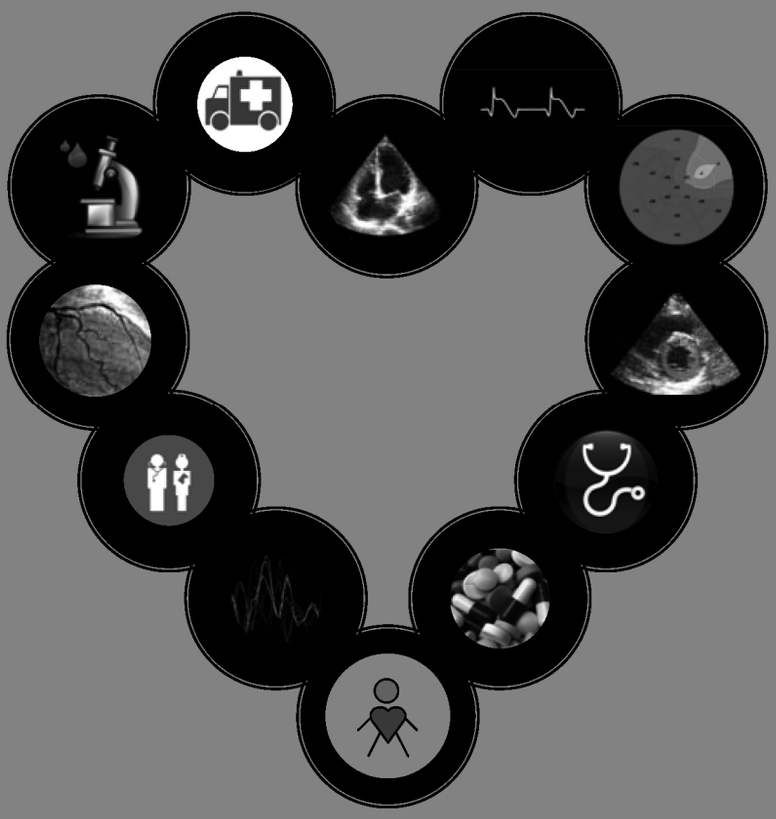
Improving risk stratification after acute myocardial infarction : focus on emerging applications of echocardiography

Antoni, M.L.



Chapter 8

Time Course of Global Left Ventricular Strain after Acute Myocardial Infarction

M. Louisa Antoni, Sjoerd A. Mollema, Jael Z. Atary,
C. Jan Willem Borleffs, Eric Boersma, Nico R.L. van de Veire,
Eduard R. Holman, Ernst E. van der Wall,
Martin J. Schalij, Jeroen J. Bax

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Abstract

Objectives

The purpose of the present study was to assess the evolution of left ventricular (LV) function after acute myocardial infarction (AMI) using global longitudinal peak systolic strain (GLPSS) during 1 year follow-up. In addition, patients were divided in groups with early, late or no improvement of LV function and predictors of recovery of LV function were established

Methods and results

A total of 341 patients with AMI were evaluated. Two-dimensional echocardiography was performed at baseline, 3, 6 and 12 months. At baseline, LV function was assessed with traditional parameters and GLPSS. GLPSS was re-assessed at 3, 6 and 12 months. Improvement of LV function was based on GLPSS and was observed in 72% of the patients. No differences were observed between patients with early and late improvement. The left anterior descending coronary artery as culprit vessel, peak cardiac troponin T level, diastolic function and baseline GLPSS were identified as independent predictors of recovery of LV function.

Conclusions

Improvement of LV systolic function occurred in the majority of patients during follow-up. GLPSS, left anterior descending coronary artery as culprit vessel, peak cardiac troponin T level and diastolic function were independent predictors of recovery of LV function. Quantification of GLPSS may be of important value for the prediction of recovery of LV function in patients after AMI.

