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ORIGINAL ARTICLE

Rate and Determinants of Slow Flow / No-Reflow in Patients Undergoing Primary Percutaneous Coronary Intervention at Sandaman Provincial Hospital Quetta

ABDUL WADOOD KAKAR[1], ANIL KUMAR[2], JAVED KHURSHED SHAIKH[3], MUHAMMAD HASHIM KALWAR[4], MUHAMMAD HASSAN BUTT[5], NADEEM HASSAN RIZVI[6]

[1]MBBS, FCPS (Interventional Cardiology) Assistant Professor /HOD Cardiology Loralai Medical College Loralai /Sandeman Provincial Hospital Quetta
[2]MBBS, FCPS (Interventional Cardiology) Assistant Professor Postgraduate Medical Institute /Bolan Medical Complex Hospital Quetta
[3]FCPS (Cardiology) Assistant Professor National Institute of Cardiovascular Diseases(NICVD) Sukkur
[4]FCPS (Cardiology) Assistant Professor National Institute of Cardiovascular Diseases(NICVD) Sukkur
[5]FCPS (Cardiology) Assistant Professor National Institute of Cardiovascular Diseases(NICVD) Sukkur
[6]MBBS, DIP, CARD, MRCP, FRCP, FACC, FAIC Professor of Interventional Cardiology National Institute of Cardiovascular Diseases(NICVD) Karachi
Correspondence to: Abdul Wadood Kakar, Email:abdulwadoodkakar227@gmail.com, Cell: 03118305162

ABSTRACT

Objective: We investigated the rate of slow/no flow during percutaneous coronary intervention, the clinical and angiographical predictor and the immediate hemodynamic role of slow / no flow.

Material & Method: The cross-sectional study was done at Sandaman Provincial Hospital, the Loralai Medical Collage Loralai, Bolan University of Medical and Health Sciences Quetta for six months from 1st July, 2021 to 31st December, 2021. We included ST-elevation myocardial infarction patients who got primary percutaneous coronary intervention (PCI). Patient information, including demographic and clinical data was collected. In this study, thrombolysis in myocardial infarction was used to determine the antegrade flow. There was an evaluation of the existence, predictors, and consequences of slow/no flow in the patients. SPSS 21 was used for data analysis.

Results: Among the 300 patients, 283 (80.9%) were males. There were 54 (18.0%) patients who had angiographic slow/no flow during the procedure. TIMI grades were 0 in 13 (4.33%), 1 in 16 (5.33%), and 2 in 25 (8.33%) patients in these affected groups in the study. Smoking status was significantly different between slow and no flow ($p=0.023$). We found significant associations between prior MI, nonappearance of pre-infarction anginal symptoms, and any cerebrovascular disease with slow/no blood flow ($p<0.05$). The class III or IV Killip score was significantly higher in the slow/low flow group than the normal-flow group ($p<0.05$). Intracoronary adenosine and epinephrine were the most often used medications for pharmacological therapy of no/slow flow. The hemodynamic instability of two of the patients (3.70%) of the ventricular tachycardia treatment (VT) cases led to their deaths, while the stability of two (3.70%) of the patients' VTs required pharmaceutical cardioversion.

Conclusion: The occurrence of slow/no flow can be predicted with a history and angiographical feature.

Keywords: Slow/no flow, Primary PCI, Angiographical predictors, Hemodynamics'.

INTRODUCTION

Performing primary percutaneous coronary intervention (PCI) as soon as feasible after a severe ST-elevation myocardial infarction (STEMI) is the most effective technique to restore antegrade blood flow and minimize myocardial ischemia. Not at all. Reflow is a recognized PCI side effect, and it results in reduced coronary blood flow despite the renovation of the artery's integrity.[12] According to No-reflow occurred in 2.3 % of cases undergoing PCI in a major study of patients with acute MI (AMI).[3] According to, LVD and progressive myocardial damage are more common in patients with no-reflow, and both conditions escalate the risk of cardiac death and morbidity for these individuals. In both animal and human investigations, the no-reflow phenomena have been linked to significant myocardial necrosis, which is well-known to be a strong indicator of death. As [4] put it: As reported in [5], The no-reflow phenomenon is a robust predictor of 5-year death in cases with STEMI treated with primary PCI. Considerably research has been done on no-reflow, however, few studies show how the phenomenon affects blood flow and heart rate immediately. To restore normal flow, characteristics related to a high frequency of no-reflow obligation be predicted and actions made to preclude their incidence. The goal of this research was to determine the rate of no-flow/slow flow in STEMI cases having primary PCI, as well as to identify its analysts and the abrupt effects on hemodynamics and cardiac rhythm in the cases.

