal coronary landmark, corrected for the vessel length) and final cTFC, and improvement in MBG after the intracoronary administration of medicine were assessed by at least two experienced interventional cardiologists blinded to the groups and then compared as the primary endpoint. Major adverse cardiovascular events, including all-cause mortality, re-infarction, repeat revascularisation and cerebrovascular accidents (CVAs) in-hospital and at 1 month, were compared as a secondary endpoint.

Statistical analyses

The sample size for the study was the number of consecutive patients that fulfilled the inclusion criteria out of the total STEMI patients presented during the study period. A power analysis showed the power of 0.657 for the detection of difference in the proportion of final TIMI III flow between the LAD-culprit STEMI and non-LAD culprit STEMI groups, at .05 level of significance. IBM SPSS Version 21 was used for the analysis of the data. Descriptive statistics such as mean $\pm$ SD or median (interquartile range (IQR)) and frequency and percentages were calculated for quantitative and qualitative variables, respectively. The outcomes were compared between the two groups with the help of an appropriate chi-square test and independent sample t test or Mann–Whitney U test. A two-sided p value of $\leqslant .05$ was taken as the criteria for significance.

Results

Out of the 201 consecutively enrolled patients with no-reflow after stenting, the proportion of patients in the LAD-culprit STEMI group was 60.19% (121) and the non-LAD culprit STEMI group was 39.8% (80) of which 82.5% (66/80) had culprit right coronary artery (RCA) and the remaining 17.5% (14/80) had culprit left circumflex artery. Among the patients with culprit LAD, 73.6% (89/121) had disease in the proximal segment, 34.8% (21/121) in the mid-distal segment, and one patient (3.2%) had disease in the diagonal. Baseline demographic and clinical profiles, including sex, mean age, hypertension, diabetes mellitus, smoking, dyslipidaemia, family history of premature CAD and history of prior CAD, were comparable in the two groups, as shown in Table 1. Moreover, procedural medication profiles, including dose of heparin, use of GP IIb/IIIa inhibitor (as per ACC-AHA indications), doses of intracoronary medications used to manage no-reflow, such as nitrate, adenosine and adrenaline and the proportion of patients that received intracoronary medication proximally via guide catheter and distally via a device were also comparable in both groups, as shown in Table 2.

Table 1. Baseline demographic and clinical characteristics.

Characteristics	Total	Group		p
		LAD	Non-LAD	
Total (*N*)	201	121	80	–
Gender				
Male	72.6% (146)	69.4% (84)	77.5% (62)	.209
Female	27.4% (55)	30.6% (37)	22.5% (18)	
Age (years)	57.15 ± 11.39	57.85 ± 11.46	56.1 ± 11.27	.287
Body mass index (BMI)	27.3 ± 4.05	27.14 ± 3.72	27.56 ± 4.52	.475
Admission blood sugar (mg/dL)	161 (130–207)	164 (129–204)	160 (133.5–217)	.247
Admission serum creatinine (mg/dL)	1 (0.8–1.2)	1 (0.8–1.2)	0.9 (0.8–1.1)	.518
Total ischemic time (minutes)	440 (320–710)	440 (320–720)	420 (320–645)	.053
Co-morbid conditions				
Hypertension	71.6% (144)	68.6% (83)	76.3% (61)	.239
Diabetes mellitus	45.3% (91)	46.3% (56)	43.8% (35)	.724
Smoking	32.8% (66)	29.8% (36)	37.5% (30)	.252
Family history of IHD	2.5% (5)	0.8% (1)	5% (4)	.063
Prior CAD	8.5% (17)	9.1% (11)	7.5% (6)	.692
Dyslipidaemia	10% (20)	12.4% (15)	6.3% (5)	.154

LAD: left anterior descending artery; IHD: ischemic heart disease; CAD: coronary artery disease.

Table 2. Procedural medication profile.

Characteristics	Total	Group		p
		LAD	Non-LAD	
Total (*N*)	201	121	80	–
Heparin dose (IU)	8039.8 ± 1398.54	7925.62 ± 1397.41	8212.5 ± 1391.15	.155
Use of GP IIb/IIIa inhibitor	21.9% (44)	24% (29)	18.8% (15)	.381
Use of nitrates	81.6% (164)	84.3% (102)	77.5% (62)	.224
Dose (mcg)	351.83 ± 267.57	374.51 ± 273.84	314.52 ± 254.69	.120
Use of adrenaline	50.2% (101)	47.9% (58)	53.8% (43)	.42
Dose (mcg)	240.1 ± 131.53	243.97 ± 133.14	234.88 ± 130.72	.634
Use of adenosine	49.8% (100)	52.1% (63)	46.3% (37)	.42
Dose (mcg)	257.5 ± 132.55	254.44 ± 133.64	262.7 ± 132.34	.667
Administration				
Distal via device	11.9% (24)	12.4% (15)	11.3% (9)	.806
Proximal via guide	88.1% (177)	87.6% (106)	88.8% (71)	

LAD: left anterior descending artery; GP: glycoprotein.