Introduction

Quantification of left ventricular (LV) systolic function is an important component in the follow-up of patients after acute myocardial infarction (AMI).[1-3] Currently, the recommended measurements for echocardiographic quantification of global and regional LV systolic function are LV ejection fraction (LVEF) and wall motion score index (WMSI).[4-6] Global longitudinal peak systolic strain (GLPSS) has recently been introduced as a novel technique to reflect LV systolic function with two-dimensional (2D) echocardiography. 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.[7] The importance of LV function after AMI has been extensively studied. However, the evolution of LV function after AMI has not been completely elucidated. The purpose of the present study was to evaluate the time course of LV systolic function quantified by GLPSS during 1 year follow-up after AMI. In addition, differences in baseline characteristics were identified between patients with early, late or no improvement of LV function and predictors of recovery of LV function were established.

Methods

Study population and protocol

Consecutive patients admitted with an AMI treated with primary percutaneous coronary intervention were evaluated. All patients were treated according to the institutional AMI protocol, which includes a prehospital, inhospital, and outpatient clinical framework for decision making and treatment. This protocol, designed to improve care around AMI, includes structurized medical therapy, 2D echocardiography performed after primary percutaneous coronary intervention and within 48 hours of admission and standardized outpatient follow-up, as described previously.[8] 2D echocardiography was performed at baseline within 48 hours after admission and during follow-up at 3, 6 and 12 months. Baseline echocardiography was used to assess LV function with traditional parameters and GLPSS quantified with the novel automated function imaging technique. During follow-up GLPSS was re-assessed at 3, 6 and 12 months to investigate the evolution of systolic function over time after AMI.

Echocardiography

Patients were imaged in the left lateral decubitus position using a commercially available system (Vivid 7, General Electric-Vingmed, Horton, Norway). Images were obtained, with a simultaneous ECG signal, using a 3.5-MHz transducer at a depth of 16 cm in the parasternal and apical views. Standard M-mode and 2D-images were acquired during breath hold and saved in cine-loop format from 3 consecutive beats. Analysis of echocardiographic images was performed randomly, offline by 2 experienced observers (EchoPac version 7.0.0, General Electric-Vingmed). The biplane Simpson's technique was used to calculate LV end-systolic volume, LV end-diastolic volume and LVEF.[6]

To calculate WMSI, the LV was divided into 16 segments. Each segment was analyzed individually and scored based on its motion and systolic thickening (1 = normokinesis, 2 = hypokinesis, 3 = akinesis, 4 = dyskinesis). WMSI was calculated as the sum of the segment scores divided by the number of segments scored.[6] 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.[9] Pulsed-wave Doppler of the mitral valve was obtained by placing the Doppler sample volume between the tips of the mitral leaflets. The early (E) and late (A) peak diastolic velocities and E-wave deceleration time were measured. The E/E'-ratio was obtained by dividing E by E', which was measured using color-coded tissue Doppler imaging at the septal side of the mitral annulus in the apical 4-chamber view.[10] Diastolic function was graded according to the most recent recommendations of the American Society of Echocardiography.[11] Diastolic function was graded as normal, when septal E' was ≥8. Diastolic dysfunction was graded as grade I(mild), when septal E' was <8, E/A ratio<8 and deceleration time>200 ms; grade II(moderate), when septal E' was <8, E/A ratio 0.8–1.5 and deceleration time 160–200 ms; grade III(severe), when septal E' was <8, E/A ratio≥2 and deceleration time<160 ms.

Global left ventricular longitudinal strain using automated function imaging

Apical 4-and 2-chamber, as well as long-axis views were used for quantification of GLPSS 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 2 dimensions.[12] All analyzed images were recorded with a frame rate of at least 40fps for reliable analysis by the software. 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 R wave and aortic valve was automatically measured and used as a reference for the 4-and 2-chamber views. After defining the mitral annulus and LV apex with 3 index points in all 3 apical views, the LV endocardial border was automatically traced at end-systole and the created region of interest was manually adjusted to the thickness of the myocardium. Tracking quality was validated in all segments from the 3 apical views. Segments which failed to track by the software were manually adjusted by the operator. Any segments which subsequently failed to track were automatically discarded by the software for the calculation of global strain. Analysis was feasible in 97% of the segments. GLPSS for the complete LV was provided by the software using a 17-segment model in a "bull's eye" plot calculated as the average of longitudinal peak systolic strain of each view (Figure 1).

