

Influence of thrombolytic therapy on early and long term left ventricular systolic function in patients with ST-segment elevation myocardial infarction

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Abstract

Background and objectives: Left ventricular systolic dysfunction complicating acute myocardial infarction are responsible for significant morbidity and mortality. This study to assess the effect of thrombolytic therapy on the left ventricular systolic function during the first week and a year after ST- elevation myocardial infarction.

Methods: In a study of 56 patients with first ST-Segment elevation myocardial infarction (42 men and 14 women; age range, 42-89 years (mean, 61 ± 10), who had been admitted to the Coronary Care Unit at Hawler Teaching Hospital from May 2008 to May 2009. study done to assess the left ventricular systolic function during the first week and first year after ST-elevation myocardial infarction.

Twenty one patients received tissue plasminogen activator (Alteplase) 12 hour after the onset of symptoms labeled as group-I, the remaining patients (35), had no chance to receive thrombolytic therapy labeled as group II.

Results: In group I the mean ejection fraction improved significantly from 51.6 ± 9.4 during the first week to 55.14 ± 11 , $P=0.034$, at first year after acute myocardial infarction, while in group II there was no significant difference of mean ejection fraction during the first week (45.97 ± 12.2) and first year of acute myocardial infarction (46.1 ± 13.2), $P=0.5$.

Conclusions: Thrombolytic therapy has a beneficial effect on left ventricular systolic function detected by echocardiography at the end of the first year of ST-elevation myocardial infarction

Key words: Left ventricular systolic function .

Introduction

Left ventricular systolic dysfunction was considered to be one of the most important predictors of outcome after acute myocardial infarction.¹ A curvilinear correlation between left ventricular ejection fraction after acute myocardial infarction and survival has been demonstrated for patients in both the prethrombolytic and thrombolytic eras². Despite significant therapeutic advances ,survivors of acute myocardial infarction are at increased risk of development of heart failure, ventricular remodeling ,and death in the weeks ,months, and

years that follow. Left and right ventricular function assessed immediately after acute myocardial infarction have been identified as important prognostic factors for mortality development of heart failure, the prognostic value of late evaluation of left ventricular and right ventricular function after MI remains unknown³. Left ventricular systolic dysfunction complicating ST-segment elevation myocardial infarction (STEMI) are responsible for significant morbidity and mortality.

The aim of the present study was to assess the effect of thrombolytic therapy on the left ventricular systolic function during

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the first week and at the end of first year of STEMI.

Methods

A total number of 163 consecutive patients with first STEMI admitted to the Coronary Care Unit (CCU) at Hawler Teaching Hospital from May 2008 to May 2009. Fifty six patients had full two-dimensional echocardiographic study during the first week and at the end of the first year of STEMI. The later represents the final study population. They were 42 men and 14 women; age range, 42-89 years (mean, 61 ± 10). Acute myocardial infarction was diagnosed if a patient had persistent chest pain lasting for > 30 minutes and had ST-segment elevation in two anterior leads or ST-segment elevation >1 mm in limb leads⁴. Twenty one patients received tissue-type plasminogen activator (Alteplase) with in 12 hour onset of symptoms of acute myocardial infarction, those patients were labeled as group-I, the remaining patients (35), had no chance to receive Alteplase either because of contraindications⁵, or because they presented to the CCU beyond the time limit of thrombolytic therapy⁶, were labeled as group II, as shown in (Figure1).

