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Original article

Role of Left Ventricular Twist and Torsion in Assessment of Reperfusion in Acute ST-Elevation Myocardial Infarction Patients

Ayman Gad^a; Mohamed Sayed Bashandy^a; Abd Elrahman Abd El-Gawad Sharaf^b

Department of Cardiology, Damietta Faculty of Medicine, Al-Azhar University, Egypt^[a]

Department of Cardiology, Faculty of Medicine, Al-Azhar University, Egypt^[b]

Corresponding author: **Ayman Gad**

Email: drgadcardio@gmail.com

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ABSTRACT

Background: Myocardial infarction is an acute emergency which needs rapid assessment, especially for myocardial reperfusion.

Aim of the work: To evaluate left ventricle [LV] twist/torsion for assessment of myocardial reperfusion after acute ST-elevation myocardial infarction [STEMI] and to explore its relationship with LV function after 3 months.

Patients and Methods: Forty-five patients with acute STEMI [30 anterior and 15 inferior] with a single culprit lesion were analyzed against 50 healthy subjects. Apical and basal rotations and LV twist/torsion were measured by two-dimensional speckle tracking imaging.

Results: LV twist [13.5 ± 2.4 vs 20 ± 0.5] and LV torsion [1.8 ± 0.2 vs 2.9 ± 0.1 , $P=0.001$] were reduced in all STEMI patients. Basal rotation was larger in anterior STEMI than in inferior STEMI and control [-8.09 ± 0.7 vs -3.1 ± 1.03 - 6.2 ± 0.6 successively], while apical rotation was significantly lower in anterior STEMI. LV twist and torsion were lower in anterior vs inferior STEMI [11.9 ± 1.1 vs 16.6 ± 1 and 1.7 ± 0.1 vs 2.1 ± 0.1 , $P=0.001$ respectively]. There was moderate positive correlation between baseline LV torsion and LV ejection fraction at 3 months [$r=0.500$, $P=0.001$], while negative moderate correlation between baseline LV torsion and LV volumes [LV end-diastolic volume & LV end-systolic volume] [$r=-0.444$ & $r=-0.479$ respectively] was reported. Post reperfusion, the best cut-off point of LV torsion predicting LV remodeling was 1.9 with sensitivity 81.3% and specificity 82.8% [AUC =0.85].

Conclusion: Global LV torsion was decreased in acute STEMI patients, soon after reperfusion it showed marked improvement in LV torsion/twist. LV torsion early after reperfusion can predict LV remodeling at 3 months follow up.

Keywords: Left ventricle; Rotation; Myocardial infarction; Speckle tracking.

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* Main subject and any subcategories have been classified according to researchers' main field of study.

INTRODUCTION

Acute ST- segment elevation myocardial infarction [STEMI] is a major cause of premature death and heart failure acutely and in the longer term. It results from total obstruction to coronary artery blood flow associated with appearance of ST segment elevation on the electrocardiogram. Emergency management is mandatory to ensure coronary reperfusion, and limiting extent of myocardial damage and possibility of heart failure or early death [1].

Outcomes in management of STEMI have dramatically improved in last years. This is mainly due to development of reperfusion therapy and modern pharmacotherapy. Nevertheless, left ventricle [LV] remodeling [LVr] is still commonly present and, among those with anterior wall STEMI, affects 30% to 35% of patients[2].

Cardiac remodeling is a dynamic ongoing process for up to 24 months after acute myocardial infarction [3].

To evaluate cardiac function, it is mandatory to understand the helical architecture of myocardial fibers and evaluate fibers regarding thickening, shortening and torsion movements in relation to cardiac axis throughout systole and diastole. Torsion of the LV is defined as wringing motion of the ventricle around its long axis produced by contracting myofibers within the LV wall [4].

Measurement of LV torsion deformation by speckle tracking echocardiography has been introduced as a sensitive marker of LV function and validated against sonomicrometry and magnetic resonance imaging [5].

AIM OF THE WORK

The present study designed to evaluate LV twist/torsion for assessment of myocardial reperfusion after acute ST-elevation myocardial infarction [STEMI] and to explore the relationship between LV twist/torsion at presentation and LV function after 3-months.