Considering the hemodynamic profiles of the LAD-culprit and non-LAD culprit STEMI groups, pulse rate was 90.24 ± 12.68 versus 72.3 ± 17.73, $p \leqslant .001$, baseline systolic blood pressure was 133.01 ± 19.5 versus 127.2 ± 15.61, $p = .027$, diastolic blood pressure was 83.26 ± 12.51 versus 78.83 ± 11.15, $p = .011$ and baseline Killip Class I was 69.4% versus 82.5%, $p = .037$, respectively. Angiographic profiling showed single-vessel disease as 44.6% versus 21.3%, $p \leqslant .001$, three-vessel disease at 28.1% versus 42.5%, $p = .035$ and the culprit vessel diameter at $3.41 \, \text{mm} \pm 0.3 \, \text{mm}$ versus $3.44 \, \text{mm} \pm 0.37 \, \text{mm}$, $p = .536$, respectively, in the LAD and non-LAD groups, as shown in Table 3.

Baseline TIMI I flow in the two groups was 71.1% versus 56.3%, $p = .028$, the TIMI II flow was 26.4% versus 41.3%, $p = .028$, and the pre-PCI high thrombus grade ($\geqslant 4$) was 43.8% versus 31.3%, $p = .074$, respectively, in the LAD versus non-LAD arm. Post-medication treatment of no-reflow in the LAD and non-LAD groups revealed a final TIMI II flow of 24.8% versus 11.3%, $p = .017$, a final TIMI III flow of 74.4% versus 87.5%, $p = .024$, a final cTFC of 28.17 ± 11.86 versus 24.38 ± 9.05, $p = .016$, an overall reduction in cTFC in each group at -25.27 ± 10.44 versus -26.84 ± 13.45, $p = .356$ and a final MBG III of 48.8% versus 57.5%, $p = .225$, respectively, as shown in Figures 1–3. Echocardiography showed LVEF of $\leqslant 30\%$ at 31.4% versus 5%, $p \leqslant .001$ and 24.8% versus 3.8%, $p \leqslant .001$ while LVEF of $> 40\%$ was 14% versus 60%, $p \leqslant .001$ and 14% versus 60%, $p \leqslant .001$, at hospital discharge and at the 30-day point,

Table 3. Haemodynamics and angiographic profile.

Characteristics	Total	Group		p
		LAD	Non-LAD	
Total (N)	201	121	80	–
Killip Class				
I	74.6% (150)	69.4% (84)	82.5% (66)	.037*
II	14.9% (30)	18.2% (22)	10% (8)	.111
III	10.4% (21)	12.4% (15)	7.5% (6)	.267
Pulse rate (bpm)	83.1 ± 17.27	90.24 ± 12.68	72.3 ± 17.73	<.001*
Systolic blood pressure (mmHg)	130.7 ± 18.24	133.01 ± 19.5	127.2 ± 15.61	.027*
Diastolic blood pressure (mmHg)	81.49 ± 12.15	83.26 ± 12.51	78.83 ± 11.15	.011*
Number of diseased vessels				
Single-vessel disease	35.3% (71)	44.6% (54)	21.3% (17)	<.001*
Two-vessel disease	30.8% (62)	27.3% (33)	36.3% (29)	.177
Three-vessel disease	33.8% (68)	28.1% (34)	42.5% (34)	.035*
Pre-procedure thrombus grade				
Low thrombus grade (1 to 3)	61.2% (123)	56.2% (68)	68.8% (55)	.074
High thrombus grade (4 and 5)	38.8% (78)	43.8% (53)	31.3% (25)	
Vessel size	3.42 ± 0.33	3.41 ± 0.3	3.44 ± 0.37	.536

