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Post Thrombolytic Angiographic Profile and TIMI Flow in Patients with ST-Elevation Myocardial Infarction Receiving Thrombolytic Therapy

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Abstract:

Background: The term "myocardial infarction" (MI) is frequently used to describe the mortality of cardiomyocytes that results from a prolonged and significant ischemia that is caused by an imbalance in oxygen supply and demand. The objective of this study was to assess the angiographic characteristics and angiographic efficacy of thrombolysis in relationship to the flow of thrombolysis in myocardial infarction patients ("TIMI") who underwent angiography subsequent to ST-segment elevation myocardial infarction (STEMI) thrombolysis. Furthermore, most frequently associated risk factors with unsuccessful thrombolysis were identified. **Methods:** This dual center and prospective observational study included 130 STEMI patients in the CCU unit, both Benha University Hospital and National Heart Institute. Patients were divided into two groups angiographic TIMI flow post-thrombolytic therapy Group I (n= 44): patients with TIMI flow ≤ 2 and group II (n=86): patients with TIMI flow 3. **Results:** In the univariate analysis, diabetes mellitus presence, hypertension, hyperlipidemia, a history of coronary artery disease, a longer duration from the onset of symptoms to the thrombolytic, a lower systolic blood pressure, a lower diastolic blood pressure, a higher random blood glucose (RBG), an increased alanine aminotransferase, a lower left ventricular ejection fraction, an increased number of vessels affected, the presence of ostial segment affection, and a high thrombus Burden Grade were all associated with an increased risk of TIMI 2 flow or less. **Conclusion:** Incidence of TIMI flow < 3 in STEMI patients received thrombolytic therapy was 33.8%.

Keywords: Thrombolytic Therapy; Thrombolysis in Myocardial Infarction Flow; ST-Elevation Myocardial Infarction; Post Thrombolytic Angiographic Profile

Introduction

The primary global cause of mortality and morbidity is myocardial infarction (MI), as it appears [1]. A term that is frequently used to describe a significant and protracted ischemia that is the consequence of an imbalance in oxygen supply and demand is MI, which is the mortality of cardiomyocytes. The electrocardiogram (ECG) trace is frequently employed to distinguish between ST-segment elevation myocardial infarction (STEMI) and Non-ST-segment elevation myocardial infarction (NSTEMI) [2].

NSTEMI does not extend throughout the entire myocardial wall, whereas STEMI is result of transmural ischemia, which is ischemia involving entire myocardium thickness. An epicardial coronary artery is wholly obstructed by a thrombus (a blood clot) that has formed on a coronary atherosclerotic plaque, resulting in transmural myocardial ischemia, the most common complication of STEMI. STEMI is defined as persistent cardiac distress and ST-segment elevation in two or more anatomically contiguous ECG leads. Furthermore, If the clinical presentation is consistent and the ECG trace demonstrates left bundle branch block (LBBB) and no ST-segment elevation, STEMI should be suspected, as total coronary occlusion may manifest as LBBB in specific circumstances [3]. In contrast, non-ST-segment elevation ACS is identified by ECG findings of transient T wave inversions, ST-segment depressions, or ST-segment elevations, which may indicate unstable angina or NSTEMI [4].

In STEMI, the most severe form of coronary artery disease (CAD), there is a high incidence of mortality and morbidity. The best method for maintaining the ischemic myocardium's viability and reducing the extent of the infarct is early reperfusion, the process of reestablishing blood flow in the obstructed artery. A STEMI diagnosis should preferably be made within ten minutes after the initial medical contact in order to start the proper treatment [5]. Initiatives have highlighted the importance of reducing the delay to reperfusion by supporting early diagnosis and transfer to a facility that offers subsequent primary percutaneous coronary intervention (PPCI) immediately and cardiac catheterization [6].

Due to the systematic implementation of early mechanical reperfusion and advancements in adjuvant pharmacology, the future prospects for patients with acute MI has significantly improved in the past two decades [7].

Our investigation aimed to evaluate the efficacy of thrombolysis and the angiographic characteristics of myocardial infarction (TIMI) 3 in patients underwenting angiography following thrombolysis for STEMI. In addition, we attempted to identify the most frequently reported factors that are linked to ineffective thrombolysis.

Patients and methods

This prospective observational study included 130 STEMI patients who underwent either routine early PCI (after successful fibrinolysis) or recue PCI (in case of failed thrombolysis) and received thrombolytic therapy (Streptokinase) within the first 12 hours of symptom onset. The study was conducted at a dual center in the CCU unit, both Benha University Hospital and National Heart Institute from April 2023 to June 2024.

The patients provided written consent that was informed. Each patient was informed of the purposes of the study and allocated a confidential code number. The research was conducted with the approval of the Research Ethics Committee (MS 33-4-2024), Faculty of Medicine, Benha University, and National Heart Institute.

