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Recovery of regional and global left ventricular systolic function after acute myocardial infarction: a comparative study between emergency percutaneous coronary intervention and fibrinolytic therapy

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Objectives

Quantitative assessment of the recovery of regional and global left ventricular systolic function after reperfusion in acute myocardial infarction.

Background

Left ventricular systolic function is an important predictor of the outcome after acute myocardial infarction.

Patients and methods

The study included 60 patients with first-time acute myocardial infarction: 30 were treated with fibrinolytic therapy (the pharmacological subgroup) and 30 with emergency percutaneous coronary intervention (the invasive subgroup). Evaluation was performed at 1 week and after 30 days by conventional echocardiography, tissue Doppler imaging, and two-dimensional strain (global longitudinal peak systolic strain).

Results

About 47% of the study population was considered as having a significant recovery in systolic function by 1 month (60% of the invasive subgroup and 40% of those who had fibrinolysis). Conventional echo parameters showed an insignificant difference from 1 week to 1 month as well as between the two subgroups. There was a significant improvement in the systolic wave by tissue Doppler from 5 ± 4 cm/s at 1 week to 7 ± 3 cm/s at 1 month, and it was higher in the invasive subgroup compared with the pharmacological subgroup (8 ± 2 vs. 5 ± 2 cm/s, $P = 0.02$). The global longitudinal peak systolic strain showed a significant improvement from $-13.5 \pm 7\%$ at 1 week to $-15 \pm 8\%$ at 1 month. It was better in the invasive group than in the pharmacological group at baseline (-15.2 ± 5 vs. $-11.9 \pm 4\%$, $P = 0.04$). At 1 month, the global longitudinal peak systolic strain improved to -12 ± 4 and $-16 \pm 3\%$ in the pharmacological and the invasive subgroups, respectively ($P = 0.04$).

Conclusion

The global longitudinal peak systolic strain and tissue Doppler parameters detected the recovery of left ventricular systolic function after myocardial infarction. Moreover, better recovery was reported in invasive reperfusion than in the pharmacological group.

Keywords:

acute myocardial infarction, reperfusion, systolic function

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Introduction

Quantification of the left ventricular (LV) systolic function is an important component in the follow-up of patients after acute myocardial infarction (AMI) [1]. Currently, the recommended measurements for echocardiographic quantification of global and regional LV systolic function are the left ventricular ejection fraction (LVEF) and the wall motion score index (WMSI) [2]. Tissue Doppler imaging is a sensitive, noninvasive echocardiographic method that records the velocity of tissue motion within the myocardium. Tissue Doppler imaging has been evaluated in both in-vitro and in-vivo studies, allowing the quantitative assessment of both the global and the regional function of the myocardium [3]. The global longitudinal peak

systolic strain (GLPSS) has recently been introduced as a novel technique to reflect LV systolic function [4]. Automated function imaging has been developed to facilitate the assessment of myocardial strain with speckle-tracking analysis. This technique has been validated as an accurate measurement of LV systolic function in patients after AMI [5]. The importance of LV function after AMI has been studied extensively. However, functional recovery and remodeling after AMI followed by reperfusion remains understood incompletely.

In this comparative study, we assessed quantitatively the recovery of the regional and the global LV systolic function after reperfusion (pharmacological or invasive) in AMI.

Patients and methods

Study design

The study included 60 consecutive patients who were admitted to the Coronary Care Unit at Benha University Hospital (Benha, Egypt) with first-time AMI, during the period from March 2011 to December 2012. Eligible patients were allocated into a prospective, controlled study. Thirty patients were treated with fibrinolytic therapy (pharmacologic reperfusion), using intravenous streptokinase (1.5 million units over 60 min), whereas 30 patients were treated with emergency percutaneous coronary intervention (PCI) (invasive reperfusion) with either primary or rescue PCI. The recovery of both the regional and the global LV systolic function was assessed with the conventional echo Doppler study, tissue Doppler, and the two-dimensional (2D) strain study 1 week after hospital admission and 30 days later. Key inclusion criteria were patients with first-time AMI eligible for reperfusion (pharmacological or invasive), whereas the key exclusion criteria were patients with a history of coronary artery disease, a history of coronary intervention, and patients with AF (interfere with measurement of strain) and cardiogenic shock.

