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ORIGINAL ARTICLE

Delayed Percutaneous Coronary Intervention Versus Conventional Medical Treatment for Management of Late Comers with Anterior ST Segment Elevation Myocardial Infarction

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Abstract

Background: **ST-segment elevation myocardial infarction (STEMI) is now treated using reperfusion treatments that attempt to minimize infarct size and maximize patient prognosis.**

Aim of the study: **To evaluate the in-hospital and three-month outcomes of a late invasive treatment strategy versus a traditional medical therapy strategy in patients with anterior STEMI who present within 48−72 h of symptom start.**

Materials and methods: **The current study was conducted at the cardiology department of the national heart institute and comprised 60 patients who presented to the emergency department with late anterior ST segment elevation myocardial infarction between 48 and 72 h after symptom onset. The patients were randomly divided (closed envelop method) into two groups: Interventional Group (A): 30 patients who underwent primary PCI and medical Group (B): 30 patients treated with conventional medical therapy.**

Results: **No significant distinction was detected between both groups regarding in-hospital MACE, ejection fraction, CKMB serial levels, MACE and ejection fraction and MPI (LAD territory) at three months follow-up.**

Conclusion: **A late invasive treatment method for patients presenting between 48- and 72-h following symptom onset with anterior STEMI is not superior to a conventional medical treatment strategy and has similar efficacy and short-term outcomes.**

Keywords: Myocardial infarction, Percutaneous intervention, Revascularization

1. Introduction

After sudden cardiac death, the most serious condition of acute coronary syndromes (ACS) is ST-segment elevation myocardial infarction (STEMI). The NRMI-4 revealed that 29% of infarction patients had STEMI[1]; however, the EHS-ACS-II (Second Euro Heart Survey on Acute Coronary Syndromes) discovered that 47% of ACS patients had STEMI.[2]

STEMI is linked with a significant risk of morbidity and death, although it is known that prompt reperfusion greatly improves patient outcomes. Up to 40% of patients with STEMI come late after the beginning of symptoms, which decreases their probability of obtaining reperfusion treatment. Controversy has surrounded the relative advantages of reperfusion treatment after 24 h from the beginning of symptoms over the past two decades. Despite much evidence supporting late reperfusion and the 'late open-artery theory', studies have shown that late reperfusion provides little benefit.[3]

Late presenters at a PCI capable center may be treatment naïve or may be referred from a non-PCI

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capable center after fibrinolysis. Majority of these late comers become pain-free by the time they reach while a few may continue to have symptoms of ischemia. About 20% of patients may have patent IRA owing to phenomenon of 'spontaneous reperfusion' without any form of reperfusion therapy. On the other hand, the efficacy of fibrinolysis in achieving successful reperfusion is far from perfect (50–60%) leaving a substantial number of patients with occluded IRAs. Thus, these late comers are in fact a heterogeneous group including totally asymptomatic to hemodynamically unstable patients, treatment naïve group, or post fibrinolysis, patent IRA versus occluded IRA at the time of presentation; posing a challenge to one size fits all approach.[4]

Clinicians and researchers have shown a great deal of interest in the idea that some of the advantages shown with early opening of the infarct-related artery may be accomplished with later open. Experimental investigations, small clinical trials, and observational studies have supported the late open-artery theory, which posits that the late open of blocked infarct-related arteries following acute myocardial infarction can enhance survival, ventricular function, and quality of life.[5] This work aimed to evaluate the in-hospital and three-months outcomes of a late invasive treatment strategy versus a traditional medical therapy strategy in patients with anterior STEMI who present within 48–72 h of symptom start with no history of previous MI and excluded patients with persistent chest pain.

2. Patient and methods

This study was done in the Cardiology Department of the National Heart Institute and included 60 patients who reported to the Emergency Department between 48- and 72-h following symptom start with late anterior ST segment elevation myocardial infarction.

