

Echocardiographic Assessment of Myocardial Viability and Prediction of Left Ventricular Functional Recovery after Acute Myocardial Infarction Using Strain and Strain Rate

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Abstract:

Background: Extent of viable myocardial tissue has been recognized as a major determinant of recovery of left ventricular (LV) function after myocardial infarction. The present research was aimed at assessment of myocardial viability and prediction of left ventricular functional recovery in patients after acute anterior ST-elevated myocardial infarction using Tissue Doppler strain. **Methods:** In this prospective observational research, 47 patients admitted into the hospital with acute anterior ST-elevated myocardial infarction were included. All patients underwent two-dimensional and strain echocardiography within 48-72 hours of admission. Follow up two-dimensional echocardiography had performed at 6 months after baseline examination. **Results:** Total 47 patients (mean age, 57±5 years) underwent two-dimensional and strain echocardiography within 48-72 hours of admission. Significant relations were observed between baseline global systolic lengthening strain and wall motion score index ($r=0.67$), change in left ventricular ejection fraction (LVEF, $r=-.844$), Global Ses ($r=.441$) and on admission troponin I ($r=0.397$). At 6-months follow-up, LV ejection fraction was reassessed. Patients with absolute improvement in LV ejection fraction $\geq 5\%$ at 6-months follow-up ($n=24$; 51%) had a higher (more negative) baseline global Ses strain ($P<0.001$) and lower global systolic lengthening ($P<0.001$). A cutoff value for baseline global systolic lengthening strain of 7.6% yielded a sensitivity of 83% and a specificity of 87% to predict LV functional recovery at 6-months follow-up. **Conclusions:** Global Left ventricular strain (Ses and Systolic lengthening) early after acute anterior ST-elevated myocardial infarction reflects myocardial viability and predicts recovery of LV function at 6-months follow-up.

Keywords: Echocardiography; Myocardial Infarction; Viability; Strain.

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Introduction:

The accurate assessment of viable myocardium and prediction of its functional recovery is an important clinical issue in patients with acute myocardial infarction (AMI) because only viable myocardium can be salvaged by reperfusion therapy. Revascularization of dysfunctional but still viable myocardium may be of prognostic importance.¹ Patients with ST-segment elevation myocardial infarctions (STEMIs) should receive reperfusion therapy according to present guidelines. However patients with AMIs are a heterogeneous group in which up to one third of those with non-ST-segment elevation myocardial infarctions (NSTEMI) have occluded culprit arteries, while some patients with STEMIs have no

occlusions. Patients might present inconsistent ST-segment elevation, lack of atypical chest pain making the decision process for therapy challenging. Some researches revealed that the culprit lesion is occluded in 24% of patients with non-STEMIS and these patients have worse outcomes.² But current recommendations for acute revascularization in AMI are based mainly on electrocardiographic features and the duration of chest pain. So that correct decision regarding reperfusion is extremely difficult in significant number of patients. Thus, the correct identification of viable tissue in patients with ischaemic heart disease could be of great clinical importance.³ Electrocardiography is currently the only bedside method for the detection of transmural injury in

AMI. In a clinical setting a range of different imaging modalities can be useful in assessing viability.⁴ But established methods for viability assessment [single photon emission computed tomography, Delayed enhanced cardiac magnetic resonance imaging (DE-CMR), and dobutamine stress echocardiography] are usually unsuitable or unavailable in acute condition and there is currently no established method to detect viable myocardium during ongoing ischemia. However, in these settings, the echocardiographic tissue Doppler strain rate imaging (SRI) technique will be very useful tool for decision making. The quantification of regional myocardial deformation using strain Doppler echocardiography is feasible as a bedside method in the acute setting, particularly at medical centers without percutaneous coronary intervention (PCI) facilities and for patients without ST-segment elevation or those with infarctions accompanied by atypical clinical features.^{5,6} Previous studies have demonstrated that strain Doppler echocardiography can detect myocardial scarring shortly after PCI and after left ventricular (LV) remodeling.⁷⁻⁹ Furthermore, strain Doppler echocardiography has improved sensitivity compared with visual assessment of left ventricular function in acute ischemia and dobutamine stress echocardiography for viability assessment.¹⁰⁻¹¹ Strain imaging to assess myocardial viability can be done by two dimensional strain (2-D strain) and Tissue Doppler strain. Though 2-D strain has some advantages over tissue Doppler strain such as angle independency, low frame rate and noise reduction, in the present research, tissue Doppler strain use to assess viability as superiority of 2-D strain over tissue Doppler strain for the assessment of viability was not proved, lack of availability of 2-D strain software and difficulties of endocardial border delineation in 2-D strain. To determine the myocardial viability and prediction of left ventricular functional recovery within 72 hours after acute anterior ST-elevated myocardial infarction using tissue Doppler strain. The present research hypothesized that tissue Doppler strain imaging can identify viable myocardium and predict left ventricular functional recovery in patients after acute anterior ST-elevated myocardial infarction.