MATERIAL AND METHOD

We conducted this cross-sectional study at Sandaman Provincial Hospital, the Loralai Medical Collage Loralai, Bolan University of Medical and Health Sciences Quetta for six months from 1st July, 2021 to 31st December, 2021. The sample size was estimated using the OpenEpi calculator[6] after clearance from the institution's ethical review committee based on a 2.3% predicted prevalence, a 95% confidence level, and a 1.25% margin of error[3] Using a nonprobability sequential sampling approach, the sample was discarded from patients aged 23-70 who had primary PCI for critical STEMI. These individuals did not include those who had undergone saphenous vein graft (SVG) or left internal mammary artery (LIMA) interventions, as well as those who had coronary artery spasms and had been treated conservatively, as well as those who had culprit lesions with less than 50% stenosis and had not undergone any intervention.

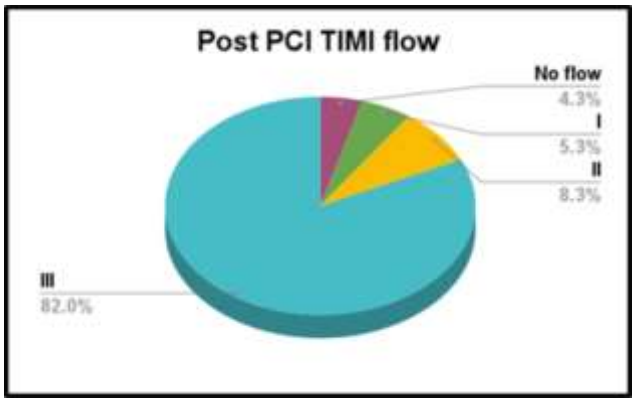
All patients who endured primary percutaneous coronary intervention (PCI) for an acute STEMI were followed up. Following coronary angiography and PCI, all patients were excluded from the study. There were no deviations from usual practice in any treatment procedure. After being admitted to the hospital, all patients without evident contraindications received 300mg of aspirin, 600mg clopidogrel, and 100IU of intravenous (IV) heparin before entering the catheterization (Cath) laboratory. The culprit lesion was identified by angiography, which was conducted either through the right brachial or right femoral channel. Finally, the final coronary angiography showed the ante grade radio-contrast flow of the artery that was associated with the infarction. The operator used the TIMI criterion to determine this flow. In the study of Harrison et al., (2013). Three different levels of TIMI flow are recognized: no antegrade flow beyond the occlusion point, minimal partial perfusion of the dissimilarity average around the clot, partial perfusion of the distal coronary bed with the contrast material, and complete perfusion of the distal coronary bed with the contrast material. TIMI flow was used to analyze the impact of slow or no flow on patients who had been treated with pharmacological intervention under recommendations. The angiographic parameters of 50% stenosis and TIMI III flow in the distal arteries were used to evaluate the final success of the procedure.[3]

SPSS 21 was used to analyze the data. Qualitative factors were quantified using rates and percentages, whereas measureable variables were quantified using means and standard

deviations (SDs). To determine the significance of a connection, chi-square and independent t-tests were used. The significance level was set at P<0.05.

RESULTS

Among the 300 patients, 283 (80.9%) were males. In overall terms, the sample had a mean age of 54.86±10.07 years. 224 (74.66%) patients had hypertension, while 100 (33.33%) had diabetes. There were 54 (18.0%) patients who had angiographic slow/no flow during the procedure, whereas 246 (82.0%) patients had normal flow. TIMI grades were 0 in 13 (4.33%), 1 in 16 (5.33%), and 2 in 25 (8.33%) patients in these affected groups (**Figure**).