Inclusion criteria were patients with STEMI who received thrombolytic therapy (Streptokinase) within the first 12 hours of clinical onset and underwent either routine early PCI (in case of successful thrombolysis) or rescue PCI (in case of failed thrombolysis) and patients with STEMI who presented with typical chest

pain for MI that lasted for at least 30 minutes and lasted for less than 12 hours. In two contiguous leads of the 12-lead ECG, a novel ST segment elevation was observed at the J point. The cut-off point was >= 0.1 mV in all leads. The cut-off values were as follows: ≥ 0.25 mV for males under the age of 40, ≥ 0.2 mV for males over the age of 40, and ≥ 0.15 mV for women respectively[10].

Exclusion criteria were patients with serious and moderate valvular heart disease, pre-existing cardiomyopathy, NSTEMI, the failure to attempt fibrinolysis, late presentation after the onset of symptoms (than 12 hours from the time of chest pain), and contraindications to thrombolytics.

Grouping: Following thrombolytic therapy, patients were categorized into two categories based on the angiographic TIMI flow: **Group I (n= 44):** patients with TIMI flow ≤ 2 and **group II (n=86):** patients with TIMI flow 3.

All patients were subjected to the following: Complete history taking, including [present history, personal history including (residence, parity, age, occupation, special habits of medical importance specially smoking), Risk factors for ischemic heart disease include hypertension, which is diagnosed by a diastolic blood pressure (DBP) of 90 mmHg or higher and a systolic blood pressure (SBP) of 140 mmHg or higher [12], smoking, dyslipidemia and diabetes which is diagnosed by 2h PPBG ⩾ 200 mg\dl or FBG ⩾126 mg\dl or classic diabetes mellitus (DM) symptoms with random blood glucose (RBG) > 200 mg\dl or HbA1c >6.5% [11], history of sensitivity to drugs, past medical history, family history of premature CAD past surgical history, prior history of CAD or coronary artery bypass graft, current medical therapy, and prior atherosclerotic cerebrovascular events.

Physical examination including **(**pulse, blood pressure, temperature and hemodynamics). **Local examination** including local cardiopulmonary examination including (cardiac auscultation for additional heart sound or murmur that may indicate mechanical complications, chest auscultation, identification of Killip class {Class I indicated rales and S3 absent, class II indicated S3 gallop, crackles, class III & elevated jugular venous pressure indicated frank pulmonary edema and class IV indicated cardiogenic shock [15]}, height ,body weight for calculation of body mass index "BMI = mass/ height^2" [13], time from pain onset to thrombolytic therapy, successful thrombolysis (Chest pain and failed thrombolysis (persistent ST-segment elevation or chest pain) disappearance; typical reperfusion arrhythmia; and ST-segment resolution > 50% at 60–90 minutes) [14].

12 Lead standard ECG:

In the following scenarios, the J-point is used to quantify ST-segment elevation, which is considered a predictor of persistent transient occlusion of the coronary artery. This is the case when two or more contiguous leads have ST-segment elevations of 2mm in men under the age of 40, at least 2.5mm in men under the age of 40, or 1.5 mm in women in leads V2–V3, and/or 1mm in the other leads [in the absence of left bundle branch block LBBB or left ventricular (LV) hypertrophy] if the J-point was administered within 10 minutes of the initial medical contact.

In order to detect concomitant right ventricular infarction and ST-segment elevation in patients with inferior MI, it is advised that right precordial leads (V4R and V3R) be recorded [16].

Routine laboratory investigations including cardiac enzymes (Creatine kinase (CK),Troponin, and creatine kinase myocardial band (CK –MB) random blood sugar, serum electrolytes including serum sodium and potassium, kidney function tests including aspartate aminotransferase (AST), urea, creatinine and alanine aminotransferase (ALT) and coagulation profile including prothrombin

concentration (PC) partial thromboplastin time (PTT), and prothrombin time (PT).

Transthoracic echocardiography (TTE): The Philips Epiq 7 machine with a 5s-1 instrument and simultaneous ECG signal was used to conduct an echo-Doppler exam on all patients within 48 hours of admission. The patients were examined in left lateral decubitus to obtain parasternal and apical long axis views through an accessible opening. The echo-Doppler parameters were obtained according to the American society of cardiology(ASC) [17]**:** ASC suggests modified Simpson method (biplane method of discs) for the acquisition of EF using 2-dimensional echocardiography. It is essential to identify in order to quantify the left ventricular ejection fraction (LVEF) using this method, the endocardial border is examined in both apical four-chamber and two-chamber views during systole and diastole. Color flow mapping and continuous wave doppler were employed to evaluate the presence and severity of the Mitral regurgitation jet, as well as to visually assess regional wall motion and thickening. With these tracings, the LV cavity is ultimately divided into a specific number of disks, which is typically 20.

