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Comparative Study between Primary Percutaneous Coronary Intervention, Pharmacoinvasive Strategy and Pharmacological Reperfusion Strategy in Acute Myocardial Infarction. A Long-Term Follow-Up Analysis

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ABSTRACT

Background: Until now there is no clear evidence to support the superiority of one strategy over the others to treat STEMI patients. Serial B-type natriuretic peptide (BNP) measurements accurately predict the risk of death or congestive heart failure in STEMI patients.

Objective: The aim of the study was to determine which strategy is the best strategy to treat acute STEMI and if BNP give an incremental prognostic value in treated STEMI patients. Patients presented with acute STEMI were enrolled in the present study.

Patients and methods: Only 93 patients were followed up with us for 6 months and divided to 3 groups according to the treatment strategy. All patient underwent BNP analysis, echocardiography and treated with either received only thrombolytic therapy (Group I), primary PCI (Group II) or received thrombolytic therapy then went to catheterization (Group III).

Results: Regarding BNP level change was higher in Group I and in Group III had high BNP level higher than Group II but this difference doesn`t reach level for statistically significant correlation between BNP levels and type of reperfusion in the three groups. Regarding LV systolic function, there was no statistically difference between the percent change in LV systolic function in Group I and Group III (P-value = 0.854, 0.152 respectively) but there was statistically significant difference between the percent change in LV systolic function in Group II (P-value=0.031)**.**

Conclusion: The best way for treatment of acute STEMI patients, is a primary PCI the most significant predictive methods are recovery of EF% and reduction of BNP, however pharmacoinavsive strategy like PCI strategy has good follow up prognosis and better than thrombolytic alone therapy.

Keywords: PCI, ST-segment elevation myocardial infarction, Pharmacoinvasive strategy, Pharmacological reperfusion strategy.

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) accounts for 25-40% of acute coronary syndrome (ACS) cases [(1)]**.** Re-establishing prompt coronary blood flow and myocardial tissue perfusion as quickly as possible remains the most important principle underlying early STEMI management [(2)]**.**

Although primary PCI is the preferred reperfusion method for STEMI, it remains difficult to implement in areas, and fibrinolytic therapy is still widely used but rather as part of a pharmacoinvasive strategy, with the patients brought to PCI-capable facilities after fibrinolysis, to perform semi-urgent coronary angiography [(3)]**.** Plasma levels of these neurohormones may thus reflect not only existing left ventricular systolic dysfunction but in addition be a sensitive index of abnormal wall stress preceding the process of ventricular remodeling [(4)]**.**

The aim of the present study was to determine which strategy is the best method to treat acute ST-segment elevation myocardial infarction (STEMI) and if BNP can predict the clinical outcome in acute STEMI treated by different modalities.

PATIENTS AND METHODS

This prospective study enrolled 93 consecutive patients admitted to Coronary Care Units of Zagazig University and Police Authority Hospital with acute ST segment elevation myocardial infarction. The study was performed in the period from March 2016 to March 2020. Institutional review board and medical ethics committee approved the study and written informed consent was taken from all participants.

Patients were divided into three groups:

Group I: including the patients who received thrombolytic therapy alone.

Group II: including the patients who had primary percutaneous coronary intervention (PCI).

Group III: (pharmacoinvasive group) including the patients who received thrombolytic therapy followed by PCI (either as rescue PCI or scheduled PCI).

Inclusion criteria: All symptomatic patients presented with acute STEMI with Killip class I or II are eligible for inclusion in the study.

Exclusion criteria: Severe valvular disease, cardiomyopathy, any form of congenital cardiac disease, and symptoms of chronic heart failure (HF).

Complete history was taken included age, gender, special habits of medical importance, and risk factors such as diabetes mellitus, hypertension, hypercholesterolemia and smoking. Also, clinical examination such as heart rate and blood pressure & manifestation of heart failure. Standard 12-lead

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electrocardiograms were performed at presentation, after reperfusion therapy then daily till discharge and the following data were obtained: Location of MI, number of leads with ST-segment elevation, sum of ST-segment elevation (sum STE).