LAD: left anterior descending artery.
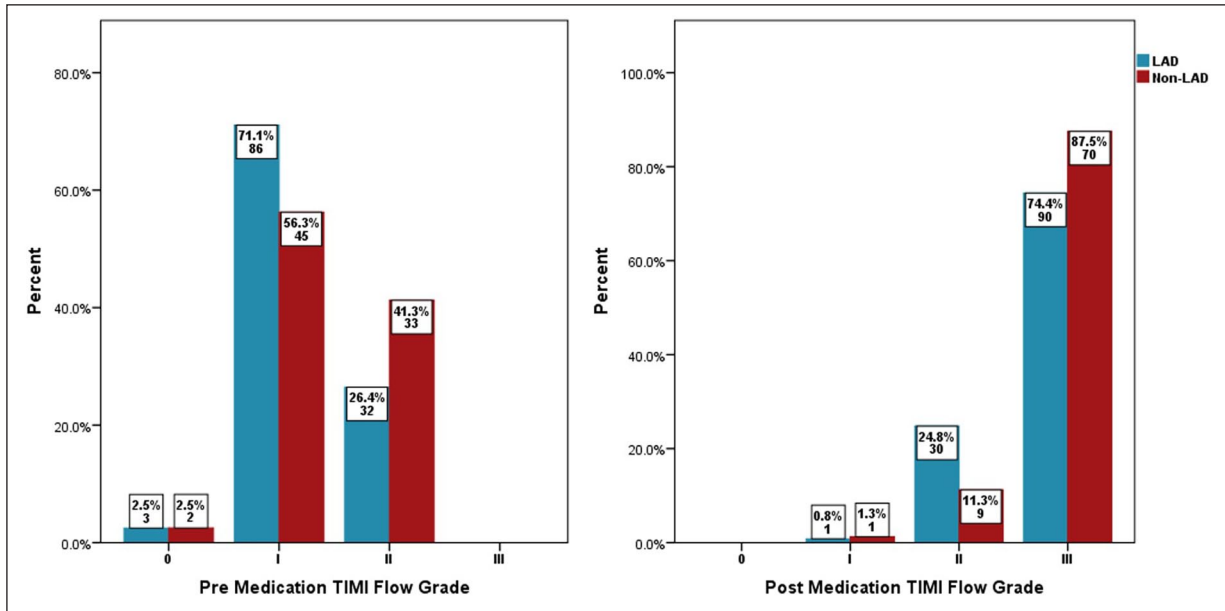*Significant at 5%.
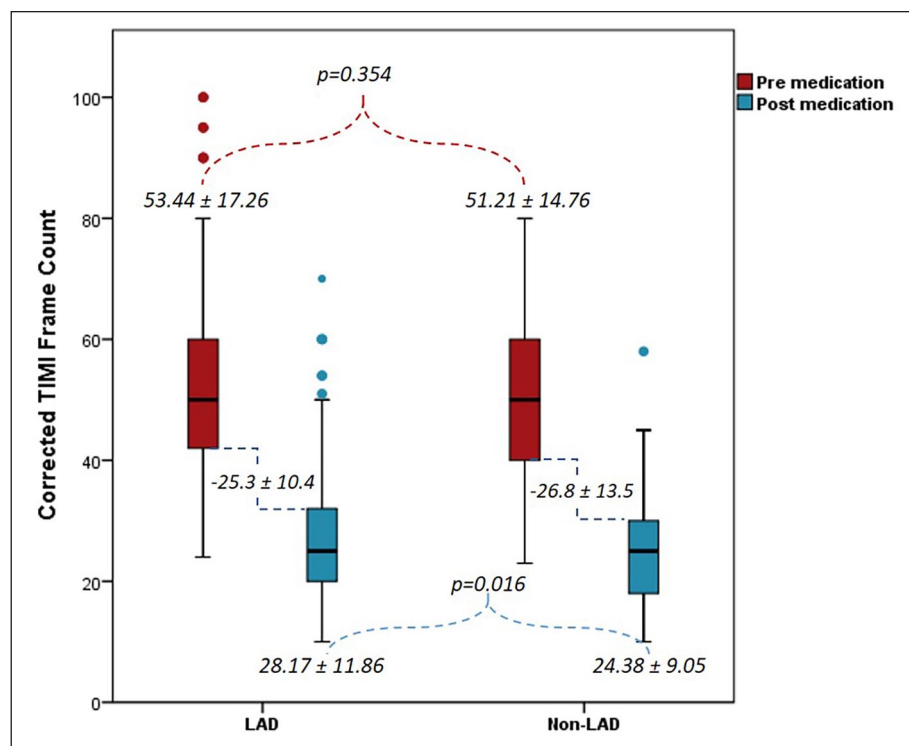
Figure 1. Pre- and post-medication corrected