Coronary angiography & intervention: PCI was routine or rescue according to successfulness of thrombolytic therapy, number of diseased vessels, culprit vessel and site of obstruction, vessel diameter (mm), thrombus burden grade according to TIMI thrombus grading, Grade 0 referred to the absence of a thrombus, Grade 1 indicated the possibility of a thrombus, Grade 2 indicated that the thrombus' greatest dimensions were less than ½ of a vessel diameter, Grade 3 indicated that thrombus' greatest dimension was between ½ and 2 vessel diameters, Grade 4 indicated that the greatest dimension was greater than 2 vessel diameter, and Grade 5 indicated total vessel occlusion. The number of stents implanted, the size and length of the stent, thrombus aspiration, drugs used during the intervention, pre- or post-stent implantation dilation, and the assessment of TIMI flow pre- and post-stent deployment were assessed.

Definition of the TIMI flow grades: Indicative of grade 0, the absence of any flow after the culprit lesion, which fails to opacify the entire artery after the occlusion site, while the flow of contrast material is indicative of grade 1. In Grade 2, the entire artery distal to the obstruction site is opacified, despite the fact that the flow is slower than normal. Normal coronary flow is defined as Grade 3 [18].

Sample size

The sample measure of the requisite quantity was determined in conformance with the G Power software version 3.1.9.7with Kumar et al., [19] with level of significance =0.05 and type II error =0.2, the minimum calculated sample size was 130 participants.

Statistical analysis

Data was analyzed using SPSS version 27 (IBM, Chicago, IL, USA). We used statistics and the Shapiro-Wilk test to make sure the data was normally distributed. Our quantitative parametric data was evaluated using an ANOVA followed by a Tukey post hoc test. The data were eventually presented as a mean with a standard deviation (SD). In order to provide quantifiable non-parametric data, the IQR and median were used. Each group was analyzed using Kruskal-Walli's test with a Mann Whitney-test. After analyzing the qualitative variables with the Chi-square test, a report was generated that included their frequency and percentage (%). Results were deemed statistically significant when the two-tailed P value $\leq$ 0.05. Divide the sum of all the data by total number of observations to obtain the mean (X). Then, add up the standard deviations (SD). The x^2 chi-square test examines the degree to which individual varieties scatter around their mean is measured by testing the hypothesis that the row and column variables are independent. No data on the strength or direction of the link is provided by this test. Using

quantitative data, unpaired student t-test was utilized to compare two groups. Tables of pivots, alternate chi-square tests, and likelihood ratio studies [20].

Results

Incidence of TIMI flow < 3 in STEMI patients received thrombolytic therapy was 33.8%. In terms of gender and age insignificant difference was observed between the two categories. There was a significant increase in incidence of diabetes mellitus and hypertension among patients in Group I (p-value < 0.001), as well as a higher incidence of dyslipidemia and a history of CAD (p-value < 0.05). Also, median duration from onset of symptoms till thrombolytic was significantly higher in patients of group I with P-value < 0.001. Nevertheless, there was insignificant difference between the two groups examined in terms of the incidence of family history of premature CAD, smoking, and history of cerebrovascular stroke (P-value > 0.05). Patients of Group I had statistically significant lower median SBP, DBP and higher median HR than those of group II (P-value < 0.05). Also, patients of group I had higher incidence of Killip class III & IV and lower Killip class I & II (P-value > 0.01). **Table 1**

The two groups did not exhibit any significant differences in the location of STEMI. Group I patients exhibited significantly elevated median peak creatine kinase myocardial band (CkMb) (IU) and Troponin levels (P-value < 0.001). Statistically, the median values of RBG and ALT were significantly higher in group I patients than in group II (P-value < 0.05). Although the two groups that were examined did not exhibit any significant differences in terms of other laboratory data **Table 2**

The median LVEF% of group I patients was considerably lower than in group II patients (P-value <0.001). Nevertheless, there was insignificant difference in terms of LVEDV, LVESV, and RWMA (P-value >0.05).The incidence of ostial lesions, the affected segment of the culprit vessel, the diseased vessels number, and the incidence of 2 and 3 vessel disease were statistically higher in group I (P-value < 0.001). Also, patients of group I had significant higher thrombus burden grade (5) than those of group II (P-value < 0.001). On the other hand, there was insignificant difference between both groups regarding culprit vessel. Significantly, the majority of patients of group I underwent rescue PCI compared with patients of group II (86.4% Vs 17.4%), also the majority of patients of group II had routine early PCI compared with patients of group I (82.6% vs 13.6%) with P-value < 0.001. The incidence of patients who underwent thrombus aspiration was statistically higher in group I (P-value <0.001). Also, patients of group I had to get 2 stents more than those of group II (P-value=0.002). Moreover, the incidence of patients who received intra-coronary tirofiban was statistically higher in group I (P-value < 0.001). **Table 3**

