Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2021.73274.2206 Volume 30, Issue 1.1, -January 2024, Supplement Issue
Manuscript ID ZUMJ-2104-2206 (R4)
DOI 10.21608/ZUMJ.2021.73274.2206

ORIGINAL ARTICLE

# Predictors of Left Ventricular Remodeling in Patients with ST-Elevation Myocardial Infarction.

**Mohammad Abdelhakam (MSc)[2], Mahmoud H Shah (MD) [1], Mohammad M Al-Daydamony (MD) [1], Nader T Kandil (MD) [1]**

*[1] Cardiology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt*
*[2] Cardiology Department, Al-Ahrar Teaching Hospital, Zagazig, Egypt*

**Corresponding author**
Mohammad Abdelhakam (MSc)
Specialist of cardiology, Al-Ahrar Teaching Hospital, Zagazig, Egypt.

**E-mail:**
drmhakam@gmail.com

**Submit Date** 2021-04-26
**Revise Date** 2021-06-08
**Accept Date** 2021-06-17

## ABSTRACT

**Background:** Left ventricular (LV) remodeling is still the leading cause of heart failure (HF) and death in patients surviving ST-segment elevation myocardial infarction (STEMI). Despite improvement in treatments of STEMI yet the outcomes did not change, and remodeling occur in about 30% of patients after STEMI. Predictors for LV remodeling are still under investigated, early prediction of LV remodeling is a necessity.

**Aims:** We aimed to identify factors that help in early prediction of LV remodeling after STEMI using standard history, examination, laboratory results, echocardiographic study, and angiographic data collection.

**Patients and Methods:** We included 107 patients with 1st acute STEMI treated by primary percutaneous coronary intervention (PCI) or by thrombolysis then PCI within 24 hours. Patients were divided into two groups according to remodeling after six months; defined as ≥20% increase in left ventricular end diastolic volume (LVEDV). Patients were subjected to history taking, cardiac examination, electrocardiography, standard investigations, echocardiography, and angiography with PCI. After 6 months another echocardiography was done.

**Results:** There was statistically significant positive correlation between the study groups regarding; time till target treatment, hsTroponin T, sum of ST segment elevation, number of leads involved ($p<0.001$), AST level ($p<0.031$), initial LVEDV ($p=0.003$), initial presence of akinesia, and a negative correlation with myocardial blush grade ($p<0.001$), All were independent predictors of remodeling.

**Conclusions:** Time till target treatment, hsTroponin, AST, sum of ST segment elevation, number of leads involved, MBG score, initial LVEDV and initial presence of akinesia are independent predictor of LV remodeling.

**Keywords:** Post STEMI remodeling; LVEDV; myocardial blush grade; akinesia and remodeling

## INTRODUCTION

Despite great advance in ST-segment elevation myocardial infarction (STEMI) management over the last decades, STEMI still a significant cause of Heart failure (HF), morbidity and death **1**. Left ventricular (LV) remodeling is the corner stone for developing HF and a determinant of prognosis post MI **2**. LV remodelling cause structural and functional changes that affects cardiac function with time. To decrease LV remodeling, risk stratification as early as possible is needed to optimally monitor and treat patients at high-risk **3.**

Earlier restoration of TIMI flow 3 in the infarct related artery by the use of primary percutaneous coronary intervention (PCI) in STEMI reduce infarction size, decrease the incidence of remodeling **4**, decrease heart failure and mortality rates when compared to thrombolysis alone **5**.

Previous researchers studied different predictors of remodeling like poorer myocardial perfusion as assessed by myocardial blush grade (MBG) **6**, LV regional and global systolic dysfunction, severe LV diastolic dysfunction **7**, lower ejection fraction at hospital discharge **8**, and symptom to balloon time

**9**, these predictors were found to be significant predictors of remodeling.

Our aim is to identify factors that help in early prediction of LV remodeling after STEMI, with confirming already identified risk factors and trying to find new undiscovered risk factors.

## PATIENTS AND METHODS

This is a Prospective Cohort study carried out in Cardiology Department of Zagazig University Hospital from October 2017 to April 2020. We included 107 patients with their first acute STEMI diagnosed according to 2018 ESC guidelines **10** by the presence of chest pain lasting > 20 minutes and ST-segment elevation ≥1mm in two contiguous limb leads or >2mm in two contiguous chest leads with elevated level of troponin **11**. Patients were prepared for either primary PCI, or thrombolysis with streptokinase followed by early invasive PCI according to time of presentation and the availability of PCI team **10**.

Patients were excluded from our study if ≥1 of the following is present: Previous coronary artery disease (CAD), previous non-ischemic heart disease, presence of disease with low life expectancy, failure of PCI, non-sinus rhythm, ECG criteria of left ventricular hypertrophy or bundle branch block, inadequate echocardiographic image quality, any valvular disease other than mild, significant lesion in non-culprit artery, MI with Non-Obstructive Coronary Artery, and cardiogenic shock.