PATIENTS AND METHODS

This prospective study included [group 1] 45 patients presented with first attack of acute ST segment elevation myocardial infarction [STEMI] admitted at cardiology department at Al-Azhar

university hospital, Damietta between July 2014 and June 2017. Patients with evidence of prior myocardial infarction, significant stenosis in any coronary artery other than culprit one, severe valvular disease, previous heart surgery, atrial fibrillation, have any condition interfering with ability to comply, have hemodynamic instability during index admission or with poor echo window in which 2 or more than 2 segments could not be visualized were excluded from the study. As a control group, we included 50 healthy subjects [group 2] who had no risk factors of cardiovascular disease and no history of cardiovascular disease with normal resting electrocardiographic and echocardiographic findings. Persons in control group were age and sex matched with our patients. All patients gave informed consent.

All study subjects underwent complete history taking, physical examination, routine laboratory investigations, resting ECG and resting echocardiography.

Echocardiographic studies

All patients underwent transthoracic echocardiography using a standard commercial system [Philips IE 33 Ultrasound, Bothell, WA. 98021 USA] using an X5-1 phased array sector probe. All studies were ECG-gated and performed according to the following protocol:

- I. Two-dimensional parasternal short axis view at both base & apex were acquired before reperfusion [0].
- II. Two-dimensional and three-dimensional echocardiography 24 to 48 hours after successful reperfusion, defined as Thrombolysis in Myocardial Infarction Trial [TIMI] grade 3 flow [1].
- III. Two-dimensional and three-dimensional echocardiography after three months [2].

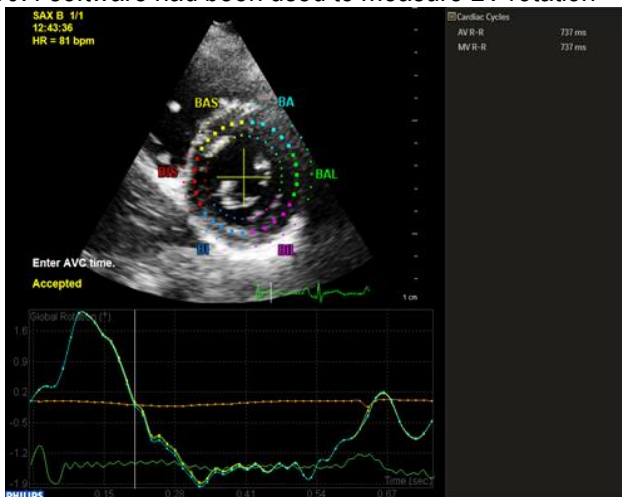
1) Two-Dimensional Echocardiography:

The following variables were obtained: LV volumes, LV ejection fraction [6], LV length and wall motion score index [WMSI][7].

All measurements were obtained following the guidelines of the American Society of Echocardiography[7].

Speckle tracking echocardiography [STE]:

i] Torsional Mechanics and LV circumferential strain Analysis: Parasternal short-axis images of LV were acquired at 2 different levels: basal level, identified by the mitral valve, and apical level, as the smallest cavity achievable distal to the papillary muscles. The endocardial border was traced at an end-systolic frame, and the regions of interest [ROI] had been chosen to fit the whole myocardium. Q-lab 10.4 software had been used to measure LV rotation



from these images. Counterclockwise rotation was determined as a positive value while clockwise rotation as a negative value on viewing from apex [figure 1]. Twist of LV is the difference [in degrees] between apical and basal rotations considering isochronal time points. Torsion of LV then had been measured as ratio between twist of LV [in degrees] and the longitudinal length between LV apex and mitral plane at end diastole [in cm] [8].