Methods

This prospective observational research was carried out in the Department of Cardiology, University cardiac center, BSMMU, Dhaka from March '14 to February '15 after taking ethical clearance from the Institutional Review Board (IRB), in accordance with Helsinki Declaration for Medical Research involving human subjects. An informed consent

was signed-up after careful demonstration of the research procedures from all patients. All patients who were admitted into the hospital with acute ST segment elevated myocardial infarction were enrolled in the research after considering following inclusion and exclusion criteria. Sample size was calculated to test difference between two means and estimated sample size was 46.¹³

Inclusion criteria

- a. Acute anterior ST-elevated myocardial infarction (STEMI)
- b. Reperfusion by fibrinolytic therapy

Exclusion criteria

- a. Myocardial Revascularization during study period
- b. History of Revascularization
- c. Old myocardial infarction
- d. Atrial fibrillation/Atrial flutter, Pacemaker rhythm, Ventilatory support and Cardiogenic shock
- e. History of CABG

Operational definitions

STEMI

STEMI is diagnosed when ECG showing ST elevation of 1 mm or more in two or more consecutive leads often with reciprocal ST depression in the contralateral leads, except in leads V2-V3 where ≥ 2 mm of ST elevation in men and ≥ 1.5 mm in women were required for accurate diagnosis with positive T waves in leads with ST segment elevation with increase in cardiac enzymes.¹⁴

Acute MI

The classic world health organization criteria for an acute MI require that two of the following three elements be present.

- i) History suggestive of coronary ischemia for a prolonged period of time (> 30 minutes)
- ii) Evolutionary changes on serial ECGs suggestive of MI and
- iii) A rise and fall in serum cardiac markers consistent with myonecrosis.¹⁵

Electrocardiography

Myocardial infarction indicate if there was either new or presumed new ST-segment elevation, new LBBB, or isolated inferobasal (posterior) MI noted on the ECG before any procedures and not more than 24 h after the initial presentation.

Choose 1 of the following:

- i) New ST-segment elevation at the J point in 2 contiguous leads with the cut point ≥ 0.1 mV in all leads other than leads V
- ii) 2 through V3, where the following cut points apply: ≥ 0.2 mV in men age ≥ 40 y, ≥ 0.25 mV in men age < 40 y, or ≥ 0.15 mV in women
- iii) New isolated ST-segment depression ≥ 0.1 mV in at least 2 contiguous leads of V1 through V3 with upright T waves
- iv) New ST-segment elevation ≥ 0.05 mV in leads V7 through V9 or ≥ 0.1 mV in men age < 40 y (inferobasal [posterior] infarction)
- v) New ST-segment elevation ≥ 0.05 mV (≥ 0.1 mV in men age < 30 y) in leads V3R, V4R (right ventricular infarction)
- vi) New ST-segment elevation ≥ 0.1 mV in lead aVR with concomitant ST-segment depression ≥ 0.05 mV in at least 2 contiguous leads

The location of each type of electrocardiographic change listed below can be divided into 4 categories: Inferior leads: II, III, aVF; Anterior leads: V1 through V6; Lateral leads: I, aVL and true posterior (inferobasal): (relevant only for tall wide R waves > 40 ms in leads V1 and V2).

Consideration can be given to recording posterior ST changes, the maximal amount of ST (if applicable), and/or the number of leads with ST.