For univariate analysis, presence of HTN, DM, hyperlipidemia, History of CAD,higher duration from symptoms onset till Thrombolytic, lower SBP, lower DBP, higher RBG, increased ALT, lower LVEF, increased number of vessels affected, presence of ostial segment affection, high thrombus Burden Grade was associated with increased occurrence risk of TIMI 2 flow or less. Using backward wald regression analysis to choose the best model of predictors of TIMI 2 flow or less, revealed that one hour increase in duration from symptoms onset till Thrombolytic was associated with 2.63 likelihood of occurrence of TIMI 2 flow or less. Also, high RBG, low LVEF, increased number of affected vessels, presence of ostial segment affection, high thrombus Burden Grade was associated with increased occurrence risk of TIMI 2 flow or less. **Table 4**

Regarding TIMI flow before and After PCI, the proportion of patients who achieved TIMI grade 3 had significantly

increased from 66.2% before the intervention to 69.2% after.

Table 1: Demographics, risk factors, physical examination and Klipp classification of the studied patients

	Variables		Group I (n=44, 33.8%)	Group II (n=86, 66.2%)	Total participants (n=130)	p-value
Demographics data	**Age (years)**		50.5 (44.0-59.0)	47.0 (42.0-57.3)	48.0 (42.0-58.0)	0.333ǂ
	Gender	**Males**	30 (68.2)	66 (76.7)	96 (73.8)	0.293ǂǂ
		Females	14 (31.8)	20 (23.3)	34 (26.2)	
Risk factors	**HTN**		38 (86.4)	43 (50.0)	81 (62.3)	**<0.001*** ǂ
	DM		39 (88.6)	27 (31.4)	66 (50.8)	**<0.001*** ǂ
	Hyperlipidaemia		27 (60.4)	24 (27.9)	51 (39.2)	**<0.001*** ǂ
	Family history		8 (18.2)	21 (24.4)	29 (22.3)	0.419 ǂ
	History of CAD		24 (54.5)	25 (29.1)	49 (37.7)	**0.005*** ǂ
	Smoking		26 (59.1)	60 (69.8)	86 (66.2)	0.223 ǂ
	Stroke		6 (13.6)	6 (7.0)	12 (9.2)	0.336 ǂǂ
	Duration from symptoms onset till Thrombolytic (hours)		8.0 (4.0-9.0)	4.0 (3.0-6.0)	5.0 (3.0-7.3)	**<0.001***ǂǂǂ
Physical examination	**SBP (mmHg)**		100.0 (90.0-110.0)	110.0 (100.0-120.0)	110.0 (100.0-120.0)	**<0.001*** ǂ
	DBP (mmHg)		60.0 (60.0-70.0)	70.0 (70.0-80.0)	70.0 (60.0-80.0)	**<0.001*** ǂ
	HR (Bpm)		96.5 (65.0-107.0)	90.0 (85.0-95.0)	90.0 (80.0-98.0)	**0.034*** ǂ
	RR		17.0 (16.0-20.0)	18.0 (17.0-19.0)	18.0 (16.0-19.0)	0.571 ǂ
	So_2 (%)		95.0 (91.3-95.8)	95.0 (95.0-96.0)	95.0 (94.0-96.0)	0.181 ǂ
Klipp Classification	**Killip class I**		20 (45.5)	62 (72.1)	82 (63.1)	**<0.001*** ǂǂ
	Killip class II		7 (15.9)	24 (27.6)	31 (23.8)	
	Killip class III		9 (20.5)	0 (0.0)	9 (6.9)	
	Killip class IV		8 (18.2)	0 (0.0)	8 (6.2)	

Data was presented as median (IQR) or frequency (%). *: statistically significant P value <0.05. IQR: Interquartile range (25th – 75th percentile), ǂMan-Whitney U test, ǂǂChi-square. DM: Diabetes Mellitus, HF: Heart failure. HTN: Hypertension, CAD: coronary artery disease, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: Heart Failure, RR: respiratory rate, So_2: Sulfur Dioxide.