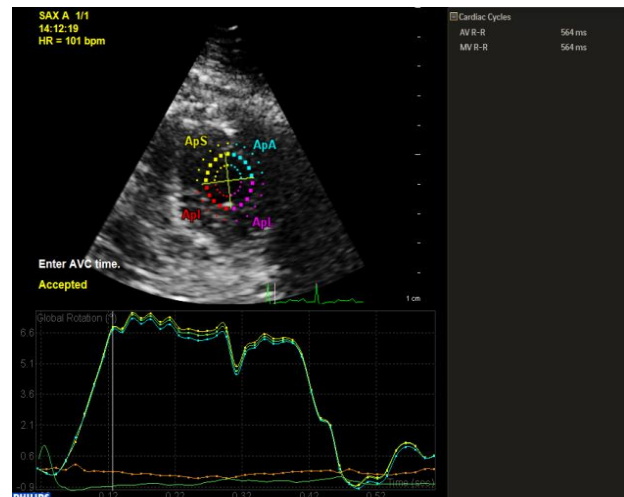


Figure [1]: Shows measurement of LV basal & apical rotation using Q-lab 10.4 for commercially available Phillips machine IE 33.

ii] Global longitudinal Strain analysis: We analyzed the digitally stored clips offline using Q lab 10.4 software. The software automatically detected the endocardium at end-systole, both regional and global longitudinal strain were calculated.

3] Three-Dimensional Echocardiography: Three-dimensional LV end-diastolic volume [LVEDV], LV end systolic volume, and LV ejection fraction [LVEF] were obtained using Q-lab 10.4 [Figure 2].

II.2.b: Invasive Coronary Angiography:

This was performed according to current guidelines [AHA 2013 & ESC 2012 STEMI guidelines] to identify the infarct-related artery, and successful angioplasty when needed, achieving Thrombolysis in Myocardial Infarction grade III flow.

II.2.c: Follow Up:

Three months after presentation, two-dimensional and three-dimensional echocardiography was performed. Then patients were classified in to two groups, those with LV remodeling and those without LV remodeling. The definition of remodeling

was an increased LVEDV of >15% compared with the echocardiographic study performed at discharge^[9].

Statistical methods

Version 21 of SPSS [statistical package of social sciences] was used for data entry and statistical analyses [SPSS Inc., Chicago, IL, USA by Baron and Kenny 1986] [10]. Based on receiver-operating characteristic [ROC] curves, the best cut-off value was obtained as the optimal point with the highest sum of sensitivity and specificity for predicting LV remodeling.

RESULTS

Demographic & clinical characteristics:

Thirty [60%] of control group were males and 20 [40%] were females while in patient group, there were 30 males [66.7%] and 15 females [33.3%] and there was statistically insignificant difference between both groups regarding sex distribution. Twenty-three patients [51.1%] were hypertensive, 15 patients [33.3%] have positive family history of CAD, 26 patients [57.8%] were smoker and 22 [48.9%]

patients were dyslipidemic. Twenty-eight patients underwent routine early PCI after receiving thrombolytic therapy, nine patients underwent primary PCI [3 with anterior STEMI and 6 with inferior STEMI] and eight patients underwent rescue PCI [7

patients with anterior and 1 patient with inferior STEMI]. Among those with routine early PCI 20 patients were presented with anterior STEMI and 8 patients with inferior STEMI [4 of them had RCA as the culprit and 4 of them have LCX as the culprit].