thrombolysis in myocardial infarction (TIMI) frame count.
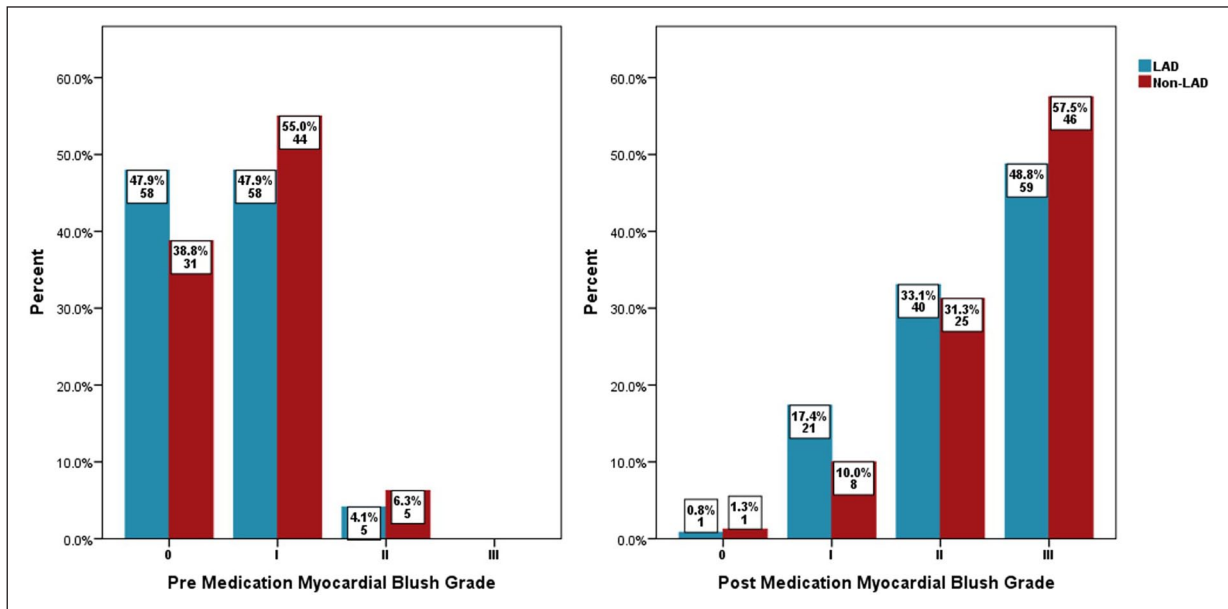LAD: left anterior descending artery; TIMI: thrombolysis in myocardial infarction.