Emergency percutaneous coronary intervention

The procedure was performed according to the standard techniques of PCI. The femoral approach was the standard in all patients using 6–7 Fr sheaths. Diagnostic coronary angiography was performed to detect the target vessel; XB or JL guiding catheters were used for left coronary lesions and a JR guiding catheter for RCA lesions. Aspiration devices and glycoprotein inhibitors were used in patients with a heavy thrombus burden and an impaired TIMI flow grade after PCI. Bare-metal stents were used in all patients. The operator determined the size and the length of the stent. Emergency PCI was performed for the lesion of interest only. The sheath was removed 6 h after the end of the procedure, and compression was performed manually. Follow-up of all patients was carried out during the hospital stay.

Echocardiographic measurements

Patients were imaged in the left lateral decubitus position using a commercially available system (Vivid 7; General Electric, Vingmed, Norway). Images were obtained with a simultaneous ECG signal.

Conventional echo study

2D images were acquired during breath hold and saved in the cine-loop format from three consecutive

beats. The biplane Simpson technique was used to calculate the LV end-systolic volume (ESV), the LV end-diastolic volume, and LVEF. Mitral regurgitation was characterized as mild (jet area/left atrial area <20% and vena contracta width <0.30 cm), moderate (jet area/left atrial area 20–40% and vena contracta width 0.30–0.69 cm), and severe (jet area/left atrial area >40% and vena contracta width ≥0.70 cm) [5]. M-Mode echo included the measurement of the left ventricular dimension in systole (LVIDs) and diastole (LVIDd), the interventricular septum (IVSd, IVSs), posterior wall thickness (PWTd, PWTs), and LVEF%. Pulsed-wave Doppler echo included pulsed-wave Doppler of the mitral valve obtained by placing the Doppler sample volume between the tips of the mitral leaflets. The early (E) and late (A) peak diastolic velocities and the E-wave deceleration time were measured.

Tissue doppler study

By activating the DTI function in the echocardiography machine, the mitral annular velocities were recorded using the pulsed-wave DTI. From the apical four-chamber and two-chamber views, the longitudinal mitral annular velocities were recorded from the septal, the lateral, the inferior, and the anterior LV sites. A mean value for the above four sites were used. Three major velocities were taken into account: the positive peak systolic velocity when the mitral ring moved toward the cardiac apex due to longitudinal contraction of the LV and two negative diastolic velocities when the mitral annulus moved toward the base, away from the apex: one during the early phase of diastole and the second wave in the late phase of diastole. A mean of three consecutive cycles was used to calculate all echo-Doppler parameters [6].

Speckle tracking

Apical four-chamber and two-chamber views and long-axis views were used for the quantification of the peak systolic strain by automated function imaging speckle-tracking analysis. This novel software analyzes motion by tracking frame-to-frame movement of natural acoustic markers on standard ultrasonic images in two dimensions. First, the LV end-systolic frame was defined by determining the closure of the aortic valve in the apical long-axis view. Then, the time interval between the R-wave and aortic valve closure was measured automatically and used as a reference for the four-chamber and the two-chamber views. After defining the mitral annulus and the LV apex with three index points in all three apical views, the LV endocardial border was automatically traced at end-systole, and the region of interest created was adjusted manually to the thickness of the myocardium. The

tracking quality was validated in all segments from the three apical views. The GLPSS for the complete LV provided by the software using a 17-segment model in a 'bull's eye' plot was calculated as the average of the longitudinal peak systolic strain of each view, and criteria of improvement of GLPSS at follow-up were defined as increased GLPSS by at least 10% from the baseline [7].

Statistical analysis

Data are presented as mean $\pm$ SD for normally distributed quantitative data and as the number and percentages for categorical data. The χ^2 -test 'Z' test and the Student 't'-tests were used for between-groups comparison of qualitative and quantitative data, respectively. The paired sample *t*-test was used for within-group comparisons. Binary logistic regression analysis was used to assess predictors of recovery of LV function. The accepted level of significance in this work was stated at *P* less than 0.05. The data collected were tabulated and analyzed using SPSS version 17 (Chicago, IL, USA).

Results

Study population

The mean age of the participants was 57 ± 8 years; 92% of the participants were male; 60% were smokers, 36% had diabetes mellitus, 22% were hypertensive, and 22% had dyslipidemia there was no family history of coronary artery disease. About 82% of them presented with anterior myocardial infarction (MI), whereas 18% had inferior MI. There was no significant difference in the baseline characteristics between the pharmacologic or the invasive groups (Table 1).