Measurement of plasma level of BNP:

Blood samples were taken for BNP measurement at two to five days (early phase) and at six months (long term) after symptom onset. According to **Choi *et al.*** [5] who measured consecutive BNP levels after primary PCI showed a biphasic peak elevation during follow up. Early phase (2-5 days after STEMI) plasma BNP level was an independent predictor of post-myocardial infarction remodeling in patients with STEMI [5].

Cardiac enzymes:

Initial samples were checked at presentation. For plasma troponin-I was measured by immunofluorescence assay manufactured by Dade-Behring. The analytic sensitivity of the assay is 0.1 ng/ml and the upper normal limit for the diagnosis of acute myocardial infarction was considered to be 1.0 ng/ml:

- Routine laboratory investigations (blood sample for analysis of high sensitivity troponin and kidney function tests).
- Echocardiography study.

Echocardiographic examinations and data were obtained using a commercially available imaging system (Vivid 7; GE Medical Systems, Milwaukee, WI, USA). It was performed on admission, after reperfusion therapy and after 6 months. All examinations included standard parasternal and apical views following the recommendations of the American Society of Echocardiography. All measurements were determined as means of 3 cycles avoiding post ectopic beats. The following were calculated:

- M-Mode measurements: measurements were done in long and short parasternal axis views including IVST (interventricular systolic thickness), PWT (posterior wall thickness), LVEDD (Left ventricular end diastolic diameter) and LVESD (Left ventricular end systolic diameter).
- Left Ventricular End Systolic and End Diastolic Dimensions: End diastole was defined as the frame with the largest cavity area that is correlated with ECG at the beginning of QRS complex and end systole as the subsequent frame with the smallest cavity area that is correlated with ECG at the end of the T wave.
- Ejection Fraction by 2D: calculated using biplane method in apical four and two chamber views.
- Diastolic Function: Mitral inflow pattern was assessed by pulsed Doppler with a sample volume between the tips of the mitral leaflets during diastole. Diastolic dysfunction was reported as: Grade I (impaired relaxation pattern): E /A ratio < 1, Grade II (pseudo normalization pattern): E/A ratio of 1 to 1.5 that became reversed by Valsalva maneuver, Grade III (reversible restrictive pattern): E/A > 2.0 that became reversed by Valsalva maneuver, Grade IV (irreversible restrictive pattern): E/A > 2.0 that not reversed by Valsalva maneuver

Treatment strategy:

Thrombolytic therapy:

1- **Alteplase:** Most of the patients treated with Alteplase, which was administered in an accelerated infusion (1.5 hrs.) using 50-mg and 100-mg vials reconstituted with sterile water to 1 mg/ml. Accelerated infusion of alteplase for AMI consists of a 15-mg IV bolus followed by 0.75 mg/kg (up to 50 mg) IV over 30 minutes and then 0.5 mg/kg (up to 35 mg) IV over 60 minutes. The maximum total dose is 100 mg for patients weighing more than 67 kg.

2- **Streptokinase:** The adult dose of streptokinase for AMI is 1.5 million U in 50 mL of 5% dextrose in water (D5W) given IV over 60 minutes. Allergic reactions force the termination of many infusions before a therapeutic dose can be administered.

Angiography and PCI:

Coronary angiography was performed by transfemoral approach or trans radial approach. Patients received 300 mg of aspirin and 600 mg of clopidogrel or 180 mg of ticagrelor before the procedure. They also received 3000-5000 IU heparin bolus after arterial sheath placement and additional 3000-5000 IU heparin before the PCI (total of 100 IU/kg).

After visualizing of left and right coronary artery, provisional stenting of culprit lesion on infarct related artery was done by standard techniques. After the procedure, all patients were admitted to coronary care unit, received aspirin 100 mg and clopidogrel 150 mg for 2 weeks then 75 mg daily and other standard concomitant therapy (beta blockers, ACE inhibitors, and statins) depending on clinical indications [6]. Thrombus aspiration: Based on these data and the results of a recent meta-analysis, routine thrombus aspiration is not recommended, but in cases of large residual thrombus burden after opening the vessel with a guide wire or a balloon, thrombus aspiration may be considered [7].

