

# Long-term follow up of left ventricular remodeling in ST segment myocardial infarction patients after primary percutaneous coronary intervention

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## Introduction

The adaptive reaction known as left ventricular remodeling after ST-elevation myocardial infarction (STEMI) involves both the infarct zone and remote zones, and it lasts for at least 2 years after STEMI. In STEMI patients having primary percutaneous coronary intervention (PPCI), this study was conducted to look at long-term consequences of left ventricular remodeling.

## Patients and methods

Ninety-three patients were successfully treated by primary PCI more than 2 years ago at Assiut University Heart Hospital were enrolled in this study. Detailed history, complete general examination and local cardiac examination were done. Electrocardiography, two-dimensional echocardiography before primary PCI, at discharge and 6 months after PPCI, coronary angiographic and PPCI data from patient admission sheet. Patients were examined by transthoracic 2DE using Phillips ie33 ultrasound system device.

## Results

Thirty-six (38.7%) patients were determined to have left ventricular remodeling when their left ventricular end diastolic volume index increased by more than 20%, compared with 57 (61.3%) patients who did not. Mean age of patients  $56.31 \pm 11.11$  years and mean overall duration of follow up was  $3.69 \pm 0.45$  years. The long-term predictors of left ventricular remodeling were: extensive anterior MI ( $P < 0.001$ ), left anterior descending as infarct-related artery ( $P = 0.03$ ), left ventricular end systolic volume index ( $P = 0.04$ ) and wall motion index ( $P < 0.001$ ).

## Discussion

Patients who had extensive anterior MI, a wall motion score index more than 1.5, and left anterior descending as infarct-related artery were seen as having a high probability of LV remodeling following PPCI. Only wall motion score index more than 1.5 and extensive anterior MI were independent indicators of LV remodeling following PPCI.

## Keywords:

left ventricular remodeling, long-term follow-up, primary percutaneous coronary intervention, ST segment myocardial infarction

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## Introduction

Acute myocardial infarction (AMI), a major cause of illness and mortality worldwide. Although it increases survival and preserves left ventricular function, since remodeling still happen, primary percutaneous coronary intervention (PPCI) is the preferred approach for handling ST-elevation myocardial infarction (STEMI) patients [1].

Left ventricular remodeling is changes in the geometry and function of the left ventricle that are a set of molecular, cellular, and interstitial changes brought on by cardiac damage. Thirty percent of STEMI patients develop postinfarct left ventricular remodeling. Left ventricular remodeling assumes a poor prognostic value because it predicts cardiac failure [2].

Left ventricular remodeling is a continuous process that involves the infarct zone and remote zones that

lasts for at least 2 years following STEMI. Increased end-systolic volume and decreased wall thickness in the remote zones are symptoms of long-term left ventricular ejection fraction (LVEF) deterioration [3].

## Patients and methods

This study was done at Cardiovascular Medicine Department, Assiut University Heart Hospital after ethical committee approval and the institutional review board approval (17100704 E) and after fully explaining the study's procedures to all participants, their informed consent was obtained. In this study, 93 patients who

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received primary PCI successfully more than 2 years ago within 12 h of the onset of chest pain or up to 24 h later if there was ongoing ischemia were enrolled, while those who presented with significant mitral regurgitation or valve disease, acute heart failure or cardiogenic shock, or who had permanent pacemaker insertion were excluded.

Detailed history was gathered including cardiovascular risk factors and history of drug intake post-PPCI that improve remodeling. History of re-infarction, re-admission with acute coronary syndrome, re-CAG, re-PCI or coronary artery bypass surgery. Complete general examination and local cardiac examination to detect heart failure, cardiogenic shock or significant valve lesion (adopted from patient admission sheet).

ECG was performed on all patients for diagnosis and location of STEMI (adopted from patient admission sheet).

Two-dimensional echocardiography before primary PCI, at discharge and 6 months after PPCI (adopted from patient sheet).

Coronary angiographic and PPCI data: including infarct related artery (IRA), number of involved vessels, TIMI flow post-PPCI, thrombus aspiration as well as coronary stenting either bare metal or drug eluting (adopted from patient admission sheet).