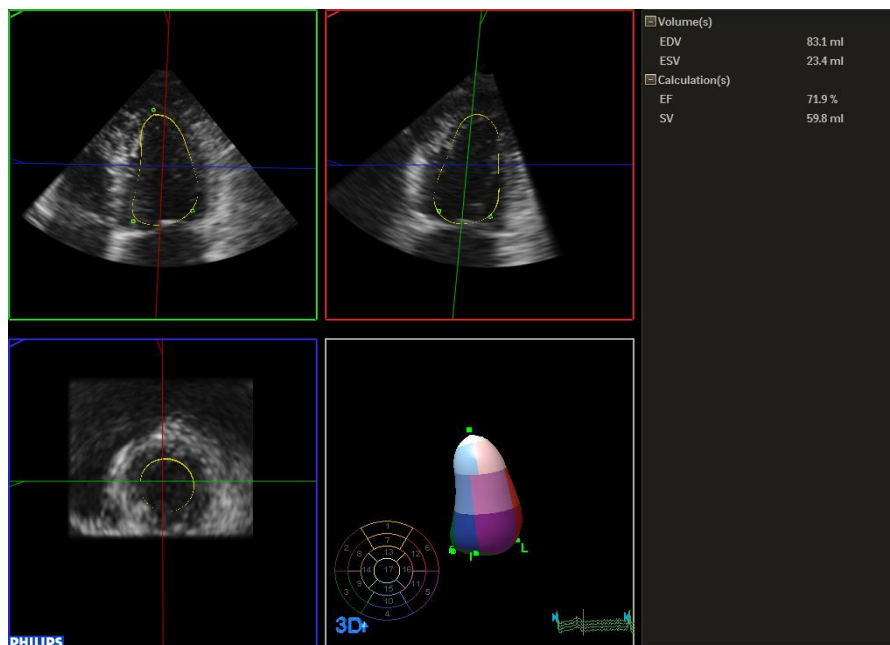


Figure [2]: Shows measurement of LV volumes & EF by 3 D ECHO using Q-lab 10.4 for commercially available Phillips machine IE 33.

Comparison between two groups as regard baseline echocardiographic parameters:

WMS index and LV volumes were larger in group 1 than in group 2 with. LV ejection fraction, LV GLS, LV apical rotation, LV torsion, LV twist and LV EF were lower in group 1, while basal rotation had no statistically significance difference between both groups [Table 1]. Table 2 shows comparison between anterior & inferior STEMI patients as regard echocardiography parameters. Both LV twist and LV torsion were decreased in all STEMI patients compared with control group, however there were markedly decreased in anterior STEMI patients comparing to those with inferior STEMI. In anterior STEMI patients LV apical rotation was markedly decreased than both control group and those with inferior STEMI. In contrast, basal rotation was significantly reduced with inferior STEMI [Table 2].

Effect of reperfusion on LV rotational mechanics

Early after reperfusion there was statistically significant increase in LV apical rotation, LV twist &

LV torsion values compared to baseline measurement [Table 3].

Follow up and prediction of LV remodeling:

Acute STEMI patients without LV remodeling showed increased LV twist and torsion to that near of normal. In anterior infarction, as the apical function was improved, basal rotation was also decreased to near normal. However, in patients with remodeling, apical rotation and LV twist were reduced, while the basal rotation was still high. [Table 4]

Correlation between LV remodeling & LV rotational mechanics:

There was moderate positive correlation between baseline LV twist1 and LV ejection fraction at follow up [r= 0.503, P =0.001], while moderate negative correlation between baseline LV twist1 and LV volumes [LVEDV & LVESV] [r= -0.467, P =0.001 & r = -0.492, P = 0.001]. There was moderate positive correlation between baseline LV torsion1 and LV ejection fraction at follow up [r = 0.500, P= 0.001], while negative moderate correlation between baseline LV torsion1 and LV volumes [LV EDV & LV

ESV] [r=0.444, P= 0.002 & r=0.479, P = 0.001]. There was strong positive correlation between baseline LV apical rotation 1 and LV ejection fraction at follow up [r = 0.809, P = 0.001], while negative moderate correlation between baseline LV apical rotation 2 and LV volumes [LV EDV & LV ESV] [r = -0.738, P = 0.003 & r = -0.779, P = 0.001]. There was a strong positive correlation between baseline LV GLS 1 and LV ejection fraction at follow up [r = 0.820, P = 0.032], while negative moderate correlation between baseline LV GLS 1 and LV volumes [LV EDV & LV ESV] [r = -0.763, P = 0.001 & r = -0.720, P = 0.004]. The best cut off point for baseline LV twist

[immediately after reperfusion] that could predict LV remodeling at 3 months follow up was 13.5° with sensitivity 81%, specificity 83% and AUC=0.84 [figure 3].

The best cut off point for baseline LV torsion [immediately after reperfusion] that could predict LV remodeling at 3 months follow up was 1.9°/cm with sensitivity 81.3%, specificity 82.8 % and AUC=0.85. The best cut off point for baseline LV apical rotation [immediately after reperfusion] that could predict LV remodeling at 3 months follow up was 5.6° with sensitivity 81%, specificity 86.2% and AUC=0.80.