Table 2: Diagnosis, cardiac biomarkers and lab data among the studied patients

	Variables	Group I (n=44, 33.8%)	Group II (n=86, 66.2%)	Total participants (n=130)	P-value
Diagnosis	**Anterior STEMI**	24 (54.5)	47 (54.7)	71 (54.6)	0.991ǂ
	Inferior STEMI	17 (38.6)	34 (39.5)	51 (39.2)	0.921ǂ
	Lateral STEMI	2 (4.5)	2 (2.3)	4 (3.1)	0.604ǂǂ
	Posterior STEMI	1 (2.3)	3 (3.5)	4 (3.1)	1.0ǂǂ
Cardiac biomarkers	**Peak CKMB (U/L)**	173.0 (110.0-220.0)	115.0 (85.5-175.0)	130.0 (88.0-190.8)	**<0.001*** ǂ
	Troponin (ng/L)	1.0 (0.8-1.2)	0.7 (0.3-0.9)	0.8 (0.4-1.0)	**<0.001*** ǂ
Lab data	**RBG (mg/dl)**	389.0 (263.0-440.0)	121.0 (104.5-225.5)	175.0 (110.0-332.0)	**<0.001*** ǂ
	Sodium (mmol/L)	136.0 (133.0-138.0)	137.0 (135.0-139.0)	137.0 (134.0-139.0)	0.103 ǂ
	Potassium (mmol/L)	3.5 (3.1-4.0)	3.6 (3.2-3.9)	3.6 (3.2-3.9)	0.724 ǂ
	Urea (mg/dl)	30.5 (19.0-53.0)	29.0 (22.5-38.3)	29.5 (21.0-39.3)	0.771 ǂ
	S. Cr (mg/dl)	1.3 (0.8-1.40)	1.1 (0.9-1.3)	1.1 (0.9-1.3)	0.718 ǂ
	INR	1.2 (1.1-1.3)	1.2 (1.0-1.3)	1.2 (1.1-1.3)	0.167 ǂ
	AST (U/l)	45.0 (36.0-64.0)	40.0 (32.0-63.0)	43.0 (35.0-63.0)	0.251 ǂ
	ALT (U/l)	39.0 (35.0-56.8)	37.5 (32.0-45.0)	38.0 (34.8-45.3)	**0.024*** ǂ

Data was presented as median (IQR) or frequency (%). *: statistically significant P value <0.05. ǂMann-Whitney U test. STEMI: ST-segment elevation myocardial infarction, CKMB: Creatine kinase myocardial band, RBG: random blood glucose, S. Cr: serum creatinine, INR: International normalized ratio, AST: aspartate aminotransferase, ALT: Alanine Aminotransferase.

Table 3: Echo parameters, thrombus and affected vessels parameters in coronary angiography and intervention parameters among the studied patients

	Variables	Group I (n=44, 33.8%)	Group II (n=86, 66.2%)	Total participants (n=130)	p-value
Echo parameters	**LVEF (%)**	36.0 (32.0-41.5)	43.5 (40.8-47.0)	42.0 (38.0-45.0)	**<0.001*** ⊦
	LVEDV (mm)	50.0 (46.0-53.0)	49.0 (48.0-52.0)	50.0 (48.0-53.0)	0.554 ⊦
	LVESD (mm)	40.0 (36.0-42.0)	39.0 (35.0-41.0)	39.0 (36.0-41.0)	0.395 ⊦
	Presence of RWMA	44 (100.0)	83 (96.5)	127 (97.7)	0.323 ⊦⊦
Thrombus and affected vessels parameters	**0**	0 (0.0)	1 (1.1)	1 (0.8)	**<0.001***⊦⊦
	1	10 (22.7)	60 (69.7)	70 (53.8)	
	2	13 (29.5)	14 (16.2)	27 (20.9)	
	3	21 (47.7)	11 (12.7)	32 (24.8)	
	Culprit Vessel (IRA)				
	LAD	25 (56.8)	46 (54.1)	71 (55.0)	0.770 ⊦
	LCX	4 (6.8)	11 (12.9)	14 (10.9)	0.379⊦⊦
	RCA	15 (34.1)	28 (32.9)	43 (33.3)	0.896⊦
	Segment				
	Ostial	14 (31.8)	6 (7.1)	20 (15.5)	**<0.001***⊦
	Proximal	19 (43.2)	37 (43.5)	56 (43.4	0.970 ⊦
	Mid-segment	18 (40.9)	42 (49.4)	60 (46.5)	0.359 ⊦
	Distal	1 (2.3)	1 (1.2)	2 (1.6)	1.0 ⊦⊦
	Thrombus Burden Grade				
	0	0 (0.0)	1 (1.2)	1 (0.8)	**<0.001***⊦⊦
	1	1 (2.3)	20 (23.3)	21 (16.2)	
	2	2 (4.5)	35 (40.7)	37 (28.5)	
	3	4 (9.1)	22 (25.6)	26 (20.0)	
	4	1 (2.3)	1 (1.2)	2 (1.5)	
	5	36 (81.8)	7 (8.1)	43 (33.1)	
Intervention parameters	**Rescue**	38 (86.4)	15 (17.4)	53 (40.8)	**<0.001*** ⊦
	Pharmaco-invasive	6 (13.6)	71 (82.6)	77 (59.2)	**<0.001***⊦
	Thrombus Aspiration	10 (22.7)	1 (1.2)	11 (8.5)	**<0.001***⊦ ⊦
	No. of stents				
	0	0 (0.0)	3 (3.5)	3 (2.3)	**0.002 ***⊦ ⊦
	1	35 (79.5)	80 (93.0)	115 (88.5)	
	2	9 (20.5)	3 (3.5)	12 (9.2)	
	Stent Diameter	3.3 (3.0-3.5)	3.5 (3.0-3.5)	3.5 (3.0-3.5)	0.849⊦⊦⊦
	Stent Length	28.0 (25.0-33.0)	28.0 (24.0-33.0)	28.0 (24.0-33.0)	0.372⊦⊦⊦
	Intra-coronary Tirofiban	41 (93.2)	24 (27.9)	65 (50.0)	**<0.001***⊦