All patients received oral aspirin 100-325 mg/day and either intravenous or subcutaneous heparin for at least three days, angiotensin converting enzyme inhibitors, statines and other anti-ischemic medications. Patients with left bundle branch block, previous history of heart failure, previous cardiomyopathy, those with permanent pacemaker, previous history of valvular heart disease and those with poor echo window were excluded from this study. The following data were recorded: gender, age, history of diabetes mellitus, hypertension, smoking status, alcohol consumption, family history of ischemic heart disease in the first relative, obesity (defined as body mass index >30)⁷ and time elapsed from onset of symptoms to thrombolytic therapy. Complete two-dimensional and Doppler echocardiography was performed within the first week of

admission to the CCU and at the end of the first year of myocardial infarction, in order to determine the left ventricular systolic function and left ventricular wall motion abnormalities. All echocardiographic examinations were performed with Philips Machine (Envisor USA), using a 2.5MHz transducer. Left ventricular ejection fraction (EF) was determined from apical and four chamber view using the Simpson's biplane formula⁸, left ventricular systolic dysfunction (LVSD) defined as left ventricular ejection fraction $\leq 50\%$ ⁹. Left ventricular segmental wall motion abnormalities was graded as follows: Normal at rest(1); hypokinetic-marked reduction in endocardial motion and systolic thickening (2), akinetic-virtual absence of inward motion and systolic thickening (3), and dyskinetic-paradoxical wall motion away from centre of the left ventricle in systole (4)¹⁰. All possible transthoracic echocardiographic views were obtained for each patient, including parasternal long axis, parasternal short axis, apical four chamber, two chamber and apical long axis view to check for the presence of hypokinesia, akinesia and dyskinesia.

Statistical methods:

t test used for the differences of proportion between the different groups and p value < 0.05 was considered as significant, SPSS version 16.0 (statistical package for social sciences) computer system by assistant of expert statistics.

Results

The study included 56 patients with first STEMI (42 men; 14 women; mean age 61 ± 10), The baseline characteristics of the patients in the two groups are presented in (Table 1). group -I patients had lower average age than group II. There was no significant difference regarding the gender, hypertension, diabetes mellitus, current smoking, obesity, previous angina and the family history of ischemic heart disease between the two patients groups (Table 1). There was no significant difference in the location of the first myocardial infarction between the two group, as shown

in (Table 2).

The mean ejection fraction at the end of seven days of STEMI was significantly higher in group I (51.6±9.4) as compared with group II (45.97±12.2), P=0.04 as shown in (Table 3).

The mean ejection fraction at the end of the first year after infarction was significantly higher in group-I (55.14±11.4) as compared with group-II (46.1±13.2), P=0,006, (Table 4).

In group I the mean ejection fraction improved significantly from 51.6±9.4 during the first week of STEMI to 55.14±11, P=0.034, at the end of the first year after the infarction, while in group II there was no significant difference of mean ejection fraction during the first week (45.97±12.2), and at the end of first year of STEMI (46.1±13.2), (46.1±13.2), P=0.5, as shown in (Table 5&6).

The proportion of patients with left ventricular regional wall abnormalities was higher but not statistically significant in group II patients at the end of the first week and the first year as (65.7%,45.7% compared with group I (52.4% , 28.6%) ; (Table 6&7).

In group –I ,left ventricular wall motion abnormalities decreased from 11 patients (52.4%) during the first week of STEMI to 6 patients (28.6%), at the end of the first year after the infarction, but the P value was not significant, (Table 9).

In group II ,left ventricular wall motion abnormalities decreased from 23 patients (65.7%) during the first week of STEMI to 16 patients (45.7%), at the end of the first year after the infarction, but the P value was not significant, (Table 10).

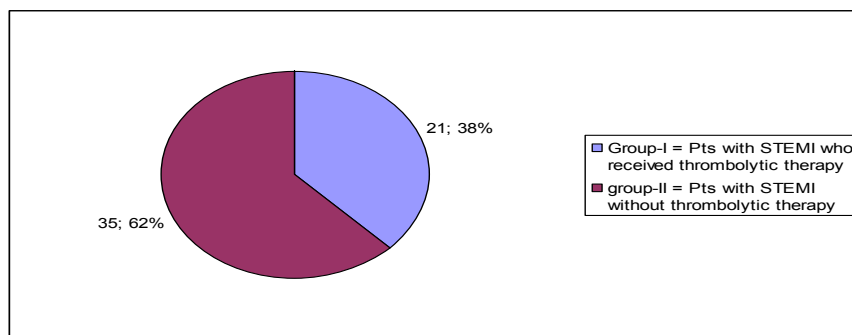


Figure 1: distribution of patients with ST- elevation myocardial infarction according to the thrombolytic therapy

Table 1: Baseline characteristics of the two groups.