In the study, gender and age did not have any statistically significant differences between slow and no flow (p>0.05). Smoking status was significantly different between slow and no flow (p=0.023). Significant associations were found between prior MI, nonappearance of pre-infarction anginal symptoms, and any cerebrovascular disease with slow/no blood flow (p0.05). At presentation, the slow/no flow group had degenerated New York Heart Association (NYHA) classes III and IV in comparison to the normal-flow group, and the class III or IV Killip score was significantly higher in the slow/low flow group than the normal-flow group (p0.05). In the present study, the mean ejection fraction (EF) was 42.94±12.27, and a statistically significant variance was found among the groups (p0.015). We found no statistically significant association with slow/no flow (p>0.05) between systolic or diastolic blood pressure (BP) or pulse rate (**Table 1**).

The coronary angiography findings showed that both groups had MI in the anterior wall, mainly due to the left anterior descending (LAD) artery. There was a significant correlation between total occlusion of the vessel and slow/no flow (p=0.03). Slow/no flow patients also had lower pre-procedural TIMI flows, but the difference didn't statistically differ (p>0.05). Slow/no flow was not significantly linked with the number of coronary arteries affected or the existence of collateral circulation (p>0.05).). More patients in the slow/no flow group had a larger thrombus load (p=0.004) (**Table 2**).

A stent was inserted in 285 (95.0%) of the patients, whereas only 15 (5.0%) had balloon angioplasty performed. There were a total of 287 successful outcomes (95.66 %). Stenting with a bare-metal stent (BMS) showed a decreased risk of developing sluggish or no flow (p<0.001) than balloon angioplasty (p=0.001) (**Table 3**).

Slow/no flow was not associated with the use of Tirofiban, aspiration thrombectomy, or numerous stents (p>0.05). Intracoronary adenosine and epinephrine were the most often used medications for pharmacological therapy of no/slow flow, with 17 (31.48%) and 4 (7.40%) patients receiving each. However, 33 patients (61.11 %) were not provided any medicine. When comparing patients with the normal flow to those with no/slow flow, the ultimate lesion success was considerably higher in normal-flow patients (p<0.05) (**Table 4**).

There were no immediate on-table effects on hemodynamics in patients 18 (33.33%), while 27 (40.0%) experienced abrupt hypotension, and experienced bradyarrhythmia 5 (9.25%). 2(3.70%) patients experienced hemodynamically uneven ventricular tachycardia (VT) that need defibrillation and resulted in death, while 2 (3.70%) cases had stable VT that obligatory pharmaceutical cardioversion (VT) (**Table 5**).

Table-1: Baseline parameters between no-flow and normal-flow patients.

Variables	Total	TIMI flow after procedure		P-value
		Normal flow	No-reflow	
Total	300 (100%)	246 (82.0%)	54 (18.0%)	-
Gender				
Male	238 (79.3%)	194 (78.8%)	44 (81.4%)	0.348
Female	62 (20.6%)	52 (21.13%)	10 (18.51%)	
Age (years)	54.86 ± 10.07	53.59 ± 10.12	57.42 ± 11.25	0.115
Age in Groups				
18 to 40 years	30 (10%)	28 (11.3%)	2 (3.7%)	0.114
41 to 60 years	180 (60%)	145 (58.9%)	35 (64.8%)	
> 60 years	90 (30%)	73 (29.6%)	17 (31.4%)	
Medical history				
Current Smoker	86 (28.66%)	67 (27.23%)	19 (35.18%)	0.023*
Ex-smoker	35 (11.66%)	27 (10.97%)	8 (14.81%)	0.142
Hypertensive	224 (74.66%)	186 (75.60%)	38 (70.37%)	0.478
Diabetes	100 (33.33%)	79 (32.11%)	21 (38.88%)	0.073
Prior Myocardial infarction	58 (19.33%)	39 (15.85%)	19 (35.18%)	<0.001*
Prior Heart Failure	29 (9.66%)	21 (8.53%)	8 (14.81%)	0.038*
Prior PCI	14 (4.66%)	10 (4.06%)	4 (7.40%)	0.051
Absent Pre-infarct Angina	66 (22.0%)	51 (20.732%)	15 (27.77%)	0.007*
Cerebrovascular Disease	5 (1.66%)	3 (1.21%)	2 (3.70%)	0.002*
Pain to percutaneous coronary intervention time (in min)	282.7 ± 456.92	269.28 ± 201.55	449.94 ± 1181.53	0.412
≤ 120 minutes	51 (17.0%)	41 (16.66%)	10 (18.51%)	0.840
121 to 240 minutes	123 (41.0%)	98 (39.38%)	25 (46.29%)	0.402
> 120 minutes	126 (43.0%)	107 (43.49%)	19 (35.18%)	0.487
End-diastolic pressure	42.94 ± 12.27	41.34 ± 12.23	37.09 ± 12.02	0.015*
NYHA class				
I	175 (58.33%)	149 (60.5%)	26 (48.14%)	0.002*
II	106 (35.33%)	85 (34.55%)	21 (38.88%)	