respectively, in the LAD and non-LAD groups, as shown in Figure 4.

Major adverse cardiovascular event (MACE) in-hospital and at 30 days revealed all-cause mortality at 3.3% versus 1.3%, $p = .36$ and 4.5% versus 2.5%, $p = .484$, re-infarction was 0.8% versus 1.3% and 1.8% versus 3.8%, $p = .391$, repeat revascularisation was 2.5% versus 1.3%, $p = .541$ and 2.7% versus 2.5%, $p = .95$, CVA was 1.7% versus 1.3%, $p = .818$ and 0.9% versus 0%, $p = .4$, and heart failure stood at 25.6% versus 10%, $p = .006$ and 3.6% versus 1.3%, $p = .326$,

Figure 2. Pre- and post-medication thrombolysis in myocardial infarction (TIMI) flow grade.
LAD: left anterior descending artery; TIMI: thrombolysis in myocardial infarction.


Figure 3. Pre- and post-medication myocardial blush grade.
LAD: left anterior descending artery.

respectively, in the LAD and non-LAD culprit STEMI groups, as shown in Table 4.

Discussion

No-reflow has always been a critical problem during primary PCI for all STEMI, but earlier studies, by Ito et al.[14] in 1996 and Pantea-Roșan et al.[15] in 2020, have reported that cases of LAD-culprit MI are more prone to having this complication and consequently experiencing more severe myocardial damage with an increased risk of future cardiovascular events and death.[7,16] The present study also supported this fact, as 60.19% (121) of patients who had developed no-reflow were found to have LAD-culprit STEMI, in contrast to 39.8% (80) with non-LAD culprit STEMI. In a recent study, Refaat et al. also reported no-reflow in 53.6% of anterior STEMI, in contrast to 43.6% of non-anterior STEMI.[17] In the present study, no significant differences were observed in the patients' demographics and clinical profiles, as depicted in Table 1, between LAD-culprit and non-LAD culprit STEMI. However,
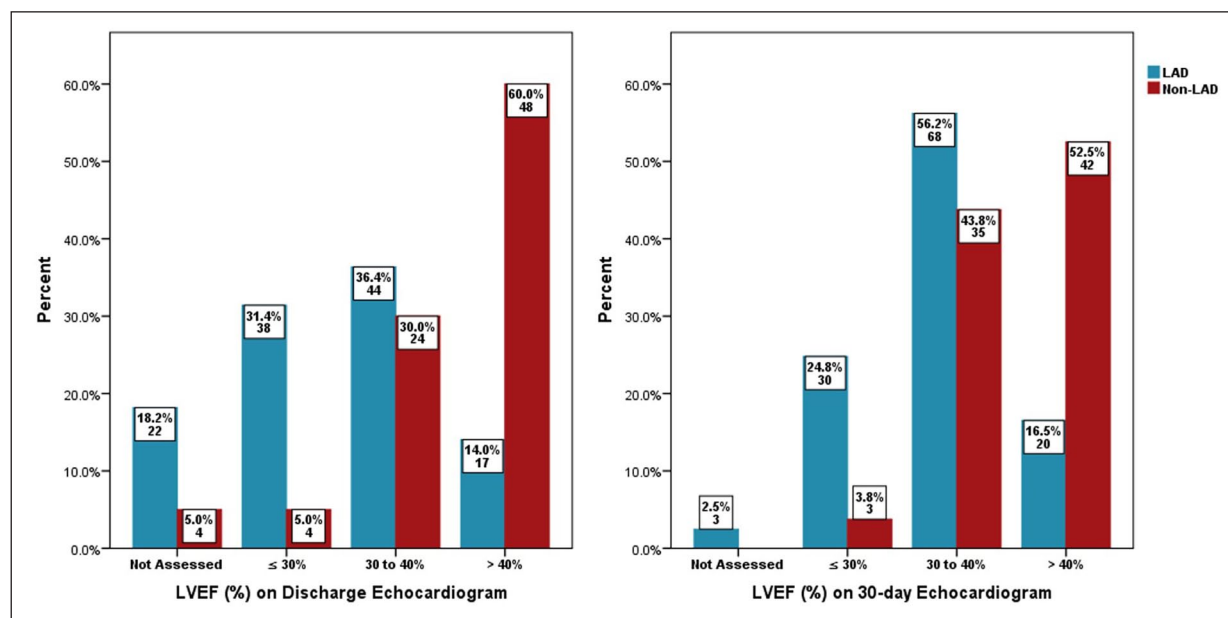
Figure 4. At discharge and 30-day (echocardiogram) left ventricular ejection fraction (%).
LAD: left anterior descending artery; LVEF: left ventricular ejection fraction.

Table 4. In-hospital and 30-day follow-up outcomes.

Characteristics	Total	Group		p
		LAD	Non-LAD	
Total (N)	201	121	80	–
In-hospital outcomes				
Mortality	2.5% (5)	3.3% (4)	1.3% (1)	.36
Arrhythmia	5% (10)	5% (6)	5% (4)	.989
Re-infarction	1% (2)	0.8% (1)	1.3% (1)	.767
Repeat revascularisation	2% (4)	2.5% (3)	1.3% (1)	.541
Cerebrovascular accident	1.5% (3)	1.7% (2)	1.3% (1)	.818
Heart failure	19.4% (39)	25.6% (31)	10% (8)	.006*
Follow-up	95% (191)	92.6% (112)	98.8% (79)	.048*
Duration of follow-up	29.77 ± 1.5	29.68 ± 1.53	29.91 ± 1.44	.291
Mortality	3.7% (7)	4.5% (5)	2.5% (2)	.484
Re-infarction	2.6% (5)	1.8% (2)	3.8% (3)	.391
Repeat revascularisation	2.6% (5)	2.7% (3)	2.5% (2)	.95
Cerebrovascular accident	0.5% (1)	0.9% (1)	0% (0)	.4
Hospitalisation due to heart failure	2.6% (5)	3.6% (4)	1.3% (1)	.326
Cumulative mortality	6% (12)	7.4% (9)	3.8% (3)	.235

LAD: left anterior descending artery.
*Significant at 5%.