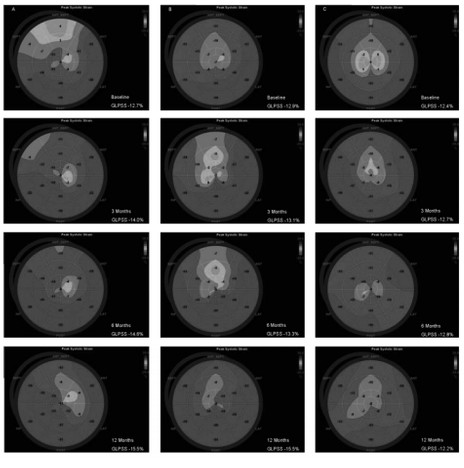

Figure 1.

Bull's-eye plots showing the evolution of segmental peak systolic longitudinal strain and GLPSS (global longitudinal peak systolic strain) of representative patients from each patient group (early improver [A]; late improver [B]; non-improver [C]).

Definition of improvement of left ventricular systolic function

Patients underwent echocardiographic assessment at baseline and during follow-up at 3, 6 and 12 months, and were divided into subgroups based on the increase in GLPSS during follow-up. The specific subgroups were improvers (patients with ≥10% increase of GLPSS between baseline and 3 months, 3 and 6 months or 6 and 12 months) and non-improvers (patients without ≥10% increase of GLPSS during follow-up). The group of improvers was

further subdivided into early improvers (patients with ≥10% increase of GLPSS at 3 months) and late improvers (patients with ≥10% increase of GLPSS in the period after 3 months).

Statistical analysis

Continuous data are presented as mean±standard deviation and were compared using Student's *t*-test for unpaired data. Continuous variables which were not normally distributed (as evaluated by Kolmogorov-Smirnov tests), were presented as medians and corresponding 25^{th} and 75^{th} percentiles and were compared using Wilcoxon Rank-Sum test. Categorical data are presented as frequencies and percentages and were compared using chi-square test. Differences in GLPSS during follow-up were evaluated as a within-subjects factor with analysis of the variance for repeated measurements. Post-hoc comparisons were performed using the Bonferroni adjustments for multiple comparisons. Univariate and multivariate logistic regression analyses were performed to identify baseline clinical (age, gender, risk factors, medical history, symptoms to balloon time (after transformation), left anterior descending coronary artery as culprit vessel, multivessel disease, thrombolysis in myocardial infarction(TIMI) 2–3 flow, peak cardiac troponin T level (after transformation), QRS duration (after transformation) and medication at 6 months follow-up) and baseline echocardiographic characteristics (LV end-diastolic volume indexed for body surface area, WMSI, moderate or severe MR, diastolic function≥grade I, GLPSS) that predict improvement of GLPSS during follow-up. All variables in univariate analysis with a p value ≤0.20 were considered for multivariate analysis. The number of covariables had to be limited because of the relatively small number of patients without improvement in LV function and the final multivariate model was constructed via backward deletion of the least significant variable, until all variables had a p value ≤0.15. Peak creatine phosphokinase level was excluded from uni- and multivariate analysis to avoid co-linearity with peak troponin T level. Thereafter, the incremental value of GLPSS in addition to known factors related to improvement of LV function (clinical information (current smoking, left anterior descending coronary artery as culprit vessel, peak cTnT level), LV end-diastolic volume index, WMSI, and diastolic function ≥grade I) was established. For this purpose, those characteristics were first entered into the model in a stepwise fashion. Subsequently,

GLPSS was entered individually. Global chi-square values, including the significance level for each step in relation to the previous value, were calculated. Reproducibility of GLPSS values was analyzed with repeated measurements by 1 experienced observer at 2 different time points and by a second experienced observer in 20 randomly selected individuals. Inter- and intra-observer agreement for GLPSS were evaluated by Bland-Altman analysis. All statistical tests were two-sided and a p value <0.05 was considered to be statistically significant.