Strain

Strain describes local myocardial deformation. Longitudinal strain is a measure of percentage shortening or percentage lengthening of the myocardium.¹⁶

Strain rate

Strain rate is a measure of the speed of myocardial deformation, with the unit s^{-1} . Longitudinal strain and strain rate values are negative when the myocardium contracts and become positive when it lengthens.¹⁶

Research procedures

All the patients who admitted for acute ST-elevated myocardial infarction underwent reperfusion therapy by thrombolysis were enrolled consecutively following the inclusion and exclusion criteria. Detailed history and examination including evaluation of associated risk factors had done on admission. Echocardiography including strain echocardiogram had performed after myocardial infarction within 48-72 hours of admission. Follow up two-

dimensional echocardiography had performed 6 months after baseline examination. Research parameters were baseline left ventricular ejection fraction (LVEF), LVEF at 6-months follow up, baseline Global LV Ses (End systolic strain), baseline Global SPss (Post systolic strain), baseline Global Systolic lengthening and wall motion score index.

Echocardiography

Echocardiographical data was acquired by second-generation tissue harmonic imaging and tissue velocity imaging with a Vivid Q ultrasound scanner (GE Medical Systems) and a 1.5-4.0 MHz phased array transducer (M4S). Two-dimensional images of the heart were obtained in all standard apical views (2-chamber, 3-chamber, 4-chamber and 5-chamber views). Regional wall motion score and Ejection fraction (Biplane Simpson's) were determined. Left ventricular ejection fraction was assessed using Simpson's biplane method from grayscale digital recordings. For analysis of regional myocardial function with tissue Doppler SRI, the left ventricle was divided into 18 segments according to the model defined by the American Heart Association, modified by the addition of apical anteroseptal and inferolateral segments.¹⁷ Color tissue Doppler loops were recorded from the apical views. Patients were asked to hold their breath if needed in order to position the myocardial wall at the centre of the imaging sector during three consecutive heartbeats. Segments with artifacts or bad quality imaging data were excluded. Strain and strain rate curves were obtained by placing the sample volume in the middle of each segment. Manual adjustments were made in order to assure that the sample volume remained within the myocardium, used default strain length of 6 x 12mm. The profiles of three consecutive heartbeats were averaged for analysis if all three strain curve are consistent. But single strain curve were analyzed if single curve were consistent and others were inconsistent. End-systole was defined by aortic valve closure.

Strain and strain rate measurements

The strain values were reported here as Lagrangian strain. In the acute phase, segmental end-systolic strain (S_{es}) and post-systolic strain (S_{PSS}) values were obtained from the strain curves. S_{PSS} were defined as the maximal negative strain value occurring after aortic valve closure. The duration of systole was defined from peak R on electrocardiography to aortic valve closure. End systolic strain, systolic lengthening (if present), were assessed.¹⁸ Duration of systolic lengthening were measured based on the time duration of passive myocardial lengthening as a percentage of total systolic duration.¹⁷ Global S_{es} , Global

Spss and Global systolic lengthening values were calculated by averaging each patient's segmental values.

Wall motion score

Segmental wall motion was semi quantitatively graded as follows: normal = 1, hypokinetic, marked reduction in endocardial motion = 2, akinetic, virtual absence of inward motion and thickening = 3, and dyskinetic, paradoxical wall motion away from the center of left ventricle in systole = 4. Wall motion score index was calculated by dividing the sum of individual segment scores by the number of interpreted segments. To determine wall motion score 18-segment model (modified) was used.

Grouping of research population

Research populations were divided into two groups considering 5% improvement or more of ejection fraction as a marker of global left ventricular functional recovery.¹⁹

Group-I: Patients with global functional recovery at 6-months follow up and

Group-II: Patients without global functional recovery at 6-months follow up.

Statistical analysis

Data were processed and analyzed through computer software SPSS (Statistical Package for Social Sciences) version 22. Continuous and categorical data were expressed as mean±SD and percentage respectively. Comparisons between groups (continuous) were done by unpaired t test and categorical by Chi-Square test or Fisher exact test. Spearman's correlation coefficient test was used to analyze correlation. The level of significance was set at 0.05 and p<0.05 was considered significant. Univariate and multivariate logistic regression model were used to analyze association between strain parameters, wall motion score index, risk factors and absolute changes of ejection fraction. Sensitivity and specificity for prediction of recovery were calculated by receiver operating characteristic curve analysis and largest sum of sensitivity and specificity were cut-off value.