According to the following protocol, all patients underwent transthoracic two-dimensional echocardiography examinations with a Phillips ie33 ultrasound system device:

(1) The modified Simpson's method was used to measure the LV volumes (LVEDV and LVESV) and the ejection fraction (EF).

LV volume indices were calculated as follows: LV volume indices = LV volumes divided by body surface area.

(2) This is how the wall motion score index (WMSI) was determined: a 17-segment model was used to segment the LV [4]. WMSI = sum of scores of all segments visualized/number of these segments.

Normal or hyperkinetic scored (1), hypokinetic (reduced thickening) scored (2), a kinetic (absence or minimal thickening, e.g. scar) scored (3), and dyskinetic (systolic thinning or stretching, e.g. aneurysm) scored (4) were the categories utilized for scoring.

(3) LV diastolic filling pattern is evaluated using pulsed wave Doppler of transmitral flow during diastole. At baseline, the following variables were calculated:

Early rapid filling wave (E) peak velocity, atrial wave (A) peak velocity, peak E/A wave velocity ratio, and deceleration time.

The studied patients divided into two groups to whom PPCI was done more than or equal to 2 years ago:

(1) Patients in group I (with LV remodeling) have an increase in left ventricular end diastolic volume index (LVEDVI) of greater than 20%.

An informal definition of ventricular remodeling is an increase of at least 20% in LVEDV from the initial postinfarction imaging, yet it is routinely used in follow-up investigations [5].

(2) Group II (without LV remodeling).

The outcome of all patients was assessed regarding the predictors of long-term LV remodeling affecting acute STEMI patients to whom PPCI were done more than 2 years ago.

### Statistical analysis

Data were verified, coded by the researcher and analyzed using IBM-SPSS 24.0 (IBM-SPSS Inc., Chicago, Illinois, USA). Continuous variables were described by mean and SD, while qualitative variables represented by number and percent. Continuous variables of both groups were compared by Student *t* test, and  $\chi^2$  test and Fisher exact test used to compare between categorical variables.

The paired *t* test was used to compare echocardiographic variables between baseline and 6 months. Clinical, angiographic, and echocardiographic variables were incorporated into logistic regression analysis to predict LV remodeling. Multivariate analysis was used to further examine variables that had statistical significance in the univariate analysis.

Statistical significance was defined as *P* value less than 0.05.

### Results

Our study enrolled 93 patients with acute STEMI to whom PPCI were done more than 2 years ago. Mean age of all studied patients  $56.31 \pm 11.11$  years. Mean overall duration of follow up was  $3.69 \pm 0.45$  years. Patients assessed to have LV remodeling who had an increase in LVEDVI more than 20% were 36 (38.7%), compared with 57 (61.3%) patients who did not.

Regarding demographic information, there was statistically insignificant distinction between the two groups ( $P > 0.05$ ) (Table 1).

Regarding electrocardiographic data: the most frequent type of MI among nonremodeling group was extensive inferior MI (38.6%) followed by anterior MI (26.3%) while in case of remodeling group, the most frequent MI was extensive anterior MI (58.3%) followed by extensive inferior MI (25%) with significant difference between both groups ( $P < 0.001$ ) (Table 2).

According to angiographic data, there was a significant difference between the two groups regarding IRAs, with patients who had remodeling experiencing much more left anterior descending (LAD) artery affection (77.8 vs. 40.4%;  $P < 0.001$ ). The number of vessels affected, the use of thrombus aspiration, the type of stent, and the post-PCI TIMI grade did not

significantly differ between the two groups ( $P$  value in all cases  $>0.05$ ) ( $P$  value in all  $>0.05$ ) (Table 2).

Regarding echocardiographic data: patients without remodeling had significantly higher ejection fraction, while patients with remodeling had significantly higher LVEDVI, left ventricular end systolic volume index (LVESVI), and WMSI ( $P < 0.001$ ). Other echocardiographic data from various evaluation times revealed insignificant difference between the two groups ( $P > 0.05$ ) (Table 3).

Regression analysis for prediction of LV remodeling after PPCI: univariate regression analysis revealed that patients with extensive anterior MI location, WMSI more than 1.5, and LAD as IRA were at substantial risk for LV remodeling after PPCI. The only the independent predictors of LV remodeling after PPCI were WMSI more than 1.5 and extensive anterior site of MI by multivariate regression study (odd's ratio; 2.98 and 2.34, respectively) (Table 4).