III	16 (5.33%)	10 (4.06%)	6 (11.11%)	
IV	3 (1.0 %)	2 (0.81%)	1 (1.85%)	
SBP (mmHg)	123.3 ± 23.61	123.65 ± 23.04	121.89 ± 28.58	0.271
DBP (mmHg)	76 ± 11.76	76.41 ± 11.33	74.15 ± 16.93	0.077
Pulse Rate (bpm)	84.44 ± 19.37	84.18 ± 19.43	88.91 ± 18.79	0.156
KILLIP class				
I	231 (77.0%)	193 (78.45%)	38 (70.37%)	0.008*
II	52 (17.33%)	40 (16.26%)	12 (22.22%)	
III	13 (4.33%)	10 (4.06%)	3 (5.55%)	
IV	4 (1.33%)	3 (1.21%)	1 (1.58%)	

Table-2: Comparison of angiographical characteristics and post-procedure TIMI flow.

Variables	Total	TIMI flow after procedure		**p-value
		Normal flow	No-reflow	
Total	300 (100%)	246 (82.0%)	54 (18.0%)	-
Infarct Locations				
Anterior	151 (50.33%)	120 (48.78%)	31 (57.40%)	0.040*
Inferior	137 (45.66%)	115 (46.74%)	22 (40.7%)	
Lateral	9 (3.0%)	9 (3.65%)	0 (0%)	
Others	3 (1.0%)	2 (0.81%)	1 (1.85%)	
Culprit Vessel				
Left Main (LM)	4 (1.33%)	3 (1.21%)	1 (1.85%)	0.478
Left anterior descending (LAD)	161 (53.66%)	131 (53.25%)	30 (55.55%)	
Left circumflex artery (LCX)	41 (13.66%)	33 (13.41%)	8 (14.81%)	
Right coronary artery (RCA)	94 (31.33%)	79 (32.11%)	15 (27.77%)	
Number of Coronary Arteries Involved				
Single vessel (SVD)	110 (36.66%)	92 (37.39%)	18 (33.33%)	0.145
Two vessels (2VD)	105 (35.0%)	87 (35.36%)	18 (33.33%)	
Three vessels (3VD)	85 (28.33%)	67 (27.23%)	18 (33.33%)	
Target Lesion Location				
Proximal	129 (43.0%)	101 (41.05%)	28 (51.85%)	0.003*
Mid	160 (53.33%)	137 (55.69%)	23 (42.59.1%)	
Distal	11 (3.66%)	8 (3.25%)	3 (5.55%)	
Type of Occlusion				
Subtotal	142 (47.33%)	122 (49.59%)	20 (37.03%)	0.003*
Total	158 (52.66%)	124 (50.40%)	34 (62.96%)	
Pre Procedure Thrombolysis in myocardial infarction flow				
No flow	166 (55.33%)	130 (52.84%)	36 (66.66%)	0.072
I	100 (33.33%)	88 (35.77%)	12 (22.22%)	
II	31 (10.33%)	25 (10.16%)	6 (11.11%)	
III	3 (1.0%)	3 (1.21%)	0 (0%)	
Pre Percutaneous coronary intervention Thrombus Score				
0-1	40 (13.33%)	33 (13.41%)	7 (12.96%)	0.004*
2	26 (8.66%)	22 (8.94%)	4 (7.40%)	
3	76 (25.33%)	66 (26.82%)	10 (18.51%)	
4	86 (28.66%)	72 (29.26%)	14 (25.92%)	
5	72 (24.0%)	53 (21. 54%)	19 (35.18%)	
Collateral Circulation				
0-1	253 (84.33%)	210 (85.36%)	43 (79.62%)	0.170
2	44(14.66%)	34 (13.82%)	10 (18.51%)	
3	3 (1.0%)	2 (0.81%)	1 (1.85%)	

Table-3: Comparison of procedural characteristics and post-procedure outcomes with post-procedure TIMI flow.