the frequency of patients with baseline Killip Class I was significantly lower in LAD-culprit STEMI compared to the non-LAD group, with a concomitant higher baseline heart rate and both systolic and diastolic blood pressure manifesting a slightly less stable presentation to start with in this group. A study by Vicent et al. reported an odd ratio of 2.4 (1.2–2.7) for anterior STEMI and 1.8 (1.2–2.7) for TIMI flow grade of <3 in patients with higher Killip Class ≥ 2.[18] Considering the angiographic profile, there was no significant difference in

the vessel diameter of the culprit vessel in the study groups; however, most of the LAD-culprit STEMI patients were found to have single-vessel involvement, in contrast to the non-LAD culprit patients. Niccoli at al. also reported similar findings in their study where 62% of patients with no-reflow exhibited single-vessel disease and LAD was culprit in 48% of them.[19]

Regarding the severity of no-reflow, the majority of patients in the LAD-culprit STEMI group had TIMI I flow in

contrast to the no-reflow severity in the non-LAD culprit group, whose proportion of baseline TIMI II flow was more than that of the LAD group, suggesting the more extensive nature of myocardial involvement with LAD-culprit STEMI. This was also shown by Elakabawi et al.'s study where, despite medical treatment, 22.2% (out of 1104) anterior STEMI patients continued to have suboptimal flow (TIMI ≤ 2).[16,20] This finding supports the earlier observation that anterior STEMI is an independent predictor of procedural no-reflow.[21]

As shown in Table 2, patients in both groups were given similar medical treatment pre and post no-reflow with no significant differences in doses of heparin, utilisation of GP IIb/IIIa inhibitor and intracoronary drugs. Nonetheless, in view of primary outcomes, there was a significantly lower frequency of final TIMI III flow with a concomitant higher frequency of final TIMI II flow and higher final cTFC post intracoronary medication in the LAD group, compared with the non-LAD group. However, there was no significant difference in individual reduction of frame count in each group and the only downwards trend was observed in the achievement of final Grade III myocardial blush in the LAD-culprit STEMI group, as shown in Figures 1–3. These findings pointed towards a more refractory nature of no-reflow in LAD-related STEMI, as shown in earlier studies by Lee et al. where 67% of patients with refractory no-reflow were from anterior STEMI. Iwakura et al. also reported a significant association between no-reflow and severe myocardial damage and worse outcome in LAD-culprit STEMI.[20,22] Furthermore, our evaluation of LVEF via echocardiography, both on discharge and at 30 days, revealed the proportion of LVEF ≤30% as significantly higher in LAD-culprit STEMI after no-reflow, in contrast to the non-LAD culprit group, suggesting the more extensive nature of anterior STEMI and associated no-reflow. Sjögren et al. have also reported significantly impaired longitudinal strain in LAD infarction and, subsequently, more heart failure symptoms in their study.[23] This further highlights the more devastating nature of LAD-culprit STEMI and no-reflow, as compared to the non-LAD group.

Finally, there was a significantly greater frequency of heart failure in LAD-culprit STEMI after no-reflow than non-LAD culprit patients, and this finding was consistent with several earlier studies.[24–26] However, there was no significant difference found in overall MACE in-hospital and at the 30-day point, except for an increasing trend in mortality in the LAD-culprit group.

In summary, despite optimal treatment of no-reflow with widely used medications in both groups, the final angiographic results, in terms of TIMI III flow, remained less impressive in the LAD group. This is likely due to the extensive nature of myocardial involvement as a result of large territorial distribution, and conversely it was reflected in less reduction of cTFC and lower levels of improvement in LVEF at 30 days in this group. Nonetheless, overall MACE, both in-hospital and at 30 days, failed to reach statistical

significance between the two groups, except a significantly higher frequency of in-hospital heart failure in the LAD group.

Our study has certain limitations, such as the exclusion of haemodynamically unstable patients, a small sample size (low power of test) and single-centred recruitment, which can become a source of selection bias. Second, due to the low-event rate of suboptimal final TIMI III flow, the sample size was not sufficient to conduct multivariable analysis. Hence, large-scale multicentre studies are needed to further validate the findings of our study.

Conclusion

No-reflow in LAD-culprit STEMI is associated with lower final TIMI III flow, higher final cTFC and relatively lower Grade III myocardial blush than non-LAD STEMI. Subsequently, there is lower LVEF and a higher frequency of in-hospital heart failure and hospitalisation due to heart failure in this group. There is an increasing trend in mortality with no-reflow in LAD-culprit group but overall, in-hospital and at 3 months MACE is similar to that of non-LAD culprit group.

Ethical approval

Ethical approval for this study was obtained from institutional ethical review board with approval number 76/2020.

Informed consent

Written informed consent was obtained from all subjects before the study.

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