Written informed consent was obtained from all participants, the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans. We did the following for them: Complete history taking, Full general and local examination, standard 12 leads electrocardiography (ECG) analysis, upon admission and 90 min after target treatment, with estimation of the sum of ST-segment elevation, number of leads involved, type of STEMI just before target treatment, and sum of ST-segment resolution before discharge **12**.

laboratory standard investigations where samples were taken from patients at time of admission that includes Highly sensitive troponin T with cut point of ≥100ng/L **13**, CKMB with cut point of 25IU/L **14**, RBS with detection of Stress hyperglycemia defined as RBS of ≥180mg/dl. We excluded pre-existing diabetes by history and HbA1c to exclude under treated diabetic patients **15**, HbA1c with a cut point for diabetes ≥6.5% **16,** Hb level, WBCs count, AST with upper level of 35U/L **17**, TG with normal range <160mg/dl for males and <133mg/dl for females **17**, and LDL level.

Two Echocardiographic examination were done; the 1st with admission time, and the 2nd was performed after 6 months from total revascularization and using the same machine: GE Vivid 9 system Ultrasound (Horten, Norway). Performed by two separate operators unaware of each other results. LV volumes and EF were estimated via the modified biplane Simpson technique from the apical 4-views and from the formula: EF=[(EDV-ESV)/EDV]×100 **18**. LV remodeling was defined as: a LVEDV increase from baseline of ≥20% at 6 months **19**.

Patients received target treatment by either one of two methods: the 1st by Thrombolysis within 30 minutes of admission if primary PCI is not available for any cause (within 90 min) Streptokinase (1.5 million units were given by intravenous infusion over 30-60 minutes) was given after exclusion of contraindications according to ESC guidelines of STEMI **10**, after which an early invasive PCI was done within 24 hours of admission. The 2nd method is primary PCI from the start within 90 minutes of admission. PCI was performed by a professional team using an automated edge detection system (GE medical system manufactured by SIMENSE (Kemnath\ Germany), PCI was done according to ESC guidelines of Revascularization of STEMI **10 20**. All data was taken by 2 different operators separately unaware of each other opinion **21 22 23**. Grouping of patients and statistical analysis was done according to the presence of remodeling after 6 months, patients were separated into two groups, **Group A** with remodeling (39 patients, 33 males and 6 females) and **Group B** with no remodeling (68 patients, 58 males and 10 females).

## STATISTICAL ANALYSIS

The data collected were revised, coded, tabulated and but into a PC using Statistical package for Social Science (SPSS version 20.0; for windows package program; Armonk, NY, USA: IBM corp.). Data then was analyzed according to the kind of data gained for each parameter. Descriptive statistics (Mean Standard deviation (±SD) for parametric numerical data. Frequency and percentage, chi square test of categorical data). Analytical statistics (Student t-test was used to assess the statistical significance of the difference between the two study groups). A P value of ≤0.05 was considered significant, and a P value ≤0.01 was considered to be highly significant. Pearson's correlation test was applied to estimate and test the relationships between LV remodeling and every parameter taken. Univariate and multivariate

logistic regression of the factors predicting LV remodeling was performed and included.

To assess the intraobserver variability, we repeated the second echocardiographic results after 1 week for 30 patients. The intraobserver and the interobserver were estimated by dividing the difference between the 2 measures by the mean of the 2 observations.

## RESULTS

We included 107 patients having their 1st STEMI as shown in table 1, there was no significant difference between the 2 groups regarding age, sex, or risk factors for CAD.

In patients with remodeling, time till treatment was significantly longer than in patients without remodeling (16.03 ±9.57, versus 7.43±3.24 hours, p <0.001).

The target treatment between the 2 groups did not have a significant difference the p=0.086.

In patients with remodeling highly sensitive troponin T was higher than patients without remodeling (2838.31 ±1618.081, versus 1117.63 ±726.348, p<0.001). Aspartate aminotransferase (AST) level also was higher in patients with remodeling (47.13±29.734, versus 39.4±26.405 mg/dl, p=0.031) (table 1,3).

In patients with remodelling the sum of ST-segment elevation was significantly higher than in patients without remodelling (24.59±10.99, versus 16.35±8.35mV, p<0.001). Also, in patients with remodelling number of electrocardiographic leads involved was significantly higher than the non-remodelling group (4.49±0.79, versus 3.53±0.938 lead, p<0.001).

MBG was significantly lower in patients with remodelling, P value was <0.001. On the other hand, TIMI flow grade and the culprit artery had no significant difference between the 2 groups (Table 1,2,3).

In the initial echocardiographic examination patients with remodelling had higher LVEDV (130.23±25.957 ml³, versus 112.59±21.486 ml³, p=0.003), and higher presence of Akinesia (26 patients 66.67%, versus 21 patients 30.88%, p<0.001).

The presence of rales on admission, stress hyperglycemia, initial EF, initial LVESV, Follow up LVEDV, LVESV, EF and the degree of change in LVEDV, All those parameters showed a significant difference between the two groups in univariate analysis, but failed to show any difference in multivariate regression analysis (Table 4)