2.1. Inclusion criteria

Anterior STEMI patients arriving within 48–72 h of the onset of chest pain.

2.2. Exclusion criteria

Patients with persistent chest pain, additional kinds of STEMI (Inferior, Lateral, Posterior, and Right), cardiogenic shock, congestive heart failure, electrical instability, and/or pulmonary edema, and inability to cooperate with research protocols were excluded.

All patients were subjected to the following:

History taking: Including sex, age, risk factors profile, time from onset of chest pain to presentation. **Clinical examination:** Heart rate, blood pressure, respiratory rate, presence of fine basal crepitations or lower limb edema and local examination. **ECG:** It was performed before and after PCI (for GROUP A) and daily for all the study population.[6] The type of anterior MI was determined according to the leads showing ST segment elevation or pathological Q waves as follows: Anteroseptal (V1–V3), Anterolateral (I,AVL, V5,V6), Anterior (V2–V4), Extensive anterior (leads I,AVL,V1–V6).[7] **Portable chest- X ray:** For assessment of pulmonary congestion. **Resting echo-Doppler study:** It was performed before discharge and at three months follow-up comparing mainly changes in the left ventricular EF using Philips Echo machine. Both groups' ejection percentage was determined using a modified version of Simpson's technique, and wall motion anomalies were evaluated. **Myocardial perfusion image (MPI) study:** It was performed using Siemens machine at three months follow up comparing myocardial ischemia and viability in both groups. **Routine laboratory investigations and serial cardiac enzymes:** Creatine phosphokinase (CPK), CKMB and Troponin. Cardiac enzymes were withdrawn on presentation, 12 h later and then daily during hospital stay. **Coronary angiography ± PCI to LAD:** Patients allocated to Group A were promptly sent to the catheterization laboratory. Patients in Group B were referred for unscheduled intrusive therapy (PCI) if they suffered hemodynamic and electrical instability, recurring severe angina, severe congestive heart failure and/or mechanical problems, new relevant electrocardiographic abnormalities, pulmonary edema, or re-infarction. The presumed re-infarction following the original myocardial infarction may be misdiagnosed by the initial ECG alterations.[8,9] **TIMI flow grade:** Pre and Post procedure: anterograde coronary flow according to the standard TIMI grade criteria.[10] **Follow-up:** All patients were followed–up one month after release with respect to the following: angina requiring hospitalization, MACE, development of heart failure, major and minor bleeding[11] and then patients were followed up after 3 months as regarding: Echo Doppler study and myocardial perfusion image study.

2.3. Statistical analysis

SPSS V.20 was involved in the statistical analysis. Quantitative data were reported as mean value and standard deviation, whereas qualitative data were expressed as frequency and percentage. Also utilized were the T test, Mann–Whitney test, X^2 (Chi ^{2}),

Table 1. The demographic data of the studied group.

	Total Number = 60
Age (years)	
Mean ± SD	53.77 ± 10.73
Range	32–72
Sex	
Female	20 (33.3%)
Male	40 (66.7%)
DM	
No	21 (35.0%)
Yes	39 (65.0%)
HTN	
No	23 (38.3%)
Yes	37 (61.7%)
Smoking	
No	20 (33.3%)
Yes	40 (66.7%)
FH	
No	45 (75.0%)
Yes	15 (25.0%)
Dyslipidemia	
No	14 (23.3%)
Yes	46 (76.7%)

and Fisher exact test. *P* value less than or equal to 0.05 was considered statistically significant.