Usage of glycoprotein IIb/IIIa inhibitor, intravenous tirofiban administration was selectively used, with 0.4 μg/kg per min for 30 min. In high thrombus burden lesions or in case of no re-flow phenomenon, tirofiban was intracoronary administrated with bolus dose of 15 μg/kg, followed by intravenous administration with maintenance dose of 0.1 μg/kg per min for 24–48 h. Intracoronary nitrates administration

was recommended to obtain maximal epicardial vessel vasodilation.

Pharmacoinvasive strategy:

Following initiation of lytic therapy, patients were transferred to a PCI center, in cases of failure of fibrinolysis, or if there is evidence of re-occlusion or reinfarction with recurrence of ST-segment elevation, immediate angiography and rescue PCI is indicated. Success of re-vascularization was defined as residual stenosis < 30% and if coronary flow in the culprit vessel after primary PCI resulted in thrombolysis in myocardial infarction (TIMI) grade ≥ 2.

Follow-up: (1) Clinical follow up for: any manifestations of heart failure (Pulmonary venous congestion, S3 gallop...), death, non-fatal reinfarction, revascularization. (2) Echocardiography to estimate EF after 6 months follow up. (3) BNP level was measured after 6 months follow up.

End points: Primary end point at 6 months after the myocardial infarction was the reduction in ejection fraction with or without clinical signs and symptoms of heart failure, death, shock or reinfarction. Angiography and PCI.

Ethical consent:

An approval of the study was obtained from Zagazig University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of participation in the study. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis

The collected data were coded, processed and analyzed using the SPSS (Statistical Package for Social Sciences) version 22 for Windows® (IBM SPSS Inc, Chicago, IL, USA). Data were tested for normal distribution using Shapiro Walk test. Qualitative data were represented as frequencies and relative percentages. Chi square test (χ^2) to calculate difference between two or more groups of qualitative variables. Quantitative data were expressed as mean ± SD. Independent samples t-test was used to compare between two independent groups of normally distributed variables (parametric data). The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the P-value ≤0.05 was considered significant. P-value ≤0.001 was considered as highly significant.

RESULTS

Patient s demographic and clinical data

In Group (I) age ranged from 37 to 75 years with a mean value of 58.16 ± 9.83 years, 25 male patients (80.6 %) and 6 female patients (19.4%). In group (II) age ranged from 30 to 73 years with a mean value of 55.74 ± 9.94 years, 30 male patients (96.8 %) and 1 female patients (3.2%) and in group (III) age ranged from 38 to 76 years with a mean value of 55.65 ± 9.41 years **,** 28 male patients (90.3 %) and 3 female patients (9.7%)**.** Statistical analysis showed non-significant difference between the three groups (Table 1).

Table (1): Demographic data distribution of the study groups

Demographic data	Group I	Group II	Group III	Test value	p-value	Multiple comparison		
						P1	P2	P3
Age (years)								
Mean ± SD	58.16±9.83	55.74±9.94	55.65±9.41	0.666	0.517	0.330	0.311	0.969
Range	37-75	30-73	38-76					
Sex								
Female	6 (19.4%)	1 (3.2%)	3 (9.7%)	4.258	0.119	0.054	0.279	0.301
Male	25 (80.6%)	30 (96.8%)	28 (90.3%)					

P1: p-value between group I and group II; P2: p-value between group II and group III; P3: p-value between group I and group III.
Group I: thrombolytic therapy alone group, Group II: primary PCI group, and Group III: pharmacoinvasive group.