Results:

The present research aimed at assessment of myocardial viability and prediction of left ventricular functional recovery in patients after acute anterior ST-elevated myocardial infarction using Tissue Doppler strain within 48-72 hours of admission and follow-up two dimensional echocardiography at 6 months after baseline investigations.

Demographic characteristics

In the present research, baseline demographic characteristics were as follows: Amongst 47 patients, 24 (51%) in the Group-I (Global left ventricular functional recovery (LVEF ≥5%) and 23 (49%) in Group-II (No global left ventricular functional recovery (LVEF <5%). The mean age was 57.0±5.06 years in Group-I and 57.69±4.39 in Group-II. Male predominance was shown in both groups: Group-I 79.2% was male and in Group-II 91.3% was male.

Risk factors of coronary artery disease

Risk factors of coronary artery disease were analyzed between two groups showed no statistically significant difference. Although the prevalence of risk factors likes diabetes mellitus, smoking, dyslipidaemia and family history of coronary artery diseases were mostly observed in Group II in comparison to Group I. However, hypertension was more prevalent in Group I (13) than Group II (8).

Biochemical investigations

Table I shows the distribution of biochemical parameters in both groups on admission where higher Troponin-I and CK-MB in Group-II (no functional recovery) which indicates higher ischaemic burden and infarct size and other parameter did not show any significant difference between groups.

Two dimensional echocardiography

Two dimensional echocardiographic variables are provided in Table II. Baseline left ventricular ejection

Table-I
Comparison of the biochemical parameters between two groups (N=47)

Biochemical parameter	Group-I (n=24)	Group-II (n=23)	p value
Troponin-I (on admission (ng/ml))	13.74±8.35	29.36±15.85	<0.001 ^s
CK-MB (on admission (ng/ml))	49.70±21.80	71.02±31.21	0.010 ^s
Serum creatinine (mg/dl)	1.20±0.27	1.24±0.30	0.599 ^{ns}

Data are presented as mean±SD

Unpaired t-test was used to compare biochemical parameters between two groups

Group-I= Global left ventricular functional recovery (LVEF ≥5%)

Group-II= No global left ventricular functional recovery (LVEF <5%)

N: Number of patients; n: Number in each group NS=Not significant

s: Significant & ns: Not significant

Table-II
Comparison of the 2-Dimensional Echocardiographic variables between two groups (N=47)

2-Dimensional Echocardiographic parameter	Group-I (n=24)	Group-II (n=23)	p value
Baseline LVEF	41.25±3.67%	38.30±6.56%	0.063 ^{ns}
Follow up (6 months) LVEF	48.46±3.92%	34.57±7.06%	<0.001 ^s
WMSI (Wall motion score index)	1.42±0.18	1.73±0.27	<0.001 ^s

Data are presented as mean±SD

Unpaired t-test was used to compare echocardiographic parameters between two groups

Group-I= Global left ventricular functional recovery (LVEF ≥5%)

Group-II= No global left ventricular functional recovery (LVEF <5%)

N: Number of patients; n: Number in each group

s: Significant & ns: Not significant

fraction (LVEF) were lower in Group-II than Group-I but statistically no significant difference. However, follow up LVEF were higher in Group-I than Group-II which indicates significant improvement of LVEF in Group-I. WMSI (Wall motion score index) were lower in Group-I than Group-II which indicates less ischaemic burden in Group-I.

Time to thrombolysis and drugs

It was observed that time to thrombolysis was more in Group-II (8.61±2.33 hours) than Group-I (7.89±2.51 hours) but not statistically significant difference was observed. In terms of using, certain drugs: Angiotensin converting enzyme inhibitor (ACE) / Angiotensin receptor blocker (ARB) and statin, most of the patients responded “yes” in both groups but these were not statistically significant. Among 24 patients in Group I- 19 (79.2%) and among 23 patients in Group II- 19 (82.6%) were answered “yes” in terms of using ACE/ARB and statin. On the other hand, in Group I- 5 (20.8%) and Group II- 4 (17.4%) patients responded “no” in terms of using ACE/ARB and statin.