	Group-I No=21	Group-II No=35	P
Men	18	24	0.15
Age (year)	55.9±8.2	64.4±9.6	0.002*
Previous angina	4	8	0.74
Hypertension	6	19	0.06
Diabetes mellitus	4	11	0.31
Current smoking	15	17	0.09
Obesity	11	14	0.4
Family history of IHD	4	4	0.4

Group-1= patients with STEMI who received thrombolytic therapy

Group-2= patients with STEMI without thrombolytic therapy

*=P value significant

Table 2: Comparison between the two patient groups according to the site of myocardial infarction.

Site of acute myocardial infarction	Group-I No=21	Group-II No=35	P
Anterior STEMI	15	24	0.82
Inferior STEMI	6	11	0.8

Group-1= patients with STEMI who received thrombolytic therapy

Group-2= patients with STEMI without thrombolytic therapy thrombolytic therapy

Table 3: Comparison of mean ejection fraction between group I and II patient at the end of first week of ST-elevation myocardial infarction.

Mean EF% (group-I patients) No=21	Mean EF% (group-II patients) No=35	P
51.6%(±9.4)	45.97%(±12.2)	0.04*

EF= Ejection fraction, Group-1= patients with STEMI who received thrombolytic therapy

Group2= patients with STEMI without thrombolytic therapy thrombolytic therapy

*= P value significant.

Table 4: Comparison of mean ejection fraction between group I and II patients at the end of first year of ST- elevation myocardial infarction

EF= Ejection fraction, Group-1= patients with STEMI who received thrombolytic therapy

Mean EF% (group-I patients) No=21	Mean EF% (group-II patients) No=35	P
55.14%(±11.42)	46.1%(±13.2)	0.006*

Group-2= patients with STEMI without thrombolytic therapy thrombolytic therapy ,

*= P value significant

Table 5: Comparison of mean ejection fraction in group I patients during the first week and at the end of first year of ST-elevation myocardial infarction.

EF= Ejection fraction, Group-1= patients with STEMI who received thrombolytic therapy

Mean EF% (during the first week of STEMI) No=21	Mean EF% (at the end of first year of STEMI) No=21	P
51.6%(±9.4)	55.14%(±11.42)	0.034*

*= p value significant

Table 6: Comparison of mean ejection fraction in group II patients during the first week and at the end of first year of ST-elevation myocardial infarction

EF= Ejection fraction, Group-II= patients with STEMI without thrombolytic therapy

Mean EF% (during the first week of STEMI) No=35	Mean EF% (at the end of first year of STEMI) No=35	P
45.97%(±12.2)	46.1%(±13.2)	0.5

Table 7: Comparison of left ventricular wall motion abnormalities between group I and II patients at the end of first week of ST- elevation myocardial infarction.

LV-WMA (group-I patients) No=21	LV-WMA (group-II patients) No=35	P
11 (52.4%)	23 (65.7%)	0.32

LV-WMA= left ventricular wall motion abnormalities,

Group-1= patients with STEMI who received thrombolytic therapy

Group-2= patients with STEMI without thrombolytic therapy

Table 8: Comparison of left ventricular regional wall abnormalities between group I and II patients at the end of first year of ST- elevation myocardial infarction.

LV-WMA(group-I patients) No=21	LV-WMA (group-II patients) No=35	P
6 (28.6%)	16 (45.7%)	0.2

LV-WMA= left ventricular wall motion abnormalities,

Group-1= patients with STEMI who received thrombolytic therapy

Group-2= patients with STEMI without thrombolytic therapy

Table 9: Comparison of left ventricular wall motion abnormalities in group I patients during the first week and at the end of first year of ST-elevation myocardial infarction.