**Table 1 Baseline data of living patients based on left ventricular remodeling**

	LV remodeling		<i>P</i>
	No ( <i>n</i> =57)	Yes ( <i>n</i> =36)	
Age (years)	55.56±11.77	55.92±10.12	0.78
Sex [ <i>n</i> (%)]			0.49
Male	44 (77.2)	27 (75)	
Female	13 (22.8)	9 (25)	
BMI (kg/m <sup>2</sup> )	26.89±2.75	26.76±2.70	0.81
Body surface area (m <sup>2</sup> )	1.82±0.12	1.79±0.12	0.18
Smoking [ <i>n</i> (%)]			0.50
None	19 (33.3)	15 (41.7)	
Current	26 (45.6)	12 (33.3)	
Ex-smoker	12 (21.1)	9 (25)	
Hospital readmission	2 (3.5)	3 (8.3)	0.29
Re-angiography	1 (1.8)	1 (2.8)	0.62
Previous PCI	1 (1.8)	3 (8.3)	0.10
Heart failure	9 (15.8)	9 (25)	0.20
Duration of follow up (years)	3.87±0.59	3.55±0.44	0.45

**Table 2 ECG and angiographic findings in studied patients based on remodeling**

	LV remodeling [ <i>n</i> (%)]		<i>P</i>
	Without ( <i>n</i> =57)	With ( <i>n</i> =36)	
ECG			<b>&lt;0.001</b>
Extensive anterior MI	11 (19.3)	21 (58.3)	
Extensive inferior MI	22 (38.6)	9 (25)	
Anterior MI	15 (26.3)	3 (8.3)	
Inferior MI	9 (15.8)	3 (8.3)	
Symptoms to balloon >4 h	15 (26.3)	5 (13.9)	0.12
Infarct-related artery			<b>&lt;0.001</b>
LAD	23 (40.4)	28 (77.8)	
RCA	22 (38.6)	7 (19.4)	
LCx	12 (21)	8 (22.2)	
TIMI flow >2	57 (100)	36 (100)	–
Multivessel disease	24 (42.1)	13 (36.1)	0.36
Thrombus aspiration	7 (12.3)	2 (5.6)	0.24
Stenting	51 (89.5)	35 (97.2)	0.12
Type of stent			0.52
Bare metal stent	36 (63.2)	22 (61.1)	
Drug eluting stent	15 (26.3)	13 (36.1)	
Total revascularization	31 (54.4)	18 (50)	0.42

Data expressed as frequency (percentage), mean (SD). *P* value was significant if  $< 0.05$ .

## Discussion

The postinfarct left ventricular remodeling that causes congestive heart failure still poses a serious challenge despite the most recent methods and advancements in the management of MI [6].

Out of the 93 patients who were studied, 36 (38.7%) experienced LV remodeling, which was consistent with the those of Bolognese *et al.* [7], who discovered that 30% of 284 patients who underwent primary PCI for AMI experienced LV dilatation with more than 20% increase in LVEDVI. Also in line with the findings of Mousa *et al.* [2], 49 (32.2%) patients who were thought to have LV remodeling underwent primary PCI for AMI, while 103 (67.8%) patients did not. Additionally, a review research from 2011 in the *European Heart Journal* reported that 30% of individuals with a history of myocardial infarction experience postinfarct ventricular remodeling [8]. While our results were discordant with Pieter *et al.* [9] who showed that LV remodeling happened in 48% of their studied patients in the first 12 months of follow up after PPCI, this is may be related to bigger sample size (1995 patients) in their study compared to our patients (93 patients), also, their patients were little bit older than ours (mean age  $60 \pm 12$  vs.  $55 \pm 11$  years).

According to clinical data and risk factors, insignificant difference between LV remodeling group and non-LV remodeling group (including age, sex and risk factors as hypertension, DM, smoking, or dyslipidemia). These findings are in line with those of Zaliaduonyte-Peksiene *et al.* [10] and Loboż-Grudzięń *et al.* [5].