Echocardiographic measurements

Conventional echo parameters

At 1 week, the mean WMSI of the study population ($n = 30$) was 1.6 ± 0.5 (1.57 ± 0.6 and 1.58 ± 0.4 for the pharmacological and the invasive groups, respectively; $P = 0.9$). At 1 month, the mean WMSI was 1.6 ± 0.55 in all patients: 1.58 ± 0.58 and 1.61 ± 0.41 in the pharmacological and the invasive groups, respectively ($P = 0.8$). Within-group comparison showed no significant changes in WMSI from baseline to 1 month in all patients ($P = 0.3$) or in the pharmacological group ($P = 0.9$) or the invasive group ($P = 0.8$). At 1 week, the mean ESV was 64 ± 27 ml in all patients, and 64 ± 26 and 65 ± 27 ml in the pharmacological and the invasive groups, respectively ($P = 0.9$). At 30 days, the ESV was 58 ± 28 ml in the study population and 58 ± 30 and 58 ± 26 ml in the pharmacological and the invasive groups, respectively ($P = 0.9$). There were no significant

changes in the ESV from 1 week to 1 month in all study groups. LVEF, measured by the modified Simpson method at 1 week, was 42 ± 15 , 42 ± 16 , and $42 \pm 15\%$, in all patients, the pharmacological group, and the invasive group, respectively ($P = 0.8$).

After 1 month, the LVEF was $42 \pm 16\%$ in the study population, and 42 ± 17 and $43 \pm 15\%$ in the pharmacological and the invasive groups, respectively ($P = 0.8$). No significant changes in LVEF from baseline to 1 month were detected (Tables 2 and 3).

Tissue Doppler imaging

The systolic mitral annular velocity (Sa) at 1 week was 5 ± 4 cm/s in all patients, and 4 ± 2 and 6 ± 1.4 cm/s in the pharmacological and the invasive subgroups, respectively ($P = 0.03$). At the 1-month follow-up, Sa increased to 7 ± 3 cm/s in the study population, and

Table 1 Baseline characteristics of the study population

Variable	Pharmacological group ($n = 30$)	Invasive group ($n = 30$)	<i>P</i> value
Age (mean $\pm$ SD) (years)	57 ± 7	59 ± 7	0.48
Male [n (%)]	28 (93)	27 (90)	0.7
Smoker [n (%)]	14 (46)	22 (73)	0.29
Diabetes mellitus [n (%)]	10 (33)	12 (40)	0.29
Hypertension [n (%)]	7 (23)	6 (22)	0.5
Dyslipidemia [n (%)]	7 (23)	6 (20)	0.5
Anterior infarction [n (%)]	24 (80)	25 (83)	0.7
Inferior infarction [n (%)]	6 (20)	5 (16)	0.4

Table 2 Echocardiographic data at 1 week

Variable	Pharmacological group ($n = 30$)	Invasive group ($n = 30$)	<i>P</i> value
WMSI	1.57 ± 0.6	1.58 ± 0.4	0.9
2D			
ESV (ml)	64 ± 26	65 ± 27	0.9
EDV (ml)	112 ± 30	111 ± 34	0.9
EF%	42 ± 16	42 ± 15	0.8
MM			
LVIDd (cm)	5.4 ± 0.4	5.5 ± 0.5	0.9
LVIDs (cm)	4 ± 0.7	4.1 ± 0.7	0.4
EF%	49 ± 18	47 ± 13	0.7
Pulsed Doppler [n (%)]			
Normal	3 (10)	2 (7)	0.09
Grade 1	17 (57)	15 (50)	0.3
Grade 2	8 (27)	10 (33)	0.5
Grade 3	2 (6)	3 (10)	0.08
2D strain (GLPSS)	-11.92 ± 4	-15.2 ± 5	0.04
TDI (Sa) (cm/s)	4 ± 2	6 ± 1.4	0.03

EF, ejection fraction; EDV, end-diastolic volume; ESV, end-systolic volume; GLPSS, global longitudinal peak systolic strain; LVIDd, left ventricular dimension in diastole; LVIDs, left ventricular dimension in systole; WMSI, wall motion score index.

5 ± 2 and 8 ± 2 cm/s in the pharmacological and the invasive subgroups, respectively. There was significant improvement in Sa at 1 month in the invasive group compared with the pharmacological group ($P = 0.02$). The increase in the mean Sa from 1 week to 1 month was statistically significant in the study population ($P = 0.01$) and within the invasive group ($P = 0.02$), but no significant increase was detected in the pharmacological group (Tables 2 and 3).

Global longitudinal peak systolic strain

At 1 week, the mean GLPSS was $-13.5 \pm 7\%$ in all patients. The GLPSS was better in the invasive group than in the pharmacological group at baseline (-15.2 ± 5 vs. $-11.9 \pm 4\%$, $P = 0.04$). After 1 month of follow-up, GLPSS improved to $-15 \pm 8\%$ in all patients, and -12 ± 4 and $-16 \pm 3\%$ in the pharmacological and the invasive subgroups, respectively, and the difference was statistically significant between both groups ($P = 0.04$).