Data was presented as median (IQR) or frequency (%). *: statistically significant P value <0.05. RWMA=Regional wall motion abnormality, ⊦Mann-Whitney U test, ⊦⊦ Fisher exact test. LVEF: Left ventricular ejection fraction, LVEDV: Left ventricular end-diastolic volume, LAD: Left anterior descending, LCX: Left circumflex, RCA: Right coronary artery.

Table 4: Binary logistic regression for predictors of TIMI 2 flow or less

Variables	TIMI 2 flow or less							
	Univariate analysis				Multivariate analysis			
	Crude OR	95% CI		P-value	Adjusted OR	95% CI		P-value
		Lower bound	Higher bound			Lower bound	Higher bound	
HTN (RF=No)	6.33	2.43	16.53	**<0.001***	--	--	--	--
DM (RF=No)	17.04	6.05	48.05	**<0.001***	--	--	--	--
Hyperlipidaemia (RF=No)	4.10	1.90	8.85	**<0.001***	--	--	--	--
History of CAD (RF=No)	2.93	1.38	6.22	**0.005***	--	--	--	--
HF(RF=No)	17.61	3.76	82.54	**<0.001***	--	--	--	--
Duration from symptoms onset till Thrombolytic (hours)	1.59	1.33	1.90	**<0.001***	2.63	1.32	5.22	**0.006***
SBP (mmHg)	0.91	0.88	0.95	**<0.001***	0.95	0.85	1.05	0.306
DBP (mmHg)	0.90	0.86	0.94	**<0.001***	1.12	0.92	1.37	0.269
So2 (%)	1.21	0.998	1.45	0.333	--	--	--	--
RBG (mg/dl)	1.01	1.01	1.02	**<0.001***	1.34	1.12	1.98	**0.045***
ALT (U/L)	1.038	1.012	1.06	**0.003***	1.01	1	1.03	0.145
LVEF (%)	0.768	0.696	0.847	**<0.001***	0.93	0.85	0.96	**0.014***
LVEDV (mm)	1.03	0.94	1.13	0.584	--	--	--	--
LVESD (mm)	0.98	0.89	1.07	0.626	--	--	--	--
No of diseased vessels	3.47	2.12	5.66	**<0.001***	1.64	1.28	9.44	**0.011***
Ostial segment affection (RF=no)	6.22	2.19	17.68	**0.001***	18.63	16.06	62.94	**0.024***
Thrombus burden grade	1.24	1.15	1.36	**<0.001**	9.70	1.27	74.15	**0.029***

Data was presented as frequency (%). *: statistically significant P value <0.05. RF: Reference category, OR: Odds ratio, CI: Confidence interval. Backward Wald method was used. HTN: hypertension, DM: Diabetes Mellitus, CAD: Coronary artery disease, HF: Heart Failure, SBP: systolic blood pressure, DBP: diastolic blood pressure, So2: Sulfur Dioxide, RBG: random blood glucose, ALT: Alanine Aminotransferase, LVEF: Left ventricular ejection fraction, LVEDV: Left ventricular end-diastolic volume, LVESD: Left Ventricular End-Systolic Dimension.

Case study

58-year-old male with past medical history of diabetes & hypertension. He is heavy smoker, presented to A&E complaining of severe agonizing chest pain of 4 hours duration.

- **Clinical examination:**

BP: 110/70 Pulse: 90beats/min
So2: 98% on room air
Auscultation: Normal Heart sounds S1 & S2 with clear chest

- **Laboratory investigations at A&E:**

Hb: 12.7 g/dl TLC: 9.8 k/ul Platlets: 265 k/ul
INR: 1
ALT: 12U/L AST: 15 U/L
CK MB: 87 U/L
Troponin: 0.4 ng/L
Creatinine: 0.9 mg/dl Urea: 23 mg/dl

- **ECG**: Sinus Rhythm with ST segment elevation at leads (II, III and avF) and with reciprocal ST segment depression at leads I & avL. **(Figure 1).**
- **Bedside Echocardiography**: showed preserved LV systolic function 67%, with RWMA in the form of hypokinetic basal inferior and basal posterior walls.
- Patient received loading dose of Aspirin 300mg & Clopidogrel 300 mg then contraindications for thrombolytic therapy were excluded and patient received Streptokinase 1.5 million units IV infusion over an hour.
- **New ECG** was done 90 minutes post thrombolytics showed resolution of >50% of ST segment elevation. **(Figure 2).**
- Then within 24 hours patient was transferred to catheterization laboratory, coronary angiography was done

showing atherosclerotic left coronary system with non-specific tight lesions or stenosis but Right Coronary Artery (RCA) showed midsegment long significant lesion followed by thrombus containing tight lesion with TIMI III flow.