**Table 3 Echocardiographic findings in patients based on remodeling**

	LV remodeling		P
	No (n=57)	Yes (n=36)	
<b>LVEDVI (ml/m<sup>2</sup>)</b>			
At discharge	91.82±50.56	115.40±37.59	<b>&lt;0.001</b>
After 6 months	86.63±21.09	117.78±36.64	<b>&lt;0.001</b>
After 2 years	66.79±48.13	114.67±37.39	<b>&lt;0.001</b>
<b>LVESVI (ml/m<sup>2</sup>)</b>			
At discharge	49.72±34.29	57.46±23.13	<b>&lt;0.001</b>
After 6 months	34.68±13.07	59.71±23.62	<b>&lt;0.001</b>
After 2 years	45.95±36.41	59.70±32.81	<b>0.04</b>
<b>Ejection fraction (%)</b>			
At discharge	63.17±8.67	46.28±9.68	<b>0.02</b>
After 6 months	52.14±9.71	51.66±8.41	0.19
After 2 years	53.45±11.83	51.82±8.42	0.58
<b>Wall motion index</b>			
At discharge	1.31±0.32	1.51±0.31	<b>&lt;0.001</b>
After 6 months	1.25±0.14	1.45±0.31	<b>&lt;0.001</b>
After 2 years	1.17±0.34	1.43±0.43	<b>0.03</b>
<b>Peak E wave (cm/s)</b>			
At discharge	22.50±24.74	57.88±21.91	0.07
After 6 months	45±15.65	90.33±28.72	0.30
After 2 years	68.89±24.07	77.22±27.87	0.13
<b>Peak A wave (cm/s)</b>			
At discharge	38.10±16.76	49.61±22.03	0.60
After 6 months	31.10±12.34	67.33±13.50	0.24
After 2 years	70.63±24.73	75.30±25.54	0.38
<b>Peak E/A ratio (&lt;1)</b>			
At discharge	19 (33.3)	17 (47.2)	<b>&lt;0.001</b>
After 6 months	36 (63.2)	22 (61.1)	0.21
After 2 years	35 (61.4)	19 (52.8)	0.27
<b>Deceleration time (s)</b>			
At discharge	219±27.07	185.69±48.97	0.80
After 6 months	189±25.45	222±79.69	0.13
After 2 years	246.43±66.76	234.27±58.98	0.43

Data expressed as frequency (percentage), mean (SD). P value was significant if < 0.05.

**Table 4 Predictors of LV remodeling**

Variables	Odd's ratio	95% CI	P
Extensive anterior MI	2.34	1.45–5.05	<0.001
LAD as infarct-related artery	2.11	1.83–4.55	0.03
LVESVI	2.10	1.76–4.22	0.04
Wall motion index	2.98	1.44–5.90	<0.001
Ejection fraction (<45%)	1.23	0.45–1.45	0.34

As opposed to Pop *et al.*'s [6] findings, who discovered that female sex, smoking, and dyslipidemia were risk factors associated with postinfarct LV remodeling in a sample of 105 STEMI patients treated with primary PCI.

According to electrocardiographic data, our study revealed that patients with extensive anterior locations of MI were at a great risk for LV remodeling following AMI, making it an independent predictor of LV remodeling. Also, the most frequent types of MI among died patients were anterior (55.9%).

These findings are in line with those of Masci *et al.* [11] who stated that anterior MI patients show significantly greater postinfarction LV remodeling and dysfunction than nonanterior MI patients due to a larger degree of permanent ischemia LV damage without any independent MI site contribution. Also, Zaliaduonyte-Peksiene *et al.* [10] they assessed the effects of clinical, echocardiographic, and angiotensinogen gene polymorphism on left ventricular remodeling following AMI in a group of 141 patients with first STEMI.

According to angiographic data, LAD was frequently the infarct related vessel was substantially more common in those who had remodeling (77.8 vs. 40.4%;  $P < 0.001$ ); this is similar to Pop *et al.* [6] who reported greater rate of LAD stenosis in remodeling group compared to the no remodeling group (48 vs. 26%,  $P = 0.002$ ). These findings are in line with those of Warren *et al.* [12] who discovered that patients with LAD blockage exhibited greater chronic dilatation than those with RCA occlusion ( $P = 0.01$ ). Also similar with those of Loboz-Grudzień *et al.* [5] who examined predictors of adverse LV remodeling after primary angioplasty and univariate regression study showed that LAD as IRA was a significant predictor of LV remodeling ( $P < 0.05$ ).