LV-WMA During first week of STEMI No=21	LV-WMA at end of first year of STEMI No=21	P
11 (52.4%)	6 (28.6%)	0.11

LV-WMA= left ventricular wall motion abnormalities, STEMI=ST-elevation myocardial infarction.

Table 10: Comparison of left ventricular wall motion abnormalities in group II patients during the first week and at the end of first year of ST-elevation myocardial infarction

LV-RWA During first week of STEMI No=35	LV-RWA At the end of first year of STEMI No=35	P
23 (65.7%)	16 (45.7%)	0.09

LV-WMA= left ventricular wall motion abnormalities, STEMI= ST-elevation myocardial infarction

Discussion

The mean ejection fraction at the end of seven days of STEMI was significantly higher in group-I (51.6 ± 9.4) as compared with group II (45.97 ± 12.2), $P = 0.04$ (Table-3), the present results were similar to Schoming *et al*¹¹ study, which showed that patients who received the accelerated t-PA regimen had significantly less depression of regional wall motion in the ischemic zone, have a slightly higher global ejection

fraction and slightly reduced end –systolic volume index following initiation of thrombolytic therapy . In group I the mean ejection fraction improved significantly from 51 ± 9.4 (during the first week of STEMI) to 55.14 ± 11 , $P = 0.034$, at the end of the first year after the infarction, while in group II there was no significant changes of mean ejection fraction during the first week (45.97 ± 12.2 and at the end of first year of myocardial infarction (46.1 ± 13.2), $P = 0.5$, Table (5&6). Rahimtoola suggested that

hibernating myocardium is a state of persistently impaired myocardial and left ventricular function at rest due to reduced coronary blood flow that can be partially or completely restored to normal either by improving blood flow or by reducing oxygen demand¹². After an myocardial infarction left ventricular and / or right ventricular function may be impaired because of several factors including location and extend of myocardial infarction, continuing ischemia ,history of prior ischemic episodes, antecedent cardiac function, and concomitant comorbidities¹³. Myocardial stunning is likely responsible for transient impairment of ventricular function¹⁵, and recovery of ventricular function is related to more aggressive therapeutic strategies¹⁴⁻¹⁶. The incidence of left ventricular wall motion abnormalities during the first week of STEMI was common in group I, 11 (52.4%) & group II 23 (65.7%), Pfeffer et al found that Left ventricle remodelling is a relatively common and unfavorable event occurring after acute myocardial infarction¹⁷.The extent of microvascular damage after reperfusion has been identified as one of the main determinant of this process^{18,19}. In group I & II patients , improvement in left ventricular wall motion abnormalities were recorded for the period from 7 days to the end of the first year after STEMI, but the P value was not significant as shown in table-9&10.Our results are in keeping with Klein *et al* showed that after myocardial infarction ,the recovery of perfusion and wall motion may continue well after the subacute phase²⁰.Several patients exhibit relative hypoperfusion in viable tissue as late as 5 weeks after infarction and progressive improvement of perfusion in the infarcted area is commonly observed between 5 weeks and 7 months²¹. Many factors may play a role in the improvement of left ventricular wall motion abnormalities in this study at the end of the first year of STEMI, one of these factors that most of the patients were on angiotensin converting enzyme inhibitor from the early phase of m

STEMI²²,further studies needed to assess the effect of thrombolytic therapy on the left ventricular wall motion abnormalities in patients with STEMI.

Conclusion

Left ventricular systolic dysfunction during the first week of ST-elevation myocardial infarction in patients who received thrombolytic therapy is not always a bad prognostic sign, the beneficial effect of thrombolytic therapy on left ventricular systolic function can be demonstrated during the first year of ST-elevation myocardial infarction by 2D-echocardiography ,and this explained by the state hebrinated myocardium.

Recommendations:

Further prospective study using weekly echocardiography is recommended to assess the average time for left ventricular systolic function improvement after ST-elevation myocardial infarction.

Limitation of study: Documentation of reperfusion after thrombolytic by angiography, unavailability of radionuclear study in our center for more accurate assessment of left ventricular wall motion abnormalities.

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