Results

Baseline characteristics of the patient population

A total of 341 patients with complete 1 year follow-up were evaluated. Twenty-eight patients (8%) died within 1 year follow-up and therefore did not have echocardiographic assessment at 3, 6 or 12 months. Data of these patients was included up to the time of their death. Baseline clinical characteristics of the study population are summarized in Table 1. Mean age was 60 ± 12 years and 77% of the patients were men. The left anterior descending coronary artery was the culprit vessel in 183 patients (54%). Peak cardiac enzymes were 2122 (947, 3717) U/l and 6 (2, 11) µg/l for creatine phosphokinase and cardiac troponin T, respectively.

Baseline echocardiographic characteristics are summarized in Table 2. Mean LV volumes were 62±20 ml for LV end-systolic volume and 114±33 ml for LV end-diastolic volume, and mean LVEF was 45±8%. Mean GLPSS at baseline was -13.7±3.3%. LVEF, WMSI and global LV strain were significantly correlated, however the correlations were not perfect: r=0.42 and r=0.61, respectively, both p <0.001. Inter- and intra-observer variability were -0.2±0.8% and 0.1±2.2% (mean±2 SD), respectively.

Time course of left ventricular global longitudinal peak systolic strain after acute myocardial infarction

During follow-up, mean GLPSS increased from -13.7 ± 3.3% at baseline to -16.0 ± 3.4% at 3 months, -16.3 ± 3.6% at 6 months and -16.8 ± 3.9% at 12 months (Figure 2). There was a significant increase of GLPSS at 3 months (p <0.001), and between 3 and 12 months (p <0.001).

Table 1. Baseline clinical characteristics

	All patients	*Improvers (N = 246)*	*Non-improvers (N = 70)*	*P**
Age(years)	60 ± 12	60 ± 12	59 ± 11	0.43
Male gender	263 (77%)	188 (76%)	58 (83%)	0.25
Current smoking	165 (49%)	127 (52%)	28 (40%)	0.09
Diabetes	32 (10%)	18 (7%)	8 (11%)	0.27
Family history of CAD	141 (42%)	103 (42%)	33 (47%)	0.45
Hyperlipidemia	66 (20%)	48 (20%)	13 (19%)	0.85
Hypertension	100 (30%)	70 (29%)	20 (29%)	0.99
Prior myocardial infarction	18 (5%)	11 (5%)	5 (7%)	0.37
Symptoms to balloon time (min)	191 (145, 264)	189 (142, 259)	185 (145, 261)	0.86
LAD culprit vessel	183 (54%)	123 (50%)	47 (67%)	0.01
Multivessel disease	178 (53%)	123 (50%)	37 (53%)	0.67
TIMI 2–3 flow	333 (98%)	242 (98%)	70 (100%)	0.28
Peak CPK level(U/l)	2645 (947, 3717)	1684 (760, 2787)	4336 (2590, 5687)	<0.001
Peak cTnT level(μg/l)	6 (2, 11)	5 (2, 8)	12 (10, 18)	<0.001
QRS duration(ms)	95 (84, 90)	90 (86, 98)	90 (82, 100)	0.65
Medication at 6-months follow-up				
ACE inhibitor/ARB	310 (98%)	241 (98%)	67 (97%)	0.67
Antiplatelets	317 (100%)	246 (100%)	69 (100%)	1.00
Beta-blocker	286 (90%)	220 (89%)	64 (93%)	0.41
Statin	312 (98%)	241 (98%)	69 (100%)	0.23