**Table (1):** comparison between the two groups

| | | Group A (Remodelling) (n = 39) | Group B (non-Remodelling) (n = 39) | P |
|---|---|---|---|---|
| **Age** | | 56.95 ± 7.49 | 54.69 ± 6.58 | 0.099 |
| **Sex** | **Male** | 33 (84.62%) | 58 (85.29%) | 0.662 |
| | **Female** | 6 (15.38%) | 10 (14.71) | |
| **HTN** | | 21 (53.85%) | 29 (42.65%) | 0.173 |
| **DM** | | 12 (30.77%) | 21 (30.88%) | 0.774 |
| **Smoking** | | 18 (46.15%) | 37 (54.41%) | 0.416 |
| **Dyslipidemia** | | 10 (25.64%) | 18 (26.47%) | 0.926 |
| **Family Hist. of SCD** | | 5 (12.82%) | 5 (7.35%) | 0.607 |
| **SBP** | | 128.08±18.05 | 124.78±20.083 | 0.218 |
| **DBP** | | 79.36±9.472 | 77.72±10.976 | 0.349 |
| **Pulse** | | 90.56±19.056 | 86.47±16.702 | 0.236 |
| **Temperature** | | 37.382±0.4285 | 37.404±0.4644 | 0.856 |
| **Time between onset and target treatment** | | 16.03±9.75 | 7.43±3.24 | < **0.001** |
| **BMI ≥ 30 (Obesity)** | | 14 (35.9%) | 28 (41.18%) | 0.427 |
| **Rales** | | 7 (17.95%) | 3 (4.41%) | **0.02** |
| **Gallop (S3 or S4)** | | 5 (12.82%) | 4 (5.88%) | 0.412 |
| **Target Treatment** | | | | 0.086 |
| • **PCI** | | 21 (53.85%) | 25 (36.76%) | |
| • **Streptokinase** | | 18 (46.15%) | 43 (63.24%) | |
| **hs Troponin T (x1000 ng/dl)** | | 2838.31±1618.081 | 1117.63±726.348 | < **0.001** |

| | | Group A (Remodelling) (n = 39) | Group B (non-Remodelling) (n = 39) | P |
|---|---|---|---|---|
| **CKMB level (IU/L)** | | 177.21±95.606 | 143.44±56.622 | 0.121 |
| **RBS (mg/dl)** | | 223.1±116.369 | 180.71±91.223 | 0.096 |
| **HbA1C (DM ≥ 6.5%)** | | 6.313±1.2958 | 6.266±1.4158 | 0.921 |
| **Stress Hyperglycemia** | | 12 (30.77%) | 10 (14.71%) | **0.013** |
| **Hemoglobin level (mg/dl)** | | 13.79±1.48 | 13.688±1.5297 | 0.926 |
| **WBCs (No. x 1000)** | | 9.149±4.6893 | 7.7±3.1804 | 0.057 |
| **S. Creatinine (mg/dl)** | | 0.933±0.2932 | 0.988±0.303 | 0.387 |
| **AST level (U/L)** | | 47.13±29.734 | 39.4±26.405 | **0.031** |
| **Triglyceride's level (mg/dl)** | | 133.44±68.337 | 120.62±50.586 | 0.191 |
| **LDL level (mg/dl)** | | 133.21±56.962 | 125.59±43.307 | 0.877 |
| **Sum of ST elevation (mV)** | | 24.59±10.99 | 16.35±8.35 | **< 0.001** |
| **Sum of ST resolution (mV)** | | 11.05±5.52 | 11.63±6.108 | 0.817 |
| **Number of leads involved** | | 4.49±0.790 | 3.53±0.938 | **<0.001** |
| **Type of MI** | | | | 0.138 |
| • **Anterior** | | 35 (89.74%) | 52 (76.47%) | |
| • **Anteroseptal** | | 0 (0%) | 5 (7.35%) | |
| • **Inferior** | | 1 (2.56%) | 7 (10.29%) | |
| • **Infero-postro-lateral** | | 3 (7.69%) | 4 (5.88%) | |
| **TIMI Flow Grade** | **0** | 0 | 0 | 0.707 |
| | **1** | 1 (2.56%) | 2 (2.94%) | |
| | **2** | 3 (7.69%) | 5 (7.35%) | |
| | **3** | 35 (89.74%) | 61 (89.71%) | |
| **Myocardial Blush Grade** | **0** | 6 (15.38%) | 0 | **< 0.001** |
| | **1** | 10 (25.64%) | 2 (2.94%) | |
| | **2** | 14 (35.9%) | 7 (10.29%) | |
| | **3** | 9 (23.08%) | 59 (86.76%) | |
| **Culprit Artery** | **LAD** | 35 (89.74%) | 57 (83.82%) | 0.468 |
| | **LCX** | 3 (7.69%) | 3 (4.41%) | |
| | **RCA** | 1 (2.56%) | 8 (11.76%) | |
| **1st EF %** | | 41.08±11.561 | 51.84±9.767 | **<0.001** |
| **1st LVESV** ml³ | | 77.69±25.919 | 54.72±18.257 | **<0.001** |
| **1st LVEDV** ml³ | | 130.23±25.957 | 112.59±21.486 | **0.003** |
| **Akinesia** | | 26 (66.67%) | 21 (30.88%) | **<0.001** |
| **2nd EF %** | | 51.54±8.136 | 59.34±4.01 | **<0.001** |
| **2nd LVESV** ml³ | | 80.08±23.301 | 47.46±10.059 | **<0.001** |
| **2nd LVEDV** ml³ | | 163.46±28.073 | 117±18.229 | **<0.001** |
| **LVEDV change in 6 months** | **n** | 33.23±6.567 ml³ | 4.41±8.331 | **<0.001** |
| | **%** | 26.464±7.937% | 4.724±7.296 | **<0.001** |