Variables	Total	TIMI flow after procedure		**p-value
		Normal flow	No-reflow	
Total	300 (100%)	246 (82.0%)	54 (18.0%)	-
Type of Intervention				
Stenting	245 (81.66%)	207 (84.14%)	38 (70.37%)	<0.001*
Balloon Angioplasty	15 (5.0%)	7 (2.84%)	8 (14.81%)	
Stenting post Pre-dilation	40 (13.33%)	32 (13.0%)	8 (14.81%)	
Stent type				
Bare metal stents	80 (26.66%)	69 (28.04%)	11 (20.37%)	<0.001*
Drug-eluting stents	206 (68.66%)	171 (69.51%)	35 (64.81%)	
Percutaneous old balloon angioplasty	15 (5.0%)	7 (2.84%)	8 (14.81%)	
Tirofiban Used	225 (75.0%)	184 (74.79%)	41 (75.92%)	0.681
Repeated Balloon Dilatation	224 (74.66%)	186 (75.60%)	38 (70.37%)	0.134
Aspiration Thrombectomy	56 (18.66%)	46 (18.69%)	10 (18.51%)	0.977
Multiple Stents	22 (7.33%)	16 (6.50%)	6 (11.11%)	0.062
Post PCI TIMI flow				
No flow	13 (4.33%)	Nil	13 (24.07%)	<0.001*
I	16 (5.33%)		16 (29.62%)	
II	25 (8.33%)		25 (46.29%)	
III	246 (82.0%)	246 (100%)	0 (0%)	

Table-4: Mediation and outcome.

Variables	Total	TIMI flow after procedure		**p-value
		Normal flow	No-reflow	
Total	300 (100%)	246 (82.0%)	54 (18.0%)	-
Intracoronary Medication				
Adenosine	19 (6.33%)	2 (0.81%)	17 (31.48%)	<0.001*
Epinephrine	4 (1.33%)	Nil	4 (7.40%)	
Sodium Nitroprusside	1 (0.33%)	1 (0.40%)	Nil	
None	276 (90.0%)	243 (98.78%)	33 (61.11%)	
Post Medicating TIMI Flow				
No flow	3 (1.0%)	1 (0.40%)	3 (5.55%)	<0.001*
I	2 (0.66%)	Nil	2 (3.70%)	
II	10 (3.33%)	1 (0.40%)	9 (16.66%)	
III	285 (95.0%)	245 (99.59%)	40 (74.07%)	
Lesion Success	287 (95.66%)	245 (99.59%)	42 (77.77%)	<0.001*

Table-5: Effect of no/slow flow on the hemodynamics of patients.

Variables	TIMI flow after procedure	
	Normal flow	No-reflow
Total	246 (82.0%)	54 (18.0%)
Effect of No flow/Slow re-flow		
None	Nil	18 (33.33%)
HTN		27 (40.0%)
Ventricular tachycardia		2 (3.70%)
Bradyarrythmia		5 (9.25%)
Tachycardia		2 (3.70%)

DISCUSSION

This is consistent with earlier results of 5 to 25% slow/no flow after initial angioplasty for acute STEMI.[7,8] An exceedingly complicated condition known as "slowed/no flow" occurs when the remaining coronary arteries receive insufficient blood flow during PCI despite no obvious angiographical evidence of blockage, spasm, or dissection of the epicardial arteries.[9–11] Myocardial contrast echocardiography (MCE) and cardiac magnetic resonance imaging (CT) are the diagnostic tools I use to make this diagnosis (CMRI).Because of its sensitivity and precision, CMRI is widely acknowledged as the most effective method for determining how much no-re-flow is present in a sample. But they are rarely required, as angiograms are sufficient in most cases.[12,13]