Table [1]: Shows Comparison between two groups as regard baseline echocardiographic parameters

Variable	Group 1 [n=45]	Group 2 [n=50]	P value
WMSI	1.6±0.3	1	0.001
GLS	14.2±2.1	-21.1±1.4	0.001
B rot	-6.4±2.5	-6.2±0.6	0.663
Ap rot	7.4±4.4	13.7±0.9	0.001
Torsion	1.8±0.2	2.9±0.1	0.001
Twist	13.5±2.4	20±0.5	0.001
LV EDV	111.9±10.7	88.2±9.2	0.001
LV ESV	55±9.9	28.9±5.6	0.001
LV EF	51.1±5	67.2±4.2	0.001
LV CS B%	-18.2±4	-20.6±1.5	0.001
LV CS apex%	-16.9±5.8	-24.7±1.8	0.001

WMSI [wall motion score index], GLS [global longitudinal strain%], B rot [left ventricular basal rotation], Ap rot [left ventricular apical rotation], LV EF [left ventricular ejection fraction], LV EDV [left ventricular end diastolic volume], LV ESV [left ventricular end systolic volume], LV CS B [left ventricular basal circumferential strain] & LV SC apex [left ventricular apical circumferential strain].

Table [2]: Shows comparison between patients with anterior STEMI & those with inferior STEMI as regard baseline echocardiographic parameters.

Variable	Group 2	Group 1		P1	P2	P3
		Anterior [n=30]	Inferior [n=15]			
WMSI 1	1	1.8±0.24	1.3±0.17	0.001	0.043	0.001
GLS1	-21.1±1.4	13.8±2.1	14.9±2.1	0.001	0.001	0.112
B rot 0	-6.2±0.6	-8.09±0.7	-3.1±1.03	0.02	0.001	0.001
B rot 1		-7.6±0.7	-4.9±1.5			0.001
Ap rot 0	13.7±0.9	4.3±0.5	13.4±0.9	0.001	0.622	0.001
Ap rot 1		5.5±1	13.6±0.9			0.001
Torsion 0	2.9±0.1	1.7±0.1	2.1±0.1	0.001	0.001	0.001
Torsion 1		1.8±0.2	2.4±0.2			0.001
Twist 0	20±0.5	11.9±1.1	16.6±1	0.001	0.003	0.001
Twist 1		13.1±1.5	18.6±1.6			0.001
LV EDV 1	88.2±9.2	116.2±10.4	103.4±4.6	0.001	0.001	0.001
LV ESV 1	28.9±5.6	59.4±8.5	46.2±6.1	0.001	0.001	0.001
LV EF 1	67.2±4.2	48.9±4	55.4±4.1	0.001	0.001	0.001
LV CS B0	-20.6±1.5	-20.8±1.5	-13.2±2.4	0.031	0.001	0.001
LV CS B 1		-20.1±1.3	-17.5±2.6			0.002
LV CS A 0%	-24.7±1.8	-13.2±2.5	-24.2±2.7	0.001	0.723	0.001
LV CS A1%		-16.3±3.3	-19.3±11.9			0.197

*P1 between group 2 and anterior, P2 between Group 2 and inferior P3 between anterior and inferior. **WMSI [wall motion score index], GLS [global longitudinal strain%], B rot [left ventricular basal rotation], Ap rot [left ventricular apical rotation], LV EF [left ventricular ejection fraction], LV EDV [left ventricular end diastolic volume], LV ESV [left ventricular end systolic volume], LV CS B [left ventricular basal circumferential strain] & LV SC A [left ventricular apical circumferential strain]; 0: refers to baseline [before reperfusion], 1: after reperfusion.