Abbreviations: HTN = Hypertension, DM = Diabetes Millitus, SCD = Sudden Cardiac Death, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, BMI = Body Mass Index, S3 = Third Heart Sound, S4 = Fourth Heart Sound, PCI = Percutaneous Coronary Intervention, hsTroponin = Highly Sensitive Troponin, CK-MB = Creatinin Kinase MB, RBS = Random Blood Sugar, HbA1c = Glycosylated Hemoglobin, WBCs = White Blood Cells, AST = Aspartate Aminotransferase, LDL = Low Density Lipoproteins, MI = Myocardial Infarction, TIMI = Thrombolysis In Myocardial Infarction, LAD = Left Anterior Descending Artery, LCX = Left circumflex Artery, RCA = Right Coronary Artery, EF = Ejection Fraction, LVEDV = Left Ventricular Diastolic Volume, LVESV = Left Ventricular Systolic Volume.

**Table (2):** Univariate regression analysis for predictors of remodelling

| Predictor | Estimate | SE | Z | p |
|---|---|---|---|---|
| **Troponin** | 0.00138 | 2.96E-04 | 4.67 | < .001 |
| **Number of Leads** | 1.27 | 0.29 | 4.38 | < .001 |
| **Sum of ST elevation** | 0.0927 | 0.0241 | 3.85 | < .001 |
| **Time till treatment** | 0.179 | 0.0401 | 4.46 | < .001 |
| **1st EF** | -0.0924 | 0.0225 | -4.11 | < .001 |
| **1st LVEDV** | 0.0276 | 0.00905 | 3.05 | 0.002 |
| **1st LVESV** | 0.0441 | 0.0109 | 4.03 | < .001 |
| **2nd LVEDV** | 0.0831 | 0.0162 | 5.14 | < .001 |
| **LVEDV change** | 7.87 | 3209 | 0.00245 | 0.998 |
| **LVEDV change _ A** | 9.79 | 3757 | 0.0026 | 0.998 |
| **2nd LVESV** | 0.128 | 0.0253 | 5.06 | < .001 |
| **2nd EF** | -0.217 | 0.0489 | -4.44 | < .001 |
| **AST** | 0.0113 | 0.00735 | 1.54 | 0.124 |
| **MBG** | -2.17 | 0.424 | -5.13 | < .001 |
| **MBG:** | | | | |
| **0 – 3** | 19.56 | 1495.296 | 0.0131 | 0.99 |
| **1 – 3** | 3.61 | 0.861 | 4.1882 | < .001 |
| **2 – 3** | 2.69 | 0.597 | 4.509 | < .001 |
| **Akinesia:** | | | | |
| **Present – Absent (Reference)** | 1.5 | 0.429 | 3.49 | < .001 |
| **Stress Hyperglycemia:** | | | | |
| **Yes – No (Reference)** | 1.187 | 0.493 | 2.41 | 0.016 |
| **Rales:** | | | | |
| **Yes – No (Reference)** | 1.556 | 0.723 | 2.15 | 0.031 |
| **Estimates represent the log odds of "Remodeling = Yes" vs. "Remodeling = No."** | | | | |

**Abbreviations:** AST = Aspartate Aminotransferase, MBG = Myocardial Blush Grade, EF = Ejection Fraction, LVEDV = Left Ventricular Diastolic Volume, LVESV = Left Ventricular Systolic Volume

**Table (3):** Spearman Correlation with the LVEDV % change after 6 months.

| Spearman Correlations | | | |
|---|---|---|---|
| | | **LVEDV % change** | |
| **Selvester score** | Spearman's rho | 0.841 | *** |
| | p-value | < .001 | |
| **Target Treatment** | Spearman's rho | -0.164 | |
| | p-value | 0.091 | |
| **Troponin** | Spearman's rho | 0.473 | *** |
| | p-value | < .001 | |
| **Sum of ST elevation** | Spearman's rho | 0.273 | ** |
| | p-value | 0.004 | |
| **Age** | Spearman's rho | 0.165 | |
| | p-value | 0.09 | |
| **MBG** | Spearman's rho | -0.516 | *** |
| | p-value | < .001 | |
| **Akinesia** | Spearman's rho | 0.258 | ** |
| | p-value | 0.007 | |
| **1st LVEDV** | Spearman's rho | -0.025 | |
| | p-value | 0.797 | |
| **RBS** | Spearman's rho | 0.088 | |
| | p-value | 0.368 | |
| **HbA1C** | Spearman's rho | 0.094 | |
| | p-value | 0.337 | |
| **DM** | Spearman's rho | 0.011 | |

| Spearman Correlations | | | |
|---|---|---|---|
| | p-value | 0.907 | |
| **AST** | Spearman's rho | 0.123 | |
| | p-value | 0.206 | |
| **CKMB** | Spearman's rho | 0.061 | |
| | p-value | 0.534 | |
| *** p < .05, ** p < .01, *** p < .001** | | | |

Abbreviations: DM = Diabetes Millitus, CK-MB = Creatinin Kinase MB, RBS = Random Blood Sugar, HbA1c = Glycosylated Hemoglobin, AST = Aspartate Aminotransferase, LVEDV = Left Ventricular Diastolic Volume.