Within-group analysis showed a significant improvement in GLPSS from 1 week to 1 month in all patients ($P = 0.03$) as well as within the invasive group ($P = 0.04$), but not in the pharmacological group (Tables 2 and 3).

The recovery of the left ventricular function

On the basis of both the improvement in 2D strain (GLPSS $\geq 10\%$ from baseline) and the increase in the mitral annular systolic velocity, 47% of the study population was considered as having a significant recovery in LV systolic function by 1 month. Within-group analysis showed that 60% of the patients who were treated invasively showed improvement at 1 month compared with 40% of those who had fibrinolysis, but this difference was not of statistical significance ($P = 0.5$; Figs 1 and 2). By multivariate

regression analysis at the end of the follow-up, the baseline GLPSS and the Sa were significant predictors of recovery of systolic function [odds ratio (OR) = 1.3, $P = 0.03$ and OR = 1.22, $P = 0.02$, respectively] and were superior to WMSI and EF% (OR = 0.89, $P = 0.12$ and OR = 0.96, $P = 0.18$, respectively). LAD as the problematic vessel was a significant predictor (OR = 0.49, $P = 0.01$). Demographic and risk factors were not significant predictors of recovery.

Discussion

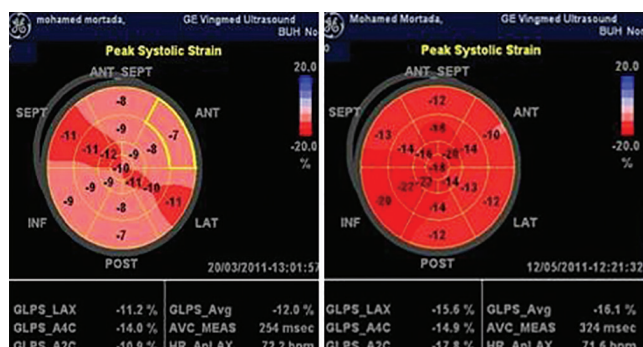
The prognostic importance of LV systolic function after AMI has been described by several large studies [8]. 2D echocardiography allows early

Table 3 Echocardiographic data at 1 month

Variable	Pharmacological group (n = 30)	Invasive group (n = 30)	P value
WMSI	1.58 ± 0.58	1.61 ± 0.41	0.8
2D			
ESV (ml)	58 ± 30	58 ± 26	0.9
EDV (ml)	104 ± 25	100 ± 27	0.7
EF%	42 ± 17	43 ± 15	0.8
MM			
LVIDd (cm)	5.4 ± 0.5	5.8 ± 0.4	0.05
LVIDs (cm)	4.5 ± 0.7	4 ± 0.8	0.2
EF%	49 ± 14	48 ± 12	0.8
Pulsed Doppler [n (%)]			
Normal	1 (3)	2 (7)	0.08
Grade 1	15 (50)	13 (43)	0.4
Grade 2	9 (30)	8 (27)	0.4
Grade 3	5 (17)	7 (23)	0.08
2D strain (GLPSS)	-12 ± 4	-16 ± 3	0.04
TDI (Sa) (cm/s)	5 ± 2	8 ± 2	0.02

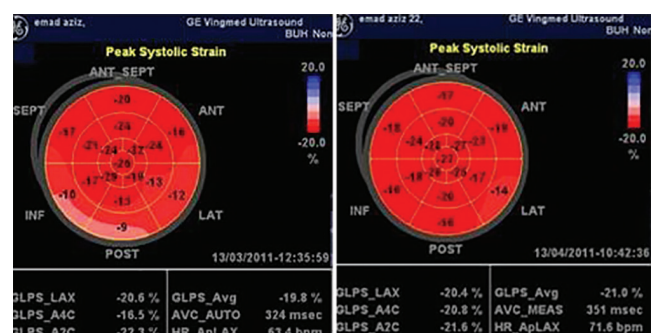
EF, ejection fraction; EDV, end-diastolic volume; ESV, end-systolic volume; GLPSS, global longitudinal peak systolic strain; LVIDd, left ventricular dimension in diastole; LVIDs, left ventricular dimension in systole; WMSI, wall motion score index.

Figure 1



The case of a 50-year-old male patient, who was a smoker, and presented with anterior myocardial infarction treated with emergency percutaneous coronary intervention. Global longitudinal peak systolic strain improved at 1 month (-12 to -16%).