*P-values are given for the comparison of improvers (patients with ≥10% increase in GLPSS during follow-up) versus non-improvers (patients without ≥10% increase in GLPSS during follow-up). ACE:angiotensin-converting enzyme; ARB:angiotensin receptor blocker; CAD:coronary artery disease; CPK:creatine phosphokinase; cTnT:cardiac troponin T; LAD:left anterior descending coronary artery; PCI:percutaneous coronary intervention; TIMI:thrombolysis in myocardial infarction.

Table 2. Baseline echocardiographic characteristics

	All patients	*Improvers (N = 246)*	*Non-improvers (N = 70)*	*P**
LVESV(ml)	62 ± 20	62 ± 20	67 ± 22	0.06
LVESVI(ml/m^2)	32 ± 9	31 ± 9	34 ± 10	0.02
LVEDV(ml)	114 ± 33	114 ± 31	121 ± 36	0.13
LVEDVI(ml/m^2)	59 ± 15	58 ± 14	62 ± 16	0.05
LVEF(%)	45 ± 8	46 ± 8	45 ± 8	0.23
WMSI	1.5 ± 0.3	1.5 ± 0.3	1.6 ± 0.2	<0.001
Moderate or severe MR	28 (8%)	17 (7%)	6 (9%)	0.60
Diastolic function				0.03
Grade0	18 (6%)	12 (5%)	5 (8%)	
GradeI	164 (51%)	137 (56%)	25 (38%)	
GradeII	69 (21%)	53 (22%)	15 (23%)	
GradeIII	71 (22%)	43 (19%)	21 (32%)	
GLPSS(%)	-13.7 ± 3.3	-14.1 ± 3.1	-13.2 ± 3.0	0.04

*P-values are given for the comparison of improvers (patients with≥10% increase in GLPSS during follow-up) versus non-improvers (patients without≥10% increase in GLPSS during follow-up). GLPSS: global longitudinal peak systolic strain; LVEDV: left ventricular end-diastolic volume; LVEDVI: left ventricular end-diastolic volume index; LVEF: left ventricular ejection fraction; LVESV: left ventricular end-systolic volume; LVESVI: left ventricular end-systolic volume index; MR: mitral regurgitation; WMSI: wall motion score index.

Figure 3 shows the cumulative percentage of patients that improved ≥10% in GLPSS during the different periods of follow-up. Most patients (54%) improved within 3 months follow-up, compared to 11% of the patients who improved between 3 and 6 months and only 7% of the patients who improved between 6 and 12 months follow-up. Finally, 28% of the patients did not increase ≥10% in GLPSS during 1 year follow-up after AMI. Subsequently, the study population was divided in 2 groups: non-improvers versus improvers. The clinical and echocardiographic characteristics of these patients are summarized in Table 1 and 2.

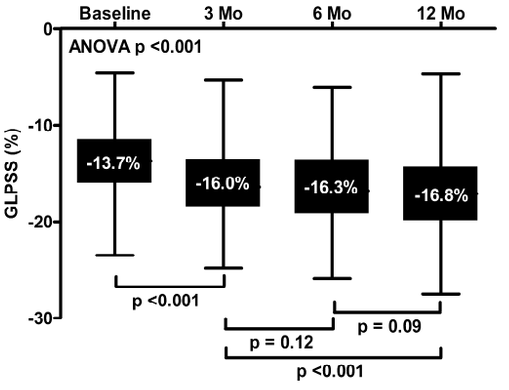

Figure 2.
Time course of global longitudinal peak systolic strain. Global longitudinal peak systolic strain (GLPSS) at baseline, 3, 6 and 12 months after AMI. Mo: months.