**Table (4):** Multivariate regression analysis for predictors of remodelling

| Predictor | Estimate | SE | Z | p |
|---|---|---|---|---|
| **Troponin** | 0.0022 | 5.76E-04 | 3.8142 | < .001 |
| **Sum of ST elevation** | -0.1227 | 0.0591 | -2.0775 | 0.038 |
| **Time till Treatment** | 0.2497 | 0.0656 | 3.804 | < .001 |
| **Age** | 0.08942 | 0.0531 | 1.6837 | 0.092 |
| **SBP** | 0.0016 | 0.0186 | 0.0859 | 0.932 |
| **Sex:** | | | | |
| **Female – Male (Reference)** | 0.76492 | 1.0428 | 0.7335 | 0.463 |
| **Rales:** | | | | |
| **Yes – No (Reference)** | -0.98473 | 1.3488 | -0.7301 | 0.465 |
| **1st LVEDV** | 0.04171 | 0.0182 | 2.2866 | 0.022 |
| **Note. Estimates represent the log odds of "Remodeling = Yes" vs. "Remodeling = No"** | | | | |
| **Predictor** | **Estimate** | **SE** | **Z** | **p** |
| **Troponin** | 0.00366 | 8.60E-04 | 4.2522 | < .001 |
| **Sum of ST elevation** | -0.06193 | 0.0585 | -1.0585 | 0.29 |
| **Time till treatment** | 0.2654 | 0.0717 | 3.70122 | < .001 |
| **Age** | 0.09069 | 0.0569 | 1.59307 | 0.111 |
| **SBP** | 1.83E-04 | 0.0219 | 0.00837 | 0.993 |
| **Sex:** | | | | |
| **Female – Male (Reference)** | -0.70498 | 1.1084 | -0.636 | 0.525 |
| **Stress Hyperglycemia:** | | | | |
| **Yes – No (Reference)** | -0.89216 | 1.231 | -0.7247 | 0.469 |
| **AST** | -0.08377 | 0.025 | -3.3537 | < .001 |
| **Note. Estimates represent the log odds of "Remodeling = Yes" vs. "Remodeling = No"** | | | | |

| Predictor | Estimate | SE | Z | p | |
|---|---|---|---|---|---|
| **Troponin** | 0.00246 | 6.75E-04 | 3.648 | < .001 | |
| **Sum of ST elevation** | -0.18107 | 0.0708 | -2.557 | 0.011 | |
| **Time till treatment** | 0.31618 | 0.0789 | 4.008 | < .001 | |
| **Age** | 0.09828 | 0.0601 | 1.635 | 0.102 | |
| **SBP** | 0.01268 | 0.0204 | 0.62 | 0.535 | |
| **Sex:** | | | | | |
| **Predictor** | **Estimate** | **SE** | **Z** | **p** | |

| Predictor | Estimate | | SE | Z | p |
|---|---|---|---|---|---|
| **Female – Male (Reference)** | 1.29628 | 1.1595 | 1.118 | 0.264 | |
| **Akinesia:** | | | | | |
| **Present – Absent (Reference)** | 2.28791 | 0.7992 | 2.863 | 0.004 | |
| **1st LVEDV** | 0.0484 | 0.0201 | 2.413 | 0.016 | |
| **Note. Estimates represent the log odds of "Remodeling = Yes" vs. "Remodeling = No"** | | | | | |

| Predictor | Estimate | SE | Z | p | |
|---|---|---|---|---|---|
| **Troponin** | 0.00221 | 6.07E-04 | 3.6476 | < .001 | |
| **Sum of ST elevation** | -0.10072 | 0.0547 | -1.8426 | 0.065 | |
| **Time till treatment** | 0.2926 | 0.0727 | 4.0241 | < .001 | |
| **Age** | 0.0546 | 0.0504 | 1.0839 | 0.278 | |
| **SBP** | 0.02128 | 0.0186 | 1.1425 | 0.253 | |
| **Sex:** | | | | | |
| **Female – Male (Reference)** | -0.03911 | 1.0035 | -0.039 | 0.969 | |
| **Akinesia:** | | | | | |
| **Present – Absent (Reference)** | 1.98291 | 0.7594 | 2.6111 | 0.009 | |
| **1st EF%** | 0.00133 | 0.0422 | 0.0314 | 0.975 | |
| **Note. Estimates represent the log odds of "Remodeling = Yes" vs. "Remodeling = No"** | | | | | |

| Predictor | Estimate | SE | Z | p | |
|---|---|---|---|---|---|
| **Troponin** | 0.00209 | 5.87E-04 | 3.565 | < .001 | |
| **Sum of ST elevation** | -0.14838 | 0.0667 | -2.225 | 0.026 | |
| **Time till treatment** | 0.28416 | 0.0713 | 3.984 | < .001 | |
| **Age** | 0.07639 | 0.0548 | 1.395 | 0.163 | |
| **SBP** | 0.01453 | 0.0199 | 0.732 | 0.464 | |
| **Sex:** | | | | | |
| **Female – Male (Reference)** | 0.53761 | 1.0586 | 0.508 | 0.612 | |
| **Akinesia:** | | | | | |
| **Present – Absent (Reference)** | 1.88251 | 0.7525 | 2.502 | 0.012 | |
| **1st LVESV** | 0.03592 | 0.0227 | 1.581 | 0.114 | |
| **Note. Estimates represent the log odds of "Remodeling = Yes" vs. "Remodeling = No"** | | | | | |