3. Results

Ten male patients (66.7%) and twenty female patients (33.3%) participated in the research. Their ages varied from 32 to 72 years, with a mean of 53.77 ± 10.73 years, representing 63% of the population. The study included 39 diabetic patients representing 65% of the study population, HTN (61.7%), smoking (66.7%), FH (25%), and dyslipidemia (76.7%) (Table 1). Regarding demographic statistics, there was no significant difference between the two groups, as shown in (Table 2). (Table 3) demonstrated no substantial difference between both groups regarding vital signs, ECG and time to presentation except maximal ST that was higher in group A than group B with statistically significant difference (Table 4). showed that majority of patients in group A had proximal LAD lesion (70%) with the mean of lesion severity 95.00 ± 6.30. 56.7% had TIMI1 flow grade 0 while 23.3% had TIMI1 flow grade 3 and 83.3% had TIMI2 flow grade 3. 83.3% of patients had no other lesions <50% while 6.7%, 6.7% and 3.3% had LCX, RCA and OM + RCA respectively. 96.7% had DES with majority of patients had no complications while only 6.7% and 3.3% had dissection and failed PCI respectively. The DTB time ranged from 34 to 114 min with the mean 76.80 ± 24.11 min. There was no considerable difference between both groups regarding in hospital course (Table 5). There was no significant difference between both groups regarding in-hospital MACE and ejection fraction (Table 6). There was no significant difference between both groups regarding CKMB serial levels (Table 7). There was no significant difference between both groups regarding MACE and ejection fraction and MPI (LAD territory) at three months follow up (Table 8).

4. Discussion

Regarding to our results there was no statistical considerable difference between the study groups regarding to age and sex. These results were

Table 2. Comparison between different groups regarding demographic data.

	Group A Number = 30	Group B Number = 30	Test value	*P* value	Sig.
Age (years)					
Mean ± SD	53.73 ± 10.95	53.80 ± 10.69	−0.024•	0.981	NS
Range	35–72	32–71			
Sex					
Female	10 (33.3%)	10 (33.3%)	0.000*	1.000	NS
Male	20 (66.7%)	20 (66.7%)			
DM					
No	12 (40.0%)	9 (30.0%)	0.659*	0.417	NS
Yes	18 (60.0%)	21 (70.0%)			
HTN					
No	13 (43.3%)	10 (33.3%)	0.635*	0.426	NS
Yes	17 (56.7%)	20 (66.7%)			
Smoking					
No	10 (33.3%)	10 (33.3%)	0.000*	1.000	NS
Yes	20 (66.7%)	20 (66.7%)			
FH					
No	23 (76.7%)	22 (73.3%)	0.089*	0.766	NS
Yes	7 (23.3%)	8 (26.7%)			
Dyslipidemia					
No	8 (26.7%)	6 (20.0%)	0.373*	0.542	NS
Yes	22 (73.3%)	24 (80.0%)			

Table 3. Comparison between both groups regarding to vital signs, ECG and time to presentation.

Vital signs, ECG and TIME to presentation	Group A Number = 30	Group B Number = 30	Test value	*P* value	Sig.
HR					
Mean ± SD	86.37 ± 11.96	85.67 ± 11.85	0.228•	0.821	NS
Range	66–110	63–116			
SBP					
Mean ± SD	139.07 ± 21.51	143.87 ± 14.74	−1.008•	0.318	NS
Range	90–182	120–170			
DBP					
Mean ± SD	82.80 ± 12.56	84.67 ± 12.16	−0.585•	0.561	NS
Range	56–110	65–117			
Type of MI					
1	8 (26.7%)	8 (26.7%)	0.000*	1.000	NS
2	6 (20.0%)	6 (20.0%)			
3	7 (23.3%)	7 (23.3%)			
4	9 (30.0%)	9 (30.0%)			
Time to presentation (h)					
Mean ± SD	59.53 ± 4.90	60.27 ± 5.55	−0.542•	0.590	NS
Range	50–70	50–70			
Maximal ST (mm)					
Mean ± SD	1.70 ± 0.70	1.37 ± 0.49	2.132•	0.037	S
Range	1–3	1–2			

comparable with **Boden** et al.[12] who found that there was no statistically significant difference between the study groups regarding to age and gender.

Table 4. Angiography and PCI data among group A.