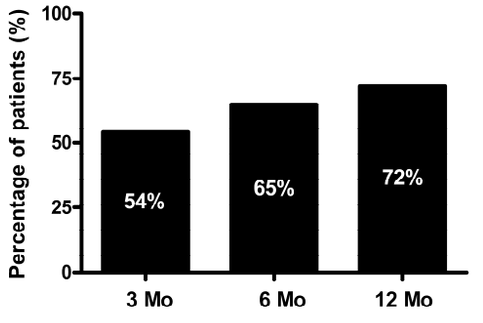

Figure 3.
Cumulative percentage of improvers (defined as ≥10% increase of global longitudinal peak systolic strain (GLPSS) between baseline and 3 months; 3 months and 6 months; or 6 months and 12 months follow-up). Mo: months.

Non-improvers were more likely to have the left anterior descending coronary artery as culprit vessel (47 (67%) vs. 123 (50%), p = 0.01) and higher peak cardiac enzymes (4336 (2590, 5687U/l vs. 1684 (760, 2787) U/l, p <0.001 and 12 (10, 18) µg/l vs. 5 (2, 8) µg/l, p <0.001 for peak creatine phosphokinase and peak cardiac troponin T level, respectively). Furthermore, non-improvers had higher WMSI (1.6 ± 0.2 vs. 1.5 ± 0.3, p <0.001), more often had diastolic dysfunction and lower GLPSS (-13.2 ± 3.0% vs. -14.1 ± 3.1%, p = 0.04). No significant differences were observed in LVEF between improvers and non-improvers (46 ± 8% vs. 45 ± 8%, p = 0.23).

Next, the group of improvers was divided into early improvers (improvement at 3 months) and late improvers (improvement in the period after 3 months). Of interest, no differences in baseline characteristics were observed between these 2 groups. In Figure 1, examples of

bull's-eye plots, providing peak systolic longitudinal strain for all left ventricular segments, are demonstrated for the 3 subgroups.

Predictors of improvement of left ventricular function

Significant univariate predictors of improvement of GLPSS during 1 year follow-up after AMI are shown in Table 3. At multivariate analysis, left anterior descending coronary artery as culprit vessel (OR 0.40, 95%CI 0.19–0.87, p = 0.02), peak cardiac troponin T level (OR 0.21, 95%CI 0.14– 0.32, p <0.001), diastolic function ≥grade I (OR 4.71, 95%CI 1.13–19.63, p = 0.03) and baseline GLPSS (OR 1.26, 95%CI 1.08–1.46, p = 0.003) were independent predictors of improvement of LV function (Table 3).

Global chi-square scores were calculated to assess the incremental prognostic value of GLPSS. GLPSS provided incremental prognostic value to baseline clinical information (current smoking, left anterior descending coronary artery as culprit vessel, peak cTnT level), LV end-diastolic volume index, WMSI, and diastolic function ≥grade I, for the prediction of improvement of LV function (Figure 4).

Table 3.Prediction of improvement of left ventricular function

	Univariate analysis			*Multivariate analysis*		
	OR	***95%CI***	***P***	***OR***	***95%CI***	***P***
Current smoking	1.60	0.93–2.74	0.09			
LAD culprit vessel	0.49	0.28–0.86	0.01	0.40	0.19–0.87	0.02
Peak cTnT level(μg/l)	0.30	0.21–0.42	<0.001	0.21	0.14–0.32	<0.001
LVEDVI(ml/m^2)	0.98	0.96–1.00	0.05			
WMSI	0.14	0.05–0.42	0.001			
Diastolic function≥grade I	2.31	0.75–7.16	0.15	4.71	1.13–19.63	0.03
Baseline GLPSS(%)	0.91	0.83–0.99	0.04	1.26	1.08–1.46	0.003

Improvement of LV function was defined as an increase of ≥10% in GLPSS during follow-up. cTnT: cardiac troponin T; DT: deceleration time; E/A: mitral inflow peak early velocity (E) / mitral inflow peak late velocity (A); E/E': mitral inflow peak early velocity (E) / mitral annular peak early velocity (E'); GLPSS: global longitudinal peak systolic strain; LAD: left anterior descending coronary artery; LVEDVI: left ventricular end-diastolic volume index; WMSI: wall motion score index.