| Predictor | Estimate | SE | Z | p |
|---|---|---|---|---|
| **Troponin** | 0.00251 | 9.45E-04 | 2.65878 | 0.008 |
| **Sum of ST elevation** | -0.01804 | 0.0837 | -0.2154 | 0.829 |
| **Time till target treatment** | 0.51715 | 0.1543 | 3.35189 | < .001 |
| **Age** | -0.02755 | 0.1037 | -0.2657 | 0.79 |
| **SBP** | 0.03626 | 0.0331 | 1.09569 | 0.273 |
| **Sex:** | | | | |
| **Female – Male (Reference)** | 0.38383 | 1.4822 | 0.25897 | 0.796 |
| **MBG:** | | | | |

| | | | | |
|---|---|---|---|---|
| **0 – 3** | 26.55412 | 2729.23 | 0.00973 | 0.992 |
| **1 – 3** | 6.86385 | 2.2169 | 3.09617 | 0.002 |
| **2 – 3** | 5.31052 | 2.3061 | 2.30285 | 0.021 |

**Note. Estimates represent the log odds of "Remodeling = Yes" vs. "Remodeling = No"**
Abbreviations**:** SBP = Systolic Blood Pressure, AST = Aspartate Aminotransferase, MBG = Myocardial Blush Grade, EF = Ejection Fraction, LVEDV = Left Ventricular Diastolic Volume, LVESV = Left Ventricular Systolic Volume.

## DISCUSSION

In our study logistic regression analysis showed that the only independent significant predictor for post STEMI LV remodelling were time till target treatment, hsTroponin T, AST, sum of ST-segment elevation, number of leads involved in STEMI, initial LVEDV, initial presence of akinesia, and MBG (Table 2,4).

A percentage of 34.45% of patients in our study developed remodelling. That is slightly higher than the average percentage which is about 30% of patients post MI found by Flachskampf et al and many other scholars who studied remodelling post MI **24**. Although we excluded unsuccessful PCI from our study which usually increase the percentage of remodelling **25**, we may attribute this increase to the delayed presentation of patients in our study. We adopted the definition of remodelling to be the increase in the LVEDV of 20% or more, as Bolognese and so many scholars did **5 19 26**.

Soon and colleagues studied time factor on outcomes post MI and found that symptoms to onset of treatment is one of the most important predictors for remodelling, as we also found **27**.

Berezin and colleagues studied the relation between Troponin level and remodelling, they found that elevated troponins is a useful independent predictive biomarker of post-AMI remodelling and HF, we found a highly significant positive correlation between serum hs-Troponin level and remodelling after 6 months **28 29**.

We found a positive correlation between elevated AST and remodelling. Our data matched that of Lofthus and many authors findings about liver enzymes especially AST and remodelling **30 31**.

Oliver Husser and colleagues studied the relation between SUM of ST-segments elevation and number of leads involved in STEMI with remodelling and found that they are good predictors for remodelling, we also found a highly positive correlation between them **32 33**.

Many scholars like Bolognese et al studied microvascular dysfunction effect on remodelling and outcomes after MI and found it to be a good predictor for remodelling **9 29**. Poli found that MBG score was associated with the degree of early and late recovery of LV. Stone et al found that MBG score predict survival rate after primary or rescue PCI **30**. We found a highly significant negative correlation between MBG score and remodelling.

But we found no significant difference between the two groups as regards to TIMI flow post PCI. This was in concordant with Goel et al and many other recent studies **31**.

Chew et al and many scholars studied the relation between initial LVEDV and remodelling and found it to be a good predictor **29 39**. We also found a highly significant positive correlation between initial LVEDV and remodelling after 6 months of STEMI.

Cokkinos and colleagues and Berezin and colleagues studied the impact of the presence of akinesia in any LV segment and remodelling and found it a strong predictor **29 40**, we also found a highly significant positive correlation between the presence of akinesia in the initial echocardiography and remodelling after 6 months of STEMI.

## CONCLUSION

Our study showed that after successfully performed primary or early invasive PCI for patients with their 1st STEMI, time from onset of MI till receiving target treatment, MBG score, initial LVEDV, initial presence of akinesia, sum of ST-segment elevation, number of leads involved in STEMI, hsTroponin T, and AST level were the only significant predictors of LV remodelling. Efforts must be made to significantly reduce time gap between onset of symptoms and receiving target treatment, including cardiac symptoms awareness among society, and educating primary healthcare providers. Along with early introduction of treatments that improve microvascular dysfunction when present after PCI.

## REFERENCES

**1. Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WB, Bravata DM et al.** Heart disease and stroke statistics—2012 update: a report from the American Heart Association. Circulation, 2012; 125(1), e2-e220.

**2. St John Sutton M, Pfeffer MA, Plappert T, Rouleau JL, Moyé LA, Dagenais GR et al.** Quantitative two-dimensional echocardiographic measurements are major predictors of adverse cardiovascular events after acute myocardial infarction. The protective effects of captopril. Circulation. 1994 Jan;89(1):68-75.