Tissue Doppler strain and myocardial viability

Table III shows significant higher Global Ses in Group-II than Group-I. There was also higher global systolic lengthening in Group-II. But there were little variations in Global Spss between two groups. Statistically significant lower strain values in Group-I indicate the presence of adequate amount of viable myocardium.

Correlation with global systolic lengthening and absolute change of LVEF, WMSI, Troponin-I and global Ses

Correlation values are summarized in Table IV. Substantial relations between global systolic lengthening and troponin –I as reflectors of enzymatic infarct size were demonstrated. Thus, lesser global systolic lengthening strain was related

to lesser infarct size which reflecting more viable myocardium. In addition, a strong relation was observed between global systolic lengthening strain and WMSI, as reflector of echocardiographic infarct size. Importantly, a strong negative relation was observed between global systolic lengthening and absolute change in LVEF during follow up. Thus lower value of global systolic lengthening was related to higher absolute change of LVEF. There were also modest relation between global systolic lengthening and global Ses.

Prediction of global functional recovery

Table V shows the global systolic lengthening, global Ses, troponin I, CK-MB, WMSI are related to change in LVEF at 6-month follow-up after acute myocardial infarction.

Table VI provides an overview of the multi variable analysis for change in LVEF during follow-up. Logistic regression analysis shows the global Ses and Global Systolic Lengthening strain as a predictor for change in LVEF during follow-up.

In addition, ROC (Receiver operating characteristic curve) analysis was performed (AUC- 0.918; 95% confidence interval, 0.844 to 0.993; *P*-0.001 for global systolic lengthening). A cut off value for global systolic lengthening 7.6% demonstrated to be predictive for recovery of LV function at 6-months follow-up after acute myocardial infarction with a sensitivity of 83% and a specificity of 87% and for global ses, sensitivity 75% and specificity 88% (Figures 1, Table VII).

AUC: Area under curve

Figure 1 Comparison of ROC curves of Global systolic lengthening (%), Global Ses and troponin-I for predicting global LV recovery (LVEF ≥5%).

Table-III
Comparison of the Tissue Doppler derived strain variables between two groups (N=47)

Variable	Group-I (n=24) Mean±SD	Group-II (n=23) Mean±SD	p value
Global Ses*	-11.36±2.26 (%)	-8.66±2.09 (%)	<0.001 ^s
Global Spss*	-8.68±3.07 (%)	-9.10±1.95 (%)	0.581 ^{ns}
Global Systolic lengthening (%)*	4.56±4.71 (%)	16.98±8.27(%)	<0.001 ^s

Unpaired t-test was used to compare strain parameters between two groups

Group-I= Global left ventricular functional recovery (LVEF e"5%)

Group-II= No global left ventricular functional recovery (LVEF <5%)

N: Number of patients; n: Number in each group

s: Significant & ns: Not significant

Global Ses: Global end systolic strain & Global Spss: Global post systolic strain

Table-IV
Correlation with Global systolic lengthening and absolute change of LVEF, WMSI, Troponin-I and Global Ses (N=47)

Variable	r value*	p value
Absolute change of LVEF	-.844	<0.001 ^s
WMSI	.672	<0.001 ^s
Troponin-I	.397	<0.006 ^s
Global Ses	.441	0.002 ^s

*Correlation was established by Spearman's correlation coefficient test

r: Correlation coefficient

N: Number of patients

S: Significant

Global Ses: Global end systolic strain; Global Spss: Global post systolic strain; LVEF: Left ventricular ejection fraction & WMSI: Wall motion score index

Table-V
Univariate logistic regression analysis for change in LVEF (LVEFe"5%) during Follow-up(N=47)

Variable	OR	95% CI	P value
Global Ses	1.684	1.235-2.297	0.001 ^s
Global systolic lengthening	1.372	1.133-1.662	0.001 ^s
WMSI	373.951	12.875-10861.018	0.001 ^s
CKMB	1.026	1.003-1.050	0.027 ^s
Troponin-I	1.119	1.047-1.197	0.001 ^s
Male sex	2.763	.479-15.954	0.256 ^{ns}
Age	1.033	.913-1.168	0.610 ^{ns}
DM	0.364	0.801-9.447	0.108 ^{ns}
ACE/ARB	1.250	0.290-5.385	0.765 ^{ns}