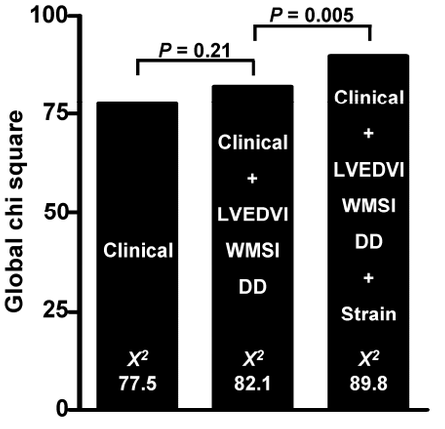

Figure 4.
Global left ventricular strain provided incremental value to clinical parameters (current smoking, left anterior descending coronary artery as culprit vessel, peak cardiac troponin T level), left ventricular end-diastolic volume index, wall motion score index and diastolic dysfunction (≥grade I).

Discussion

The major findings of the present study can be summarized as follows. (1) During 1 year follow-up of patients after AMI, 72% of patients showed improvement of LV function (≥10% increase of GLPSS). (2) The majority of patients (54%) demonstrated improvement at 3 months. Only 11% of the patients revealed improvement at 6 months and 7% at 12 months follow-up. (3) No significant differences in baseline characteristics were observed between early improvers (at 3 months) and late improvers (after 3 months). (4) Left anterior descending coronary artery as culprit vessel, peak cardiac troponin T level, diastolic function and baseline GLPSS were independent predictors of improvement of LV function during follow-up. Baseline GLPSS provided incremental value to traditional parameters for the prediction of recovery of LV function.

Quantification of left ventricular systolic function

The prognostic importance of LV systolic function after AMI has been described by several large studies.[2,3,13] 2D echocardiography permits early noninvasive assessment of LV systolic function after AMI. In addition to the currently recommended measurements LVEF and WMSI, strain has been introduced to quantify LV systolic function.[3,5,13] Previous studies have demonstrated that GLPSS correlated well with LVEF in the overall population and good intra- and inter-observer agreement have been shown. In patients after AMI, the correlation between LVEF and GLPSS was less strong, suggesting that the 2 parameters are

not identical and reflect different aspects of LV systolic function.[7] The present study is the first to evaluate the time course of LV systolic function, quantified with GLPSS during 1 year follow-up after AMI.

Time course of left ventricular systolic function after AMI

In the current study, most patients showed early improvement of LV function, which can be explained by myocardial stunning. Stunning is defined as postischemic reversible dysfunction of the myocardium and recovers during a period of weeks to months.[14] Usually, stunning occurs in the border zone of the infarct area because of an acute reduction of blood flow, which is resolved before necrosis can occur. Therefore, baseline echocardiographic assessment of infarct size may be overestimated.[15] Solomon et al. investigated the recovery of LV function in 249 patients with echocardiography performed on day 1, day 14 and day 90 after AMI. The authors 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.[16]

Few serial echocardiographic studies have been performed to investigate the late recovery of LV function. In a group of 108 patients with AMI, Sheiban et al. described the time course of LV function with LV volumes, LVEF and WMSI and observed significant improvement of LV systolic function between day 1 and day 180 in patients with primary percutaneous coronary intervention within 4 hours from symptom onset. However, no further significant improvement of LV function was observed between 3 and 6 months and no significant improvement was observed in patients reperfused after 4 hours.[17] Parodi et al. performed echocardiography within 24 hours, at 1 and 6 months after AMI and reported a recovery rate of 58% after 6 months (defined as ≥10% increase of LVEF). The 5-year cardiac death rate was significantly lower in patients with recovery of LV function as compared to patients without recovery (8% vs. 18%, respectively, $p = 0.024$). Interestingly, no differences were observed in the probability of survival between patients with early recovery (1 month) and late recovery (6 months) of LV function (91% vs. 93%, $p = 0.92$).[18] In the present study, 18% of the patients revealed late improvement of LV systolic function.[18] This finding implies that, although the majority of LV recovery occurs in the first weeks after revascularization, improvement can continue for up to 6 months or more.[19]