**3. Hendriks T, Hartman MHT, Vlaar PJJ, Prakken NHJ, van der Ende YMY, Lexis CPH et**

**al.** Predictors of left ventricular remodeling after ST-elevation myocardial infarction. Int J Cardiovasc Imaging. 2017 Sep;33(9):1415-1423.

**4. Keeley EC, Boura JA, Grines CL.** Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet. 2003 Jan 4;361(9351):13-20.

**5. Bolognese L, Neskovic AN, Parodi G, Cerisano G, Buonamici P, Santoro GM et al.** Left ventricular remodeling after primary coronary angioplasty: patterns of left ventricular dilation and long-term prognostic implications. Circulation. 2002 Oct 29;106(18):2351-7.

**6. Ahn KT, Song YB, Choe YH, Yang JH, Hahn JY, Choi JH et al.** Impact of transmural necrosis on left ventricular remodeling and clinical outcomes in patients undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. Int J Cardiovasc Imaging. 2013 Apr;29(4):835-42.

**7. Loboz-Grudzień K, Kowalska A, Brzezińska B, Sokalski L, Jaroch J.** Early predictors of adverse left ventricular remodelling after myocardial infarction treated by primary angioplasty. Cardiol J. 2007;14(3):238-45. PMID: 18651467.

**8. Wita K, Filipecki A, Lelek M, Bochenek T, Elżbieciak M, Wróbel W et al.** Prediction of left ventricular remodeling in patients with STEMI treated with primary PCI: use of quantitative myocardial contrast echocardiography. Coron Artery Dis. 2011 May;22(3):171-8.

**9. Farag EM and Al-Daydamony MM.** Symptom-to-balloon time and myocardial blush grade are predictors of left ventricular remodelling after successful primary percutaneous coronary intervention. Cardiovasc J Afr. 2017 May/Jun 23;28(3):186-190.

**10. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H et al.** ESC Scientific Document Group. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2018 Jan 7;39(2):119-177.

**11. Coppola G, Carità P, Corrado E, Borrelli A, Rotolo A, & Guglielmo M.** Italian Study Group of Cardiovascular Emergencies of the Italian Society of Cardiology. ST segment elevations: always a marker of acute myocardial infarction. Indian Heart J, 2013; 65(4), 412-23.

**12. Brener SJ, Dizon JM, Mehran R, Guerchicoff A, Lansky AJ, Farkouh M et al.** Complementary prognostic utility of myocardial blush grade and ST-segment resolution after primary percutaneous coronary intervention: analysis from the HORIZONS-AMI trial. Am Heart J. 2013 Oct;166(4):676-83.

**13. Pham MX, Whooley MA, Evans GT Jr, Liu C, Emadi H, Tong W et al.** Prognostic value of low-level cardiac troponin-I elevations in patients without definite acute coronary syndromes. Am Heart J. 2004 Nov;148(5):776-82.

**14. Murakami M, Kodama M, Yoshida R, Asada T, Sano T, Sano M et al.** Establishment of a cut-off value for the diagnosis of acute myocardial infarction using a new CK-MB activity measurement reagent containing anti-MtCK antibody. Rinsho Byori. 2011 Jul;59(7):649-55. Japanese. PMID: 21874790.

**15. Marik PE and Bellomo R.** Stress hyperglycemia: an essential survival response! Crit Care. 2013 Mar 6;17(2):305.

**16. American Diabetes Association.** Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2020. Diabetes Care. 2020 Jan;43(Suppl 1):S14-S31.

**17. Goldfarb NM.** Mosby's Diagnostic & Laboratory Test Reference. 2009.

**18. Albin G and Rahko PS.** Comparison of echocardiographic quantitation of left ventricular ejection fraction to radionuclide angiography in patients with regional wall motion abnormalities. Am J Cardiol. 1990 Apr 15;65(15):1031-2.

**19. Bière L, Donal E, Jacquier A, Croisille P, Genée O, Christiaens L et al.** A new look at left ventricular remodeling definition by cardiac imaging. Int J Cardiol. 2016 Apr 15;209:17-9.

**20. Do D, West JA, Morise A, Atwood JE, Froelicher V.** An agreement approach to predict severe angiographic coronary artery disease with clinical and exercise test data. Am Heart J. 1997 Oct;134(4):672-9.

**21. TIMI Study Group.** Comparison of invasive and conservative strategies after treatment with intravenous tissue plasminogen activator in acute myocardial infarction. Results of the thrombolysis in myocardial infarction (TIMI) phase II trial. N Engl J Med. 1989 Mar 9;320(10):618-27.

**22. Chesebro JH, Knatterud G, Roberts R, Borer J, Cohen LS, Dalen J et al.** Thrombolysis in Myocardial Infarction (TIMI) Trial, Phase I: A comparison between intravenous tissue plasminogen activator and intravenous streptokinase. Clinical findings through hospital discharge. Circulation. 1987 Jul;76(1):142-54.

**23. van 't Hof AW, Liem A, Suryapranata H, Hoorntje JC, de Boer MJ, Zijlstra F.** Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: myocardial blush grade. Zwolle Myocardial Infarction Study Group. Circulation. 1998 Jun 16;97(23):2302-6.