Table [3]: Shows effect of reperfusion on LV rotational mechanics

Variable	Base line		After reperfusion		P
	Mean	SD	Mean	SD	
Twist	13.5	2.4	14.9	3.1	0.001
Torsion	1.8	0.2	2.1	0.3	0.001
Apical Rotation	7.4	4.4	8.2	2.9	0.002
Basal Rotation	-6.4	2.5	-6.7	1.6	0.031

Table [4]: Shows comparison between two groups those with remodeling & those without remodeling as regard echocardiographic parameters

Variable	Group 1		P 1
	Remodeling		
	[With LV recovery [n=29]	[With remodeling [n=16]	
Time	2.5±1.1	4.6±1.4	0.001
WMSI 1	1.5±0.2	1.9±0.2	0.001
WMSI 2	1.1±0.1	1.6±0.2	0.001
GLS1	14.6±1.8	13.3±2.5	0.058
GLS 2	19.1±4	15.8±1	0.003
B rot 0	-6±2.6	-7.1±2.2	0.177
B rot 1	-6.6±1.4	-6.9±2	0.588
B rot 2	-6.4±0.7	-6.7±1.5	0.370
Ap rot 0	8.2±4.4	5.9±3.9	0.096
Ap rot 1	9.2±3.8	6.4±3.7	0.021
Ap rot 2	10.1±2.7	6.5±3.4	0.001
Torsion 0	1.9±0.2	1.7±0.2	0.007
Torsion 1	2.2±0.3	1.8±0.2	0.001
Torsion 2	2.3±0.3	1.8±0.2	0.001
Twist 0	14.1±2.2	12.3±2.4	0.016
Twist 1	16±2.6	12.8±2.3	0.001
Twist 2	16.6±2.5	13.3±2.2	0.001
LV EDV 1	108±7.2	119.1±12.5	0.004
LV EDV 2	102.6±8.1	130.6±15.6	0.001
LV ESV 1	51.5±8.4	61.2±9.7	0.002
LV ES V 2	45.1±9.4	70.8±12.8	0.001
LV EF 1	52.4±5.1	48.7±4	0.016
LV EF 2	56.3±5.5	46±4.4	0.001

*WMSI [wall motion score index], GLS [global longitudinal strain%], B rot [left ventricular basal rotation], Ap rot [left ventricular apical rotation], LV EF [left ventricular ejection fraction], LV EDV [left ventricular end diastolic volume], LV ESV [left ventricular end systolic volume], LV CS B [left ventricular basal circumferential strain] & LV SC apex [left ventricular apical circumferential strain]; 0: refers to baseline [before reperfusion], 1: after reperfusion, 2: after 3 months.

DISUCSSION

The results of this study showed that LV torsion is significantly impaired early after acute myocardial infarction [AMI], due to either a reduction of basal or apical rotations. In additionally torsion early after STEMI was significantly related to the occurrence of LV remodeling at 3-month follow-up. Also, LV torsion was related to LV systolic function. These results show agreement with **Takeuchi et al.**^[11] who concluded that there was significant reduction in LV twist early post AMI & also there was significant correlation between LV twist & LV ejection fraction.

Our results showed also agreement with **Han & his colleagues**^[12], they studied 35 AMI patients

before and one month after revascularization, as well as in 32 normal controls. They found that prior to revascularization, LV peak global and regional torsion in patients with AMI were significantly decreased as a result of reduced apical and basal rotation in relation to those of control group; one month after revascularization, there were significant changes in peak rotation at either the base or apex relative to pre-revascularization values. Similarly, peak global and regional LV torsion were increased significantly. Global torsion inversely correlated with EDV [r0.605, P=0.028] and ESV [r0.638, P=0.019]; and positively correlated with LVEF [r0.630, P=0.021].

Spinelli et al.^[13] studied 75 patients with initial anterior wall STEMI before and after PCI and at 6 month follow-up aiming to identify impact of global LV torsion on reverse remodeling post AMI, they found that there was significant reduction in LV torsion early before revascularization, LV torsion showed significant improvement after revascularization especially in patients who showed reverse LV remodeling at follow up. They concluded that improvement of global LV torsion following coronary artery revascularizations the major predictor of reverse LV remodeling, $1.34^\circ/\text{cm}$ for LV torsion after revascularization [sensitivity 88 % and specificity 80 %] was the optimal cutoff value in predicting reverse remodeling of LV.