OR: Odds ratio & 95% CI: 95% Confidence interval

N: Number of patients

WMSI: Wall motion score index; DM: Diabetes Mellitus

ACE/ARB: Angiotensin converting enzyme inhibitor/Angiotensin receptor blocker

CK-MB: Creatine kinase MB fraction

s: Significant & ns: Not significant

Table-VI

Multivariate logistic regression analysis for Change in LVEF (LVEF_e ≥5%) during Follow-up (N=47)

Variables	OR	95% CI	P value
Global systolic lengthening	1.419	1.08-1.86	0.012 ^s
Global Ses	1.891	1.00-3.355	0.048 ^s
WMSI	12.041	0.14-983.46	0.268 ^{ns}
Troponin-I	1.675	0.97-1.18	0.156 ^{ns}

OR: Odds ratio & 95% CI: 95% Confidence interval

N: Number of patients

WMSI: Wall motion score index; Global Ses=Global end systolic strain

s: Significant & ns: Not significant

Table-VII

Analysis from ROC curves to predict recovery of Global LV function in patients with AMI, on the basis of absolute change of LVEF e_e ≥ 5%: cut off value, with respective AUC, for Global systolic lengthening, global Ses and WMSI (N=47)

Variable	Cutoff value	AUC	P value	95% CI
Global systolic lengthening	7.66	0.918	<0.001 ^s	.844-.993
Global Ses	-10.26	0.838	<0.001 ^s	.726-.949

N: Number of patients

s: Significant

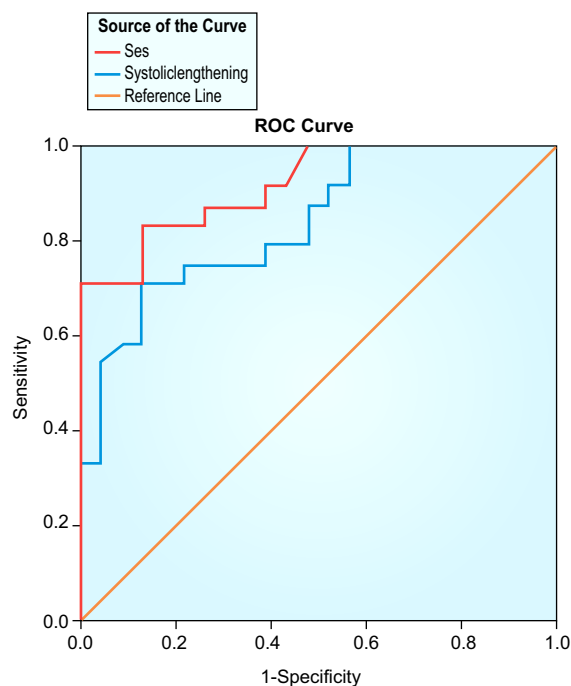


Fig.-1: Comparison of ROC curves of Global systolic lengthening (%), Global Ses and troponin-I for predicting global LV recovery (LVEF ≥5%).

Discussion:

The present research addressed the assessment of myocardial viability and prediction of left ventricular

functional recovery in patients after acute anterior ST-elevated myocardial infarction using Tissue Doppler strain within 48-72 hours of admission and follow-up two dimensional echocardiography at 6 months after baseline investigations. In the present research, 47 consecutive patients admitted with acute anterior STEMI treated with thrombolysis were evaluated. In Group I (n=24) was presented by acute anterior STEMI patients with global left ventricular functional recovery (absolute change of LVEF ≥5%) and those patients who had no left ventricular functional recovery (absolute change of LV<5%) represents Group II (n=23). The mean age of Group I (absolute change of LVEF ≥5%) was 57±5 years and 57±4 years in Group II (absolute change of LV<5%) which was not consistent with that of American research conducted by Sjoerd et al. (2010) but similar with the result of Framingham cohort study.^{20,21} Male predominance was observed in both groups and similar pattern has also been found among many other researches where more than 80% male. This is may be due to male gender more risk for developing coronary artery diseases.²²⁻²³ But there were no statistically significant difference between two groups. On considering, clinical presentation and risk factors such as family history of coronary artery diseases, diabetes mellitus, hypertension and smoking status were compared in both groups which revealed no significant difference between two groups that were similar to the research of Sucu et al. (2003) and Yang et al. (2005).²²⁻²³ Results of biochemical investigations were