ANGIO + PCI	Group A Number = 30
LAD lesion	
Proximal	21 (70.0%)
MID	8 (26.7%)
Distal	1 (3.3%)
Lesion severity	
Mean ± SD	95.00 ± 6.30
Range	80–100
TIMI1	
0	17 (56.7%)
1	3 (10.0%)
2	3 (10.0%)
3	7 (23.3%)
TIMI2	
0	1 (3.3%)
2	4 (13.3%)
3	25 (83.3%)
Other lesions >50%	
No	25 (83.3%)
LCX	2 (6.7%)
RCA	2 (6.7%)
OM + RCA	1 (3.3%)
Stent	
No	1 (3.3%)
DES	29 (96.7%)
Complication	
No	27 (90.0%)
Dissection	2 (6.7%)
Failed	1 (3.3%)
DTB (min)	
Mean ± SD	76.80 ± 24.11
Range	34–114

Our study showed that there was no statistical significant difference regarding to HR, SBP, DBP and type of MI while there was significant difference regarding maximal ST. In concordance with our results, **Mancini** et al.[13] assessed the clinical outcomes utilizing revascularization and aggressive drug evaluation showed that there was statistical significant difference regarding to maximal ST segment between the study groups.

Regarding to our results there was no statistical significant difference regarding to in-hospital course. In agreement with our results, **Murphy,**[14] who assessed the clinical outcomes utilizing revascularization and aggressive drug evaluation showed that there was no statistical significant difference between both groups regarding to death, MI, stroke and hospitalization.

Similarly, in the DECOPI (DEsobstruction COronaire en Post-Infarctus) study, in which 212 asymptomatic patients presenting 2–15 days after symptom onset with Q-wave on ECG were randomized to PCI or medical therapy, even though invasive treatment was associated with significant improvement in the left ventricular ejection fraction, there was no benefit at 34 months of follow up in terms of cardiac death, nonfatal MI, or ventricular tachyarrhythmia Steg P.G. et al.[15] Enrollment at the earliest possible time after a myocardial infarction was connected to an increase in event rates, but there was no linkage between randomization time and treatment impact. The absence of impact with PCI was also observed when time was examined as a categorical variable with early (3 days) and intermediate (7 days) cutoffs. This research verifies the

Table 5. Comparison between both groups regarding to in-hospital course.

In-hospital course	Group A Number = 30	Group B Number = 30	Test value	*P* value	Sig.
ST segment resolution					
No	13 (43.3%)	17 (56.7%)	1.067*	0.302	NS
Yes	17 (56.7%)	13 (43.3%)			
Peak CK-MB level					
Mean ± SD	89.40 ± 19.09	95.03 ± 18.22	−1.169•	0.247	NS
Range	63–150	65–144			
Recurrent chest pain					
No	28 (93.3%)	27 (90.0%)	0.218*	0.640	NS
Yes	2 (6.7%)	3 (10.0%)			
Need for urgent intervention					
No	27 (90.0%)	26 (86.7%)	0.162*	0.688	NS
Yes	3 (10.0%)	4 (13.3%)			
Arrhythmia					
No	27 (90.0%)	26 (86.7%)	0.162*	0.688	NS
Yes	3 (10.0%)	4 (13.3%)			

parent trial's therapeutic applicability for the management of stable patients with verified total occlusion during the whole 24-h to 28-day study enrolment period. Additionally, **Cantor** et al.[16] shown that protocol-assigned PCI had no effect for OAT patients with mild-to-moderate ischemia in the IRA area.

Existing clinical practice guidelines say that PCI can be safely postponed in patients with late entrants if aggressive, comprehensive medical treatment is initiated and maintained. Our data support these guidelines. The majority of late-arriving patients can get appropriate medical therapy without regular PCI as an initial management strategy.[17]

Regarding left ventricular function, volume, and clinical outcomes, small-scale, randomized studies of PCI versus therapeutic studies for complete blockage in the myocardial infarction subacute phase have yielded contradictory findings, ranging from benefit to damage for each objective. Three out of four studies indicate that re-infarction rates in the PCI group are 1.5–3.5 times higher than in the medical treatment group.[15] At one year, the angiographic ancillary substudy revealed that the ejection percent was comparable between the two groups. In a subset of patients for whom volume measures were available, PCI seemed to predict a slightly lesser rise in left ventricular volume. The potential advantage of

Table 6. Comparison between both groups regarding in-hospital MACE and ejection fraction.