Our results showed also significant decrease in LV torsion early before revascularization and significant improvement after revascularization especially in patients who showed recovery of LV functions at follow up, however our results showed cut off point $1.9^\circ/\text{cm}$ for LV torsion after reperfusion [Torsion 1] [sensitivity 81.3 %, specificity 82.8 %] in predicting LV remodeling, this value is greater than it showed by Spinelli and his colleagues, this difference may be related to difference in patients criteria as regard they studied only anterior STEMI patients, also they did not exclude patients with other significant coronary artery stenosis. In our study we had both patients with anterior & inferior AMI; also, any patient with significant stenosis at any coronary artery other than infarct related artery was excluded from our study^[13].

Results of our study shows also great match with results of a study done by **Nucifora et al.**^[14]. They studied 120 patients with a first ST-segment elevation AMI. All patients underwent primary percutaneous coronary intervention. Forty-eight hours later, speckle tracking echocardiography was performed for assessment of LV torsion; also, they assessed infarct size by myocardial contrast echocardiography. At 6-month follow-up, LV volumes and LV ejection fraction were reassessed to identify patients with LV remodeling. Their results showed that peak LV torsion in AMI patients was significantly impaired [$1.54 \pm 0.64^\circ/\text{cm}$ vs $2.07 \pm 0.27^\circ/\text{cm}$, $P < 0.001$] compared to control group. Their results also showed that peak LV torsion showed modest significant incremental value in prediction of remodeling. By ROC curve analysis, $1.44^\circ/\text{cm}$ as a

peak LV torsion provided the best sensitivity [95%] and specificity [77%] predicting LV remodeling^[14].

Our results came in agreement with these results. Although our results showed higher values for LV torsion than their results, this may be due to presence of 41 patients included in their study have multi vessel disease represent 34% of total patients number, this may contributed to more reduction of LV torsion values compared to this study which included only patients with single vessel disease.

There was agreement between our results and results of a study done by **Seong-Mi and his colleagues**; they studied 66 patients with AMI, 35 of them with anterior and the remaining with inferior infarction, all had a single culprit lesion treated with successful primary percutaneous coronary intervention, in addition to thirty age-matched healthy subjects as a control group. Basal and apical rotations were obtained and then LV torsion and twist were measured by 2D speckle tracking imaging. They were aiming to evaluate impacts of AMI on LV rotational mechanics and to compare the alterations in basal and apical rotation between patients with anterior and inferior AMI. They found that compared with normal, LV twist was reduced in all AMI patients. Basal rotation was larger in anterior AMI than in those with inferior AMI and normal, although apical rotation was less in anterior AMI. In patients with anterior AMI, by univariate analysis, several variables were significantly related to LV recovery: as LV torsion, twist, deceleration time [DT], LV ejection fraction, and apical rotation. However, by multivariate analysis, only LV torsion and DT were independently associated with recovery of LV. Based on ROC curve analysis, sensitivity and specificity for prediction of LV recovery were 100.0 and 50.0% [AUC 0.778], using $1.54^\circ/\text{cm}$ as a cut-off value of LV torsion. While, in inferior AMI patients, only basal rotation was related to LV recovery^[15]. In the previous study, the LV torsion was significantly reduced in patients with AMI, but there was no significant difference between patients with inferior AMI and anterior AMI although WMS index and LV ejection fraction were worse in anterior AMI patients, this study showed that LV torsion was much lower in anterior than inferior STEMI, this difference may be due to difference in number of patients, also all of their patients were treated with primary PCI while in this study only 9 patients treated with primary PCI

while rest of patients treated with pharmaco-invasive strategy. Basal LV rotation was increased significantly in patients with anterior AMI and this was in agreement of our results.

Study limitations: This study included small number of patients. Also, we did not assess the myocardium with other imaging modalities, such as cardiac magnetic resonance imaging. Accuracy of speckle tracking is dependent on two-dimensional image quality and frame rates. Also, 2D speckle tracking cannot abolish errors introduced by through-plane motion.

Conclusion: LV twist and torsion are reduced in both anterior and inferior STEMI; they showed significant improvement early after successful reperfusion. Our findings suggest that the basal rotation plays an important role in LV function. In addition, baseline measurements of LV rotational mechanics after STEMI could identify patients who are prone to develop LV remodeling and may benefit from aggressive therapy to prevent heart failure and poor outcome.

Financial and Non-Financial Relationships and Activities of Interest

None

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