Risk factors

In Group (I), there were 13 smokers (41.9 % of the group), 17 hypertensive patients (54.8 % of the group), 17 diabetic patients (54.8 % of the group), 12 dyslipidemic patients (38.7% of the group), 4 patients with previous history of CVD (12.9 % of the group). In Group (II), there were 16 smokers (51.6 % of the group), 20 hypertensive patients (64.5 % of the group),18 diabetic patients (58.1 % of the group), 20 dyslipidemic patients (64.5 % of the group), 10 patients with previous history of CVD (32.3 % of the group). In Group (III), there were 18 smokers (58.1 % of the group), 18 hypertensive patients (58.1 % of the group), 18 diabetic patients had diabetes (58.1 % of the group), 17 dyslipidemic patients (54.8 % of the group), 7 patients with previous history of CVD (22.6 % of the group). Statistically there was no significant difference between the three groups (Table 2).

Table (2): Risk factors distribution of the study group

Risk factors	Group I	Group II	Group III	Test value	p-value	Multiple comparison		
						P1	P2	P3
Smoking	13 (41.9%)	16 (51.6%)	18 (58.1%)	1.635	0.442	0.445	0.204	0.610
Hypertension	17 (54.8%)	20 (64.5%)	18 (58.1%)	0.623	0.732	0.437	0.798	0.602
Diabetes mellitus	17 (54.8%)	18 (58.1%)	18 (58.1%)	0.088	0.957	0.798	0.798	1.000
Dyslipidemia	12 (38.7%)	20 (64.5%)	17 (54.8%)	4.227	0.121	0.042	0.203	0.437
Previous history of CVD	4 (12.9%)	10 (32.3%)	7 (22.6%)	3.321	0.190	0.068	0.319	0.393

P1: p-value between group I and group II; P2: p-value between group II and group III; P3: p-value between group I and group III

Difference in BNP level at admission and after 6 months follow up

In group (I), statistical analysis showed statistically non-significant difference between BNP level on admission and BNP level after 6 months with P- value =0.860. In Group (II), statistical analysis showed statistically significant difference between BNP level on admission and BNP level after 6 months with P- value =0.005. In Group (III), statistical analysis showed statistically non-significant difference between BNP level on admission and BNP level after 6 months with P- value =0.134 (Table 3).

Table (3): Differences in BNP levels in each treatment modalities

BNP	Group I	Group II	Group III
BNP (Pg/ml) At admission			
Mean ± SD	174.48± 136.01	170.48± 128.00	173.13± 119.94
Range	51-718	18-567	12-592
BNP (Pg/ml) After 6 months			
Mean ± SD	172.23± 165.03	125.06± 100.65	151.39± 141.29
Range	30-789	20-450	10-813
Wilcoxon Signed Ranks Test			
z-test	-0.176	-2.793	-1.499
p-value	0.860	0.005*	0.134

Comparison between Echocardiographic parameters between discharge and after 6 months.

In group (I), patients had mean LVEDD of 46.77 ± 6.31 mm before discharge and 47.84 ± 7.45 mm after 6 months (p-value = 0.245), mean LVESD was 31.74 ± 7.23 mm before discharge and 32.90 ± 7.75 mm after 6 months (p-value = 0.390), mean LVEDV of 97.87 ± 33.12 ml before discharge and 101.94 ± 35.53 ml after 6 months (p-value = 0.168), mean LVESV of 49.90 ± 18.67 ml before discharge and 53.10 ± 21.14 ml after 6 months (p-value = 0.249).

Mean ejection fraction of 47.23 ± 14.30 % before discharge and 46.87 ± 13.36 % after 6 months (p-value = 0.854).

In group (II), patients had mean LVEDD of 49.13 ± 6.04 mm before discharge and 47.00 ± 6.80 mm after 6 months (p-value = 0.003), mean LVESD of 29.29 ± 6.26 mm before discharge and 26.03 ± 7.34 mm after 6 months (p-value = <0.001) , mean LVEDV of 104.19±35.78 ml before discharge and 97.58 ± 36.58 ml after 6 months (p-value = 0.037), mean LVESV of 50.16 ± 16.85 ml before discharge and 41.58 ± 13.35 ml after 6 months (p-value = <0.001), mean Ejection Fraction of 50.16 ± 11.28 % before discharge and 54.23 ± 10.92 % after 6 months (p-value = 0.031).