compared in both groups. There were significantly lower level of CK-MB and troponin-I in patients with global left ventricular functional recovery (Group I, mean value 49.70 ± 21.80 ng/mL and 13.74 ± 8.35 ng/mL respectively) than patients without left ventricular functional recovery (Group II, mean value 71.02 ± 31.21 and 29.36 ± 15.85 ng/mL respectively) during admission which reflects less myocardial injury, more viable myocardium and potential for recovery of left ventricular function. Similar results were observed in the research of Antoni et al. (2010).²⁴ There was no statistically significant difference in serum creatinine between two groups. Standard treatment protocol of acute ST-elevated myocardial infarction was followed and the mean time from onset of symptom to thrombolytic therapy was 7.89 ± 2.51 hrs and 8.61 ± 2.33 hrs in Group I and Group II respectively. This parameter did not show statistically significant difference between two groups that was also similar to other researches.^{22, 24, 25} Time interval between onset of chest pain and thrombolysis is an important factor for outcome of the patients. Drugs such as ACE/ARB and statin had no statistically significant difference between two groups.

On considering echocardiographic characteristics, mean WMSI was lower in Group I than Group II. Regarding LVEF, higher left ventricular function in Group I (absolute change $\geq 5\%$) and lower left ventricular function in Group II (absolute change $< 5\%$) reflects better recovery in Group I which signify the adequate amount of viable myocardium in Group I. While looking for tissue Doppler strain parameters mean Global systolic strain (Ses) was lower in both groups which indicates significant myocardial injury but there were less negative strain value in Group II (-8.66 ± 2.09) than in group I (-11.36 ± 2.26) with lesser viable myocardium in Group II. Mean global post systolic strain (Spss) was almost similar between two groups and there's no statistically significant difference. This is a unique finding in our research. According to Vartdal et al. (2012) Duration of Systolic lengthening is a novel strain parameter to predict segmental recovery and identification of viable myocardium.¹⁷ But no research demonstrated the significance of systolic lengthening to predict global functional recovery. In the present research, we have developed a new marker of strain imaging; global systolic lengthening that was the average of all segmental systolic lengthening and mean global systolic lengthening was statistically significant between two groups. Substantial relations between global Systolic lengthening and change of LVEF, Troponin-I, global Ses and WMSI were demonstrated. Univariate logistic regression analysis showed global systolic lengthening, global LV strain, level of troponin I, level of CK-MB and

WMSI, are related to change in LVEF at 6-months follow-up after acute myocardial infarction. On the other hand, multivariate analysis has shown that global systolic lengthening is a predictor for change in LVEF during follow-up. In addition, ROC curve analysis showed a cutoff value for global end systolic strain of 10.26% demonstrated to be predictive for recovery of LV function at 6-months follow-up after acute myocardial infarction with a sensitivity of 75% and a specificity of 88%. A cut off value for global systolic lengthening of 7.66% demonstrated to be predictive for recovery of LV function at 6-months follow-up after acute myocardial infarction with a sensitivity of 83.33% and a specificity of 87.00%. In the present research, we showed that global systolic lengthening is an independent parameter of tissue Doppler strain that can detect viable myocardium and predict left ventricular functional recovery. Along with global systolic end systolic strain, global systolic lengthening can accurately detect myocardial viability after myocardial infarction.

Limitations of the research

1. Smaller sample size in a single centre with focusing anterior AMI treated successfully with thrombolysis < 12 hours after symptom onset.
2. Assessing resting WMAs alone cannot predict the transmural extent of AMI, which is best evaluated by MRI.
3. As Coronary angiography was not performed, so vessel anatomy and amount of flow cannot established.

Conclusion and recommendation

Tissue Doppler strain particularly global systolic lengthening was an independent predictor of recovery in anterior ST-elevated Myocardial infarction. Using a cutoff value of 7.66% for baseline global systolic lengthening strain, a sensitivity of 83% and a specificity of 87% were obtained to classify recovery of LV function at 6-months follow-up. Global systolic lengthening and global LV Ses strain can be used to assess myocardial viability and prediction of recovery after myocardial infarction but high Troponin-I and Wall Motion Score index should not use an independent tool to assess viability.

Conflict of interest

The authors declare that there are no conflicts of interest.

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