Pre-dilatation was done using Mini Trek 2x20mm balloon, Then PCI was done by 2 DES deployed at site of lesion (Promus Elite 3.5x28mm DES was deployed distally & Promus Elite 3.5x33mm DES was deployed overlapping and proximal to the 1st stent) with TIMI III flow end result.**(Figures 3 & 4).**

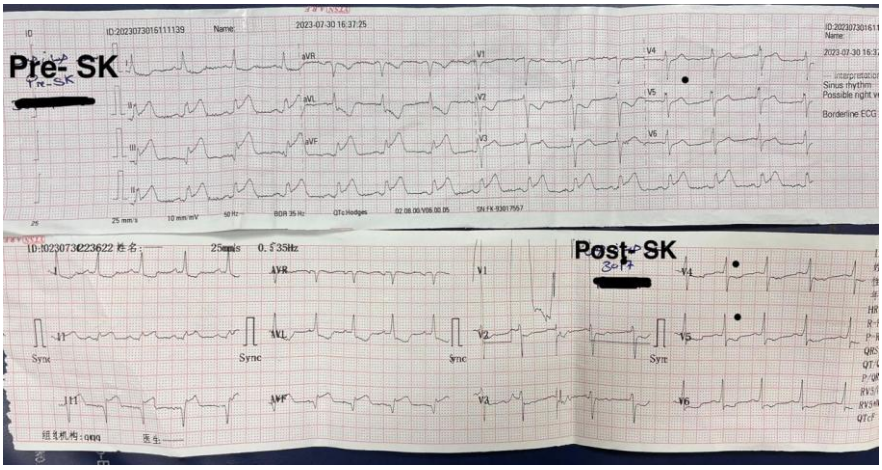

Figure 1: Pre-thrombolytic ECG showing ST segment elevation at leads II, III, avF & Post-thrombolytic ECG showing Resolution >50% of ST segment elevation at inferior leads

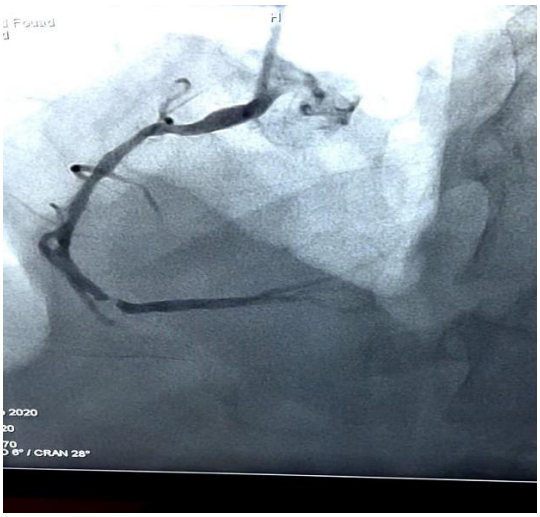

Figure 2: Right Coronary Artery (RCA) showing proximal significant lesion & Distal significant lesion containing thrombi

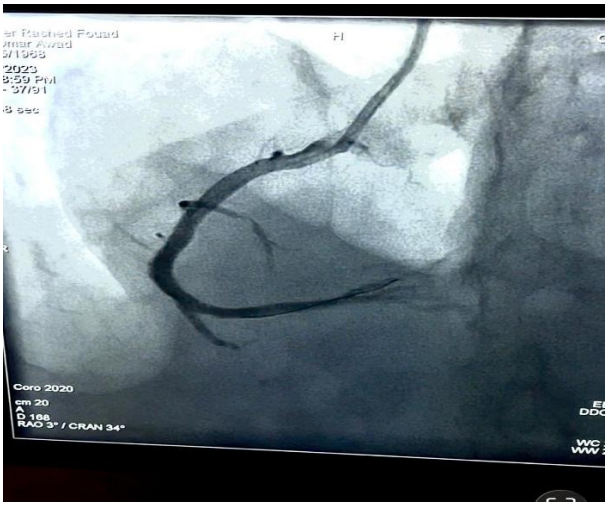

Figure 3: RCA after PCI by 2 DES with TIMI III flow

Discussion

To enhance prognosis of patients with STEMI PCI has been demonstrated in comparison to thrombolysis, predominantly as a result of the restoration of angiographic TIMI III flow grade in a significant number of patients. Mechanical devices and adjunctive pharmacological agents further bolster this enhancement [21].