Figure 2



The case of a 45-year-old, hypertensive, male patient, who presented with inferior myocardial infarction treated with thrombolytic therapy. Global longitudinal peak systolic strain improved at 1 month (-19.8 to -21%).

noninvasive assessment of the LV systolic function after AMI. In addition to the currently recommended measurements (LVEF and WMSI), strain has been introduced to quantify the LV systolic function [9]. Previous studies have demonstrated that GLPSS correlated well with LVEF in the overall population, and good intraobserver and interobserver agreements have been shown. In patients, after AMI, the correlation between LVEF and GLPSS was weaker, suggesting that the two parameters are not identical and reflect different aspects of the LV systolic function [10]. This study presented a quantitative assessment of the recovery of the LV systolic function after AMI and its predictors. At the 1-month follow-up, all conventional echocardiographic parameters showed a statistically insignificant difference in the study population and between the invasive and the pharmacological subgroups. In contrast, the average GLPSS and mitral annular systolic velocity parameters showed significant improvement between 1 week and 1 month in the study population and were significantly higher in the invasive subgroup than in the pharmacological subgroup.

These findings correlate with a prior study on 80 patients (20 controls and 60 patients with AMI), in which longitudinal strain was a good parameter to monitor the recovery of regional and global LV systolic function after MI [11]. Charlotte *et al.* [12] who conducted a study on 31 consecutive patients admitted with first-ST-segment elevation MIs using longitudinal strain on days 2, 3, and 7, reported superiority of longitudinal strain over EF% and WMSI as a discriminative parameter for the changes in LV systolic function as well as being better to assess near normalization on day 7 in patients treated early with PCI. Another study on 38 patients evaluated the impact of different therapeutic strategies on longitudinal regional myocardial systolic function in the early phase of AMI (within 1 week) using longitudinal strain (10 patients were treated with primary PCI, 16 patients were treated by thrombolytic therapy using streptokinase, and 12 were followed up conservatively): the study showed that strain can assess the efficacy of PCI accurately compared with different kinds of treatment with regard to the recovery of systolic function of the myocardium during the early stage after AMI [13].

In our study, the increase in the mean Sa from 1 week to 1 month was statistically significant for the study population and the invasive subgroup patients. This was in agreement with the tissue Doppler study on 202 patients with first MI, in which the Sa could help detect early changes in the myocardium and mild myocardial damage that was not detected by conventional echo parameters [14].

In the present study, 47% of the patients showed significant improvement in the LV systolic function (combined improvement of GLPSS and Sa). This may be explained by myocardial stunning, which is defined as postischemic reversible dysfunction of the myocardium, which recovers during a period of weeks to months. Usually, stunning occurs in the border zone of the infarct area because of an acute reduction of blood flow, which is resolved before necrosis occurs. Sjoerd *et al.* [15] found that 54% of the AMI patients improved at 3 months of follow-up and it increased to 72% of the patients at 1 year of follow-up. Solomon *et al.* [16] investigated the recovery of LV function in 249 patients with echocardiography performed on days 1, 14, and 90 after AMI, and reported a similar recovery rate of LV function in 58% of patients at 90 days, where most of the changes occurred in the first 14 days. Hence, the majority of the LV recovery occurs in the first weeks after revascularization, and the improvement can continue for months.

The baseline average GLPSS (−13.54%) was a significant predictor of the recovery of the LV function, and this is in agreement with a study by Mollema *et al.* [17], who found that a cut-off value for the baseline global LV strain of −13.7% was a predictor of LV functional recovery at follow-up and concluded that global LV strain early after AMI reflects myocardial viability and predicts the recovery of LV function.

Clinical implications

LV dysfunction after AMI is caused by a combination of myocardial stunning and necrosis. As residual LV function is of important prognostic value for long-term survival, information on the evolution of LV function is important. In the current study, improvement of the LV function was 47% at 1 month after AMI; therefore, baseline echocardiographic assessment of the infarct size may be overestimated, and serial echocardiography during follow-up is essential; baseline average GLPSS and Sa were identified as predictors of improvement of LV function. It may be reasonable to use GLPSS for better assessment of the recovery of LV systolic functions after AMI rather than conventional echocardiography.

Study limitations

- (1) Small sample size.
- (2) Short-term follow-up.
- (3) Lack of randomization.
- (4) A cut-off of at least 10% the increase in GLPSS was used to define improvement of global LV

function during follow-up. However, few studies have assessed the time course of LV strain, and future studies have to focus on defining an optimal cut-off for the improvement in LV function assessed with strain.

Conclusion

The average GLPSS and Tissue Doppler parameters detected the recovery of LV systolic function after AMI. Moreover, better recovery was reported in invasive reperfusion than in the pharmacological group.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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