However, other studies show a significant correlation of age >60 years with slow/no flow.1414 For instance, in one study, someone positively associated smoking with slow/no flow, but in the current study, smoking was insignificant.[14] This might be because of the lower mean age of 54.86±10.07 in the present study compared to 59.19±10.25 years in the other study.[14] The current study did not find a significant correlation between age and gender with slow/no flow. The development of slow/no blood flow is related to prior MI, prior heart failure (HF), a high NYHA class, and a low EF.[15] Smoking induces platelet adhesion and a pro-inflammatory response caused by wall changes. One study tested the significance of NT-proBNP levels as a predictor of slow/no flow in acute MI, showing a resilient relationship with the existence of HF. The absence of pre-infarction angina was also related to no-flow in prior studies.[16] Another study aimed to determine pre-procedure levels of N-terminal (NT)-prohormone brain natriuretic peptide (NT-proBNP) as predictors of no-flow and another focused on NT-prohormone (NT-proBNP) levels as predictors of no-flow. There was a strong association between low/slow blood flow and low Killip class III and IV showing severe congestive heart failure (CHF).[17,18] This may occur because of small infarcts that may produce collaterals that may lower the probability of no-flow. Researchers found that slow/no-flow in those with HF is associated with thrombus burden, inflammation, and endothelial dysfunction associated with total occlusion of the culprit artery.[19] They also found a strong correlation between total occlusion of the culprit artery and endothelial dysfunction. Because of plaque rupture and fissuring, a microvascular embolism can occur spontaneously in acute coronary syndrome (ACS).[20] While direct stenting and BMS use reduced the risk of no/slow blood flow, use of tirofiban, aspiration thrombectomy, and multiple stenting did not have any significant associations, according to the results of the current study.[21,22] I have found intravascular ultrasonography to be a useful tool for assessing the risk of no/low flow in patients with acute MI. Adenosine and epinephrine are the most commonly used medications for treating slow or no blood flow. However, sodium nitroprusside and verapamil were never used intracoronarily. Research has shown that both substances may be effective intracoronary. Evidence from the trial also revealed that tirofiban administered with primary PCI after 600mg of clopidogrel resulted in better results and reduced bleeding at 30 days and one year without altering main outcomes.[23] Evidence also demonstrated that tirofiban had no effect on main outcomes at 30 days or 1 year when compared to placebo.[22] Amistad II (AMISTAD-II) was designed especially to investigate the impact of adenosine in STEMI,[24] but no change in the primary endpoints of new CHF, rehospitalization for CHF, or death from any cause at 6 months was found. The use of customised balloons and localised adenosine instillation can successfully minimise noreflow.[25] Epinephrine has also been shown to help patients with STEMI who are experiencing refractory no-flow, however it does produce a significant but controllable rise in heart rate.[24] The clinical appearance of the no-reflow phenomenon varies dramatically depending on the therapeutic setting. The clinical manifestation of no-reflow during on-table short-term treatments is typically more rapid and apparent. The patient is experiencing chest pain due to a blockage in the coronary artery, which may lead to hemodynamic compromise. It is possible that atheroembolism and a drop in blood flow in the non-culprit arteries are to blame for the sudden change in haemodynamics.[242526]Following severe hypotension and bradyarrhythmias, including blockages, the most prevalent acute effects of slow/no flow were discovered in the current investigation. Many people died from VT even though it is extremely rare. There are a number of drawbacks to the existing research. First, a non-randomized study was conducted in a single location with a smaller number of participants. There was no specific testing for glycated hemoglobin (HbA1c) or fasting lipid profile, for example, but instead depended on patient history. Angiogram TIMI score was solely utilized in this study in order to determine blood flow and perfusion, however other more specialized procedures such as myocardial blush and ST segment resolution might have also been utilized in the investigation. Another explanation for the reduced frequency of slow/no flow may be due to primary angioplasty's high prevalence of tirofiban usage and thrombus aspiration I.

CONCLUSION

Only 18.0% of initial PCI patients had slow or no flow. Slow or no blood flow might be predicted by several factors, including current smoking, past MI, lack of pre-infarction angina, and a history of vascular disease. There was a considerable correlation between the patient's NYHA and Killip class as well as poor EF and slow/no flow. Angiographically, thrombus load and complete vessel blockage were strong indicators of poor prognosis. Direct stenting and the application of BMS lessened the likelihood of little or no

flow. adenosine was the most commonly prescribed drug, and it had the largest impact on blood pressure by causing hypotension

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