In this study, the incidence of STEMI patients with impaired TIMI flow (≤ 2) post thrombolytic therapy was 33.8%.

This was concordant with Raghuram at al., [22] detected that the incidence of patient with TIMI 0, 1 or 2 was 44%.

In the current study, DM, HTN, dyslipidemia, and a history of CAD were all statistically significantly more common (P-value 0.05). However, the two patient groups didn't show any statistically significant difference in terms of age or gender (P-value > 0.05).

This was consistent with Bianco et al., [23] found insignificant difference between patients with ST-segment reduction and those without ST-segment reduction as a result of age and male gender (P-value > 0.05). Additionally, the investigation revealed that patients who did not undergo St-segment reduction in STEMI patients exhibited dyslipidemia, a higher prevalence of HTN, DM, and a history of CAD (P-value < 0.05).

Again, Raghuram at al., [22], Patients with TIMI flow 3 and those with TIMI flow ≤ 2 did not exhibit a significant difference in relation to age and gender (P-value < 0.05).

Our research demonstrated that patients with TIMI flow ≤ 2 experienced a significantly higher median duration from the onset of symptoms to the receipt of thrombolytic therapy (P-value < 0.05).

This was concordant with Raghuram et al., [22] who determined that the mean total ischemic time (hours) and the mean duration of chest pain (hours) were statistically significantly higher in patients with impaired TIMI flow (5.1+/- 2.5 Vs. 4.4 +/- 24 & 5.7 +/- 2.6 Vs. 4.9 +/- 2.5; respectively with P-value < 0.05).

In this study, it was demonstrated that patients with impaired TIMI flow exhibited a lower median SPB and DBP (P-value <0.001), a higher heart rate (P-value=0.03), and a higher incidence of Killip class III and IV (P-value < 0.001). On the other hand, the two groups that conducted the analysis did not demonstrate a statistically significant difference in the location of the STEMI (P-value > 0.05).

This was concordant with Salih Kilic et al., [24], who showed that SBP & DBP were statistically different among the studied groups (P-value 0.002 & 0.001; respectively). Also, Biancov., et al., (23) revealed that patients without ST-segment

reduction had high heart rate (80 (68-95) Vs. 75 (66-88) bpm, P-value < 0.001).

Again, revealed that patients with thrombolysis failure had higher incidence of Killip class II-IV than those with thrombolysis success {12 patients (25.5%) vs. 7 patients (10.3%), p value = 0.03}. They also detected insignificant difference regarding anterior and non-anterior wall MI (p value= 0.351).

Patients who experienced impaired TIMI flow were classified as "Group I" in our investigation. They demonstrated a significantly higher median troponin level than "Group II" (1.0 vs. 0.7; respectively, p value < 0.001).

This was concordant with Prabhakaran et al., [25], who demonstrated that a significantly lower rate of reperfusion after thrombolysis was associated with positive troponin results on admission (P-value <0.05).

Patients with impaired TIMI flow exhibited a significant increase in the incidence of 2 & 3 vessel disease, ostial lesions, and a higher thrombus burden in comparison to those with TIMI flow 3 (P-value < 0.05). Based on the angiographic data, this was demonstrated.

This was concordant with Manikandan et al., [26], who detected that patients with failed thrombolysis had high incidence of multi-vessel affection that those with successful thrombolysis (26 patients (43.3%) Vs. 1 patient (1.1%), P-value < 0.001).

This was in line with Kumar et al., [19], This study determined that prevalence of multi-vessel disease was statistically significantly higher in patients with TIMI flow < 3 (19 patients (50%) vs. 2 patients (2.7%), P-value < 0.001).

The study's limitations were characterized by a relatively small sample size which decreased statistical power of analysis. Additionally, the results were less generalizable due to the dual-center design. Coronary artery characterization was conducted using angiography, but no modern visualization techniques, such as intravascular ultrasound, were employed.

Conclusion

Thrombolytic therapy was administered to 33.8% of patients diagnosed with STEMI who had a TIMI flow grade of less than 3. An elevated risk of TIMI flow < 3 is associated with the presence of ostial segment lesions, multi-vessel affection, and thrombus burden grade. Additionally, patients who have demonstrated a history of coronary artery disease , diabetes mellitus and hypertension . A higher incidence of TIMI flow <3 was associated with a prolonged period from the onset of symptoms to thrombolysis, as well as a lower LVEF, systolic, and diastolic blood pressure.

Therefore, research suggests that screening for risk factors prior to the initiation of thrombolysis can accelerate a development of alternative treatment methods that decrease the chance of failure and redirect resources to more effective treatment options. Additionally, the study suggests that conducting additional multi-center studies with larger sample sizes can yield more precise results.

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Conflicts of interest

No conflicts of interest

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