The comparison of patients with early and late improvement of LV function demonstrated no significant differences in baseline characteristics.

Prediction of improvement of left ventricular function

It has been well recognized that enzymatic infarct size is an important predictor of all-cause mortality.[20] The results of the present study support previous studies that peak cardiac troponin T level is strongly related to the recovery of LV function and is more important than other early clinical measures.[16-18] In addition to peak cardiac troponin T level, diastolic dysfunction was a significant predictor of recovery of LV function. Thus far, no data are available about the importance of diastolic function and the recovery of LV function. However, deceleration time has been extensively studied in relation to LV remodeling and the occurrence of adverse events Poulsen et al. described that the presence of early diastolic dysfunction after AMI was associated with an increased risk for LV remodeling and development of congestive heart failure. Furthermore, deceleration time ≤140 ms best identified patients at risk of development of congestive heart failure and cardiac death.[21] More recently, a meta-analysis (MeRGE-AMI) of 12 prospective post-infarction trials (3739 patients) showed that the presence of restrictive filling pattern (defined as high E/A ratio and/or shortened deceleration time) after AMI was a strong predictor of mortality.[22] Of note, the novel parameter GLPSS measured at baseline was found to be an early predictor of improvement of LV function. At multivariate analysis GLPSS remained an independent predictor of recovery of LV function and was superior over LV end-diastolic volume index and WMSI. In addition, GLPSS provided incremental value to traditional parameters for the prediction of improvement of LV function.

Clinical implications

LV dysfunction after AMI is caused by a combination of myocardial stunning and necrosis. Since 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 LV function was observed up to 1 year after AMI, where 54% of the patients showed improvement of LV systolic function at 3 months. Therefore, baseline echocardiographic assessment of infarct size may be overestimated and serial

echocardiography during follow-up is essential, particularly during the first 3 months. The left anterior descending coronary artery as culprit vessel, peak cardiac troponin T level, diastolic function and GLPSS were identified as independent predictors of improvement of LV function. Importantly, although changes during follow-up in longitudinal strain were modest, GLPSS provided incremental value to known factors for the prediction of improvement of LV function after AMI.

Limitations

The importance of this study seems limited because LV function is relatively preserved at baseline and changes in GLPSS during follow-up are modest. However, longitudinal strain has been well validated in previous studies and the reproducibility is good and it does not significantly add to the time needed to analyze a study.[7,23] In addition, longitudinal strain has found to be superior to LVEF and WMSI for the prediction of outcome and may become the optimal method for the assessment of LV systolic function.[23-24] In the current study, GLPSS provided incremental value to clinical and traditional echocardiographic parameters for the prediction of improvement of LV function. Finally, a cut-off of ≥10% increase of GLPSS was used to define improvement of global LV function during follow-up. However, no previous 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.

Conclusions

Improvement of LV systolic function after AMI occurred in the majority of patients (72%) during 1 year follow-up and most of these patients (54%) improved in LV function at 3 months follow-up. Together with left anterior descending coronary artery as culprit vessel, peak cardiac troponin T level and diastolic function, GLPSS measured at baseline was an independent predictor of recovery of LV function. Although changes in GLPSS during follow-up were modest, GLPSS provided incremental value to traditional parameters for the prediction of improvement of LV function. Quantification of GLPSS at baseline may be of important value for the prediction of recovery of LV function in patients after AMI.

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