However, our results are not in line with those of Bolognese *et al.* [7] on affected vessels number. They investigated LV remodeling in patients with AMI treated by primary angioplasty and discovered that high peak creatine kinase value and the presence of multi-vessel coronary artery disease were independent predictors of late LV dilatation. Additionally not in line with Pop *et al.* [6]. They noticed postinfarct LV remodeling predictors in STEMI patient who underwent initial PCI and discovered that LV remodeling was strongly predicted by multivessel coronary artery disease.

According to echocardiographic data, LVEDVI were greater in patients who experienced LV remodeling (114.67 ± 37.39 vs. 66.79 ± 48.13 cm/m<sup>2</sup>;  $P < 0.001$ ). Also shown to be at significant risk for LV remodeling are those with low LVEF and high WMSI more than 1.5. WMSI more than 1.5 is an independent predictor of LV remodeling, according to multivariate regression analysis.

Our results were in line with those of Bolognese *et al.* [7] who investigated LV remodeling following primary angioplasty in AMI patients and discovered that elevated WMSI consistently indicate early LV dilatation. Further, in line with studies from Loboz-Grudzień *et al.* [5], and also, similar to Mousa *et al.* [2] who reported patients with low LVEF less than or equal to 45% and high WMSI more than 1.5,

this univariate analysis identified them as being at high risk for LV remodeling.

Predictors of LV remodeling: patients with extensive anterior MI site, LAD as IRA, LVESVI, wall motion index and LVEF less than or equal to 45% were at great risk for LV remodeling after AMI. Multivariate regression analysis, however, only identified extensive anterior MI site and WMSI more than 1.5 as independent predictors of LV remodeling following primary PCI.

This consistent with Zaliaduonyte-Peksiene *et al.* [10]. They discovered that independent determinants of LV remodeling following AMI were anterior infarct localization, whereas Masci *et al.* [11] who said that anterior MI patients had more severe postinfarction LV remodeling than nonanterior MI patients because they have more permanent ischemic LV damage without any other independent factors. Our results were in line with those of Bolognese *et al.* [7] who investigated LV remodeling following primary angioplasty with AMI and discovered that high WMSI independently predictors of early LV dilatation. In line with those of Lobo-Grudziński *et al.* [5] who discovered that high WMSI more than 1.5 is an independent indicator of LV remodeling after primary angioplasty.

## Conclusion

Bedside transthoracic echocardiography is a simple, cheap, accessible and excellent tool for primary assessment of STEMI patients undergoing PPCI at high risk for long-term LV remodeling.

In these patients, extensive anterior MI, the LAD as an IRA, LVESVI, and segmental wall motion index were the long-term predictors of left ventricular remodeling. But according to multivariate regression study, the only independent indicators of LV remodeling after PPCI were WMSI more than 1.5 and extensive anterior MI site.

## Study limitations

- (1) Small sample size.
- (2) The size of the infarct or the ventricular volumes cannot be determined with great accuracy by two-dimensional echocardiography; CMR imaging is a preferable method.
- (3) Unaware of the late IRA's existence. We could not follow up with coronary angiography, thus we are unable to rule out the potential that repeated ischemia contributed to the remodeling process.
- (4) After primary PCI, we did not assess myocardial perfusion, which may be crucial in the emergence of LV remodeling.

List of abbreviations: AMI, Acute myocardial infarction; CABG, Coronary artery bypass surgery; CAD, Coronary artery disease; CMR, Cardiac magnetic resonance; ECG, Electrocardiography; EF, Ejection fraction; IRA, Infarct related artery; LAD, Left anterior descending artery; LV, Left ventricle; LVEDV, Left ventricle end diastolic volume; LVEDVI, Left ventricular end diastolic volume index; LVEF, Left ventricular ejection fraction; LVESV, Left ventricular end systolic volume; PPCI, Primary percutaneous coronary intervention; STEMI, ST-Elevation Myocardial Infarction; WMSI, Wall motion score index.

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## Conflicts of interest

There are no conflicts of interest.

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