**24. Flachskampf FA, Schmid M, Rost C, Achenbach S, DeMaria AN, Daniel WG.** Cardiac imaging after myocardial infarction. Eur Heart J. 2011 Feb;32(3):272-83.
**25. Galli A and Lombardi F.** Postinfarct Left Ventricular Remodelling: A Prevailing Cause of Heart Failure. Cardiol Res Pract. 2016;2016:2579832. doi: 10.1155/2016/2579832. Epub 2016 Feb 18. PMID: 26989555; PMCID: PMC4775793.
**26. Bochenek T, Wita K, Tabor Z, Grabka M, Krzych Ł, Wróbel W et al.** Value of speckle-tracking echocardiography for prediction of left ventricular remodeling in patients with ST-elevation myocardial infarction treated by primary percutaneous intervention. J Am Soc Echocardiogr. 2011 Dec;24(12):1342-8.
**27. Soon CY, Chan WX and Tan HC.** The impact of time-to-balloon on outcomes in patients undergoing modern primary angioplasty for acute myocardial infarction. Singapore Med J. 2007 Feb;48(2):131-6. PMID: 17304392.
**28. Berezin AE.** Circulating biomarkers in heart failure: diagnostic and prognostic importance." Journal of Laboratory and Precision Medicine 3.4 (2018).
**29. Berezin AE and Berezin AA.** Adverse Cardiac Remodelling after Acute Myocardial Infarction: Old and New Biomarkers. Dis Markers. 2020 Jun 12;2020:1215802. doi:10.1155/2020/1215802. PMID:32626540; PMCID: PMC7306098.
**30. Reinstadler SJ, Feistritzer HJ, Reindl M, Klug G, Mayr A, Mair J at al.** Combined biomarker testing for the prediction of left ventricular remodelling in ST-elevation myocardial infarction. Open Heart. 2016 Sep 20;3(2):e000485.
**31. Lofthus DM, Stevens SR, Armstrong PW, Granger CB, Mahaffey KW.** Pattern of liver enzyme elevations in acute ST-elevation myocardial infarction. Coron Artery Dis. 2012 Jan;23(1):22-30.
**32. Husser O, Bodí V, Sanchis J, Núnez J, Mainar L, Rumiz E et al.** The sum of ST-segment elevation is the best predictor of microvascular obstruction in patients treated successfully by primary percutaneous coronary intervention. Cardiovascular magnetic resonance study. Rev Esp Cardiol. 2010 Oct;63(10):1145-54. English, Spanish.
**33. Lund GK, Stork A, Muellerleile K, Barmeyer AA, Bansmann MP, Knefel M et al.** Prediction of left ventricular remodeling and analysis of infarct resorption in patients with reperfused myocardial infarcts by using contrast-enhanced MR imaging. Radiology. 2007 Oct;245(1):95-102.
**34. Bolognese L, Carrabba N, Parodi G, Santoro GM, Buonamici P, Cerisano G et al.** Impact of microvascular dysfunction on left ventricular remodeling and long-term clinical outcome after primary coronary angioplasty for acute myocardial infarction. Circulation. 2004 Mar 9;109(9):1121-6.
**35. AboulEnin M and Nasif A.** Impact of myocardial blush on left ventricular remodeling in patients treated successfully with primary or rescue coronary intervention. Zagazig University Medical Journal, 2016; 22(1), 1-10.
**36. Poli A, Fetiveau R, Vandoni P, del Rosso G, D'Urbano M, Seveso G et al.** Integrated analysis of myocardial blush and ST-segment elevation recovery after successful primary angioplasty: Real-time grading of microvascular reperfusion and prediction of early and late recovery of left ventricular function. Circulation. 2002 Jul 16;106(3):313-8.
**37. Aboelkasem Ali Mousa M, Abdelsabour Abdallah M, Shamseddin Mohammad H, Ahmad Aly Youssef A.** Early predictors of left ventricular remodeling after primary percutaneous coronary intervention. Egypt Heart J. 2018 Dec;70(4):403-407. Epub 2018 Aug 24. PMID: 30591763; PMCID: PMC6303352.
**38. . Goel PK, Bhatia T, Kapoor A, Gambhir S, Pradhan PK, Barai S et al.** Left ventricular remodeling after late revascularization correlates with baseline viability. Tex Heart Inst J. 2014 Aug 1;41(4):381-8. PMID: 25120390; PMCID: PMC4120500.
**39. Chew DS, Wilton SB, Kavanagh K, Southern DA, Tan-Mesiatowsky LE, Exner DV; APPROACH Investigators.** Left ventricular ejection fraction reassessment post-myocardial infarction: Current clinical practice and determinants of adverse remodeling. Am Heart J. 2018 Apr;198:91-96. Epub 2017 Dec 5. PMID: 29653653.
**40. Cokkinos DV and Belogianneas C.** Left Ventricular Remodelling: A Problem in Search of Solutions. Eur Cardiol. 2016 Aug;11(1):29-35. doi: 10.15420/ecr.2015:9:3. PMID: 30310445; PMCID: PMC6159423.

## How to cite

**Abdelhakam, M., Shah, M., Al-Daydammony, M., Kandil, N. Predictors Of Left Ventricular Remodelling In Patients With ST-Elevation Myocardial Infarction. *Zagazig University Medical Journal*, 2024; (234-243): -. doi: 10.21608/zumj.2021.73274.2206**