Statistical analysis showed significant difference between LVEDD, LVESD, LVEDV, LVESV and EF before discharge and after 6 months with (p– value = 0.003, p– value =<0.001, p– value= 0.037, p– value= <0.001, p– value = 0.031) respectively (Table 4).

Table (4): Comparison between echocardiographic parameters between discharge and after 6 months in group II

Echo finding	At discharge	After 6 months	ANOVA	p-value
EDD (mm)				
Mean ± SD	49.13±6.04	47.00±6.80	6.497	0.003*
Range	39-64	33-59		
ESD (mm)				
Mean ± SD	29.29±6.26	26.03±7.34	7.312	<0.001**
Range	20-42	13-39		
EDV (ml)				
Mean ± SD	104.19±35.78	97.58±36.58	4.365	0.037*
Range	50-178	52-162		
ESV (ml)				
Mean ± SD	50.16±16.85	41.58±13.35	8.198	<0.001**
Range	18-88	21-73		
EF%				
Mean ± SD	50.16±11.28	54.23±10.92	-4.516	0.031*
Range	23-64	23-69		
DD				
I	30 (96.8%)	29 (93.5%)	0.522	0.770
II	1 (3.2%)	2 (6.5%)		

EDD: End-diastolic dimension, ESD: end-systolic dimension, EDV: End diastolic volume, ESV: end systolic volume, EF: Ejection fraction, DD: diastolic dysfunction.

In group (III), patients had mean LVEDD of 49. 49.10 ± 6.48 mm before discharge and 48.90 ± 5.42 mm after 6 months (p-value = 0.810), mean LVESD of 30.61 ± 6.87 mm before discharge and 28.94 ± 5.12 mm after 6 months (p-value = 0.048), mean LVEDV of 110.77 ± 37.38 ml before discharge and 109.81 ± 32.05 ml after 6 months (p-value = 0.801), mean LVESV of 56.03 ± 19.23 ml before discharge and 52.61 ± 13.23 ml after 6 months (p-value = 0.223).

Mean ejection fraction of 47.81 ± 11.57 % before discharge and 50.13 ± 9.53 % after 6 months (p-value = 0.152). Statistical analysis showed significant difference between LVESD before discharge and after 6 months with p– value = 0.048 but there was no statistically significant difference in other parameters (Table 5).

Table (5): Comparison between Echocardiographic parameters between discharge and after 6 months in group III

Echo finding	At discharge	After 6 months	ANOVA	p-value
EDD (mm)				
Mean ± SD	49.10±6.48	48.90±5.42	0.484	0.810
Range	41-66	40-64		
ESD (mm)				
Mean ± SD	30.61±6.87	28.94±5.12	3.939	0.048*
Range	18-45	20-38		
EDV (ml)				
Mean ± SD	110.77±37.38	109.81±32.05	0.509	0.801
Range	60-210	60-180		
ESV (ml)				
Mean ± SD	56.03±19.23	52.61±13.23	2.491	0.223
Range	21-100	32-93		
EF%				
Mean ± SD	47.81±11.57	50.13±9.53	-2.937	0.152
Range	28-73	28-65		
DD				
I	29 (93.5%)	27 (87.1%)	1.094	0.579
II	2 (6.5%)	4 (12.9%)		

EDD: End-diastolic dimension, ESD: end-systolic dimension, EDV: End diastolic volume, ESV: end systolic volume, EF: Ejection fraction, DD: diastolic dysfunction.

DISCUSSION

In our study, we found that BNP level at admission and after 6 months were higher in patients treated by thrombolytic and patients who underwent pharmaco-invasive therapy had high BNP level, which are higher than those who underwent primary PCI but this difference did not reach level for statistically significant correlation between BNP levels and type of reperfusion in the three groups.