In-hospital course	Group A Number = 30	Group B Number = 30	Test value	*P* value	Sig.
H. failure					
No	10 (33.3%)	9 (30.0%)	0.077*	0.781	NS
Yes	20 (66.7%)	21 (70.0%)			
Minor bleeding					
No	27 (90.0%)	29 (96.7%)	1.071*	0.301	NS
Yes	3 (10.0%)	1 (3.3%)			
Acute kidney injury					
No	30 (100.0%)	30 (100.0%)	–	–	–
Yes	0 (0.0%)	0 (0.0%)			
Major bleeding					
No	29 (96.7%)	30 (100.0%)	1.017*	0.313	NS
Yes	1 (3.3%)	0 (0.0%)			
Stroke					
No	30 (100.0%)	30 (100.0%)	–	–	–
Yes	0 (0.0%)	0 (0.0%)			
Death					
No	29 (96.7%)	29 (96.7%)	0.000*	1.000	NS
Yes	1 (3.3%)	1 (3.3%)			
Ejection fraction					
Mean ± SD	36.63 ± 7.31	36.27 ± 5.36	0.222•	0.825	NS
Range	25–54	26–46			

Table 7. Comparison between both groups regarding CKMB serial levels.

CKMB serial levels	Group A Number = 30	Group B Number = 30	Test value	*P* value	Sig.
CKMB 0					
Mean ± SD	108.17 ± 43.68	96.13 ± 38.53	1.132•	0.262	NS
Range	59–177	57–175			
CKMB 1					
Mean ± SD	95.03 ± 32.02	93.20 ± 21.63	0.260•	0.796	NS
Range	49–164	58–150			
CKMB 2					
Mean ± SD	75.03 ± 28.66	72.40 ± 25.46	0.376•	0.708	NS
Range	40–150	43–145			
CKMB 3					
Mean ± SD	53.45 ± 20.79	52.07 ± 16.77	0.275•	0.785	NS
Range	23–98	29–84			

Table 8. Comparison between both groups regarding MACE, ejection fraction and MPI (LAD territory) at three months follow up.

Three months follow-up	Group A Number = 29	Group B Number = 28	Test value	*P* value	Sig.
H. failure					
No	12 (41.4%)	10 (35.7%)	0.193*	0.661	NS
Yes	17 (58.6%)	18 (64.3%)			
Stroke					
No	29 (100.0%)	28 (100.0%)	–	–	–
Yes	0 (0.0%)	0 (0.0%)			
Repeated hospitalization					
No	28 (96.6%)	26 (92.9%)	0.390*	0.532	NS
Yes	1 (3.4%)	2 (7.1%)			
Death					
No	29 (96.7%)	28 (93.3%)	0.351*	0.554	NS
Yes	1 (3.3%)	2 (6.7%)			
Ejection fraction					
Mean ± SD	38.00 ± 7.73	34.32 ± 7.25	1.852•	0.069	NS
Range	25–55	18–46			
MPI (LAD territory)					
Scar	17 (58.6%)	19 (67.9%)	0.522*	0.470	NS
Viable	12 (41.4%)	9 (32.1%)			

reduced left ventricular remodeling may be offset by an increase in nonfatal reinfarctions.[18]

4.1. Conclusion

A late invasive treatment strategy in patients with anterior STEMI, presenting between 48 and 72 h after symptom onset, is not superior to a conventional medical treatment strategy and has similar efficacy and short-term outcomes.

Conflicts of Interest

None declared.

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