This disagrees with **Kurt *et al.*** [8], who studied 86 patients with STEMI and 80 patients with NSTEMI patients. Hs-CRP and BNP were measured and TIMI risk index was calculated in all patients. Coronary angiography was performed in all patients for principally determining TIMI flow rate. They found that who were treated by streptokinase in acute ST segment elevation myocardial infarction had statistically

significant higher BNP levels than those who were treated by primary PCI.

Regarding LV systolic function, we a found that there was no statistically difference between the percent change in LV systolic function in group I and group III where with P-value = 0.854, 0.152 respectively but there was statistically significant difference between the percent change in LV systolic function in group II where P-value=0.031.

Also, we found significant reduction in proportion of patients presented with systolic LV dysfunction in patients who treated with primary PCI, p-value=0.049 and patients who treated with pharmaco-invasive strategy, p-value =0.038 but there was no significant reduction in patients who were treated with thrombolytic therapy alone (p-value = 0.625). This agrees with **Tycińska *et al.* [9],** who evaluate the long-term prognostic value of a single measurement of plasma BNP in low-risk patients with first ST-elevation myocardial infarction (STEMI). Plasma BNP concentrations were analyzed on admission in 211 patients admitted with first STEMI and treated with primary percutaneous coronary intervention (PPCI). Left ventricular ejection fraction (LVEF) was assessed by echocardiography during the first 24 hours. They found that patients with the highest BNP levels had longer time of chest pain, higher TIMI risk score and the lowest LVEF. Likewise, **Manola *et al.* [10]** assessed the concentration of B-type natriuretic peptide (BNP) as a predictor of heart failure in patients with acute ST elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (PCI) with successful and complete revascularization. Only patients with acute STEMI undergoing primary PCI who had single vessel disease and were successfully revascularized were included in the study. They found that group with reduced ejection fraction had higher mean values of BNP at 0, 24 hours, and 7 days with significant difference at 24 hours and 7 days.

In our study we found that there was highly negative correlation between BNP level at admission and ejection fraction after six months in the three groups with (P-value < 0.001 in the three groups). This agrees with **Dilić *et al.* [11],** who evaluated brain natriuretic peptide (BNP) release in acute myocardial infarction (AMI), and found statistically significant real negative correlation ($p<0.05$) between BNP concentration and left ventricle ejection fraction (LVEF) with high correlation coefficient ($r=-0.684$). Likewise, **Fazlinezhad *et al.* [12]** measured plasma BNP level for 42 consecutive patients with acute ST elevation myocardial infarction and 42 healthy, age- and gender-matched subjects. They found that there was significant reverse association between BNP level and EF ($P = 0.006$, $r = -0.47$).

CONCLUSION

There was significant reduction in proportion of patients presented with systolic LV dysfunction in patients who were treated with primary PCI and patients who were treated with pharmaco-invasive strategy. But there was no significant reduction in patients who were treated with thrombolytic therapy alone. There was highly negative correlation between BNP level at admission and ejection fraction after six months in the three groups. The best cut off value of BNP after 24 hours in Discrimination of LV systolic dysfunction (EF ≤ 40), which was >170 pg/ml, with sensitivity of 83.3%, specificity of 60% positive predictive value of 53.3%, negative predictive value of 93.7% and diagnostic accuracy of 70.8 %

RECOMMENDATIONS

- Primary PCI is still the gold slandered therapy of acute STEMI, however a pharmacoinvasive strategy including thrombolysis and routine non-immediate angioplasty represents a widely available and logistically attractive approach that yields identical short-term echocardiography left ventricular outcome.
- Monitoring of BNP and LVEF can give an idea about follow up outcome.
- BNP guide treatment before clinical heart failure (BNP guided treatment) in the form of afterload reduction medication or medication to improve myocardial infarction healing.
- Nevertheless, further studies with rigorous design, large sample size and multiregional cooperation are required to make more accurate cut off value.
- Long follow-up period to assess the clinical outcomes of the included patients is recommended.

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