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Original article

# Assessment of Baseline and Post-PCI Electrocardiographic Parameters as Predictors of Left Ventricular Systolic Dysfunction after Primary PCI in STEMI

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## Abstract

**Background:** ST-segment elevation myocardial infarction (STEMI) is the most acute manifestation of coronary artery disease, with substantial morbidity and mortality. **This study aimed to** assess the relationship between selected baseline and post-PCI ECG variables and the presence of Left ventricular systolic dysfunction6 months after a first STEMI. **Methods:** This prospective study was conducted on 191 patients who underwent percutaneous coronary intervention (PCI) for a first STEMI. After PCI some patients devolved systolic dysfunction and other did not. **Results**: LVEF, prevalence of presence of reciprocal ST-segment depression ≥ 1mm and ST-segment resolution and the prevalence of ST-segment resolution (≥50%) was significantly reduced in left ventricular systolic dysfunction (LVSD) group compared to non-LVSD group (P<0.001). QRS duration and anterior location of STEMI were significant predictors for the presence of LVSD 6 months after discharge. **Conclusion:** The baseline and post-PCI electrocardiographic parameters, including elevated heart rate, anterior location of STEMI, and extensive ST-segment elevation are significant predictors of LVSD six months after a first STEMI. These findings show the critical importance of early, comprehensive electrocardiographic and echocardiographic evaluation in patients experiencing a first STEMI for identifying those at increased risk of developing LVSD, facilitating early intervention and potentially improving long-term cardiac outcomes.

**Keywords:** PCI; Electrocardiographic; Left Ventricular Systolic Dysfunction; ST-Segment Elevation Myocardial Infarction.

## Introduction

ST-segment elevation myocardial infarction (STEMI) is the most acute manifestation of coronary artery disease, with substantial morbidity and mortality (1). The primary goal of therapeutic strategy for acute STEMI is restoration of myocardial blood flow as soon as possible. Based on the latest guidelines, primary percutaneous coronary intervention (PCI) was recommended as the preferred reperfusion strategy markedly reducing morbidity and mortality (2).

PCI is a non-surgical, invasive procedure with the goal of relieving the narrowing or occlusion of the coronary artery and improve blood supply to the ischemic tissue. This is usually achieved by different methods, the most common being ballooning the narrow segment or deploying a stent to keep the artery open (3).

Electrocardiography (ECG), invented by Willem Einthoven nearly 120 years ago, remains one of the essential diagnostic modalities in cardiology, shaping the elementary division of acute coronary syndromes into those with and without persistent ST-segment depression, affecting the timing and mode of management and adding to short- and long-term risk stratification (4).

It is estimated that left ventricular systolic dysfunction (LVSD), recognized as a long-term consequence of myocardial infarction (MI), may affect up to 60% of post-MI patients. Its occurrence mainly depends on the presence of frozen myocardium, size of post-MI necrosis, and occurrence of left ventricular remodeling (5).

Left ventricular ejection fraction (LVEF), measured with echocardiography, is by far the most popular method for diagnosing LVSD in the clinical setting (6).

LVSD is a well-recognized marker of unfavorable prognosis in post-MI patients, translating into a 3–4-fold increase in mortality and higher rates of cardiovascular adverse outcomes, such as cardiac rupture, sudden cardiac arrest, recurrent myocardial infarction, ventricular arrhythmias, stroke, prolonged hospitalization and rehospitalization (7).

The mortality rate among post-MI patients with asymptomatic LVSD after 12 months of MI is as high as 12% and amounts to 36% in symptomatic patients. LVSD independently predicts short-, mid- and long-term mortality after MI (5)**.**

There are many reports regarding the predictive value of ECG with respect to the development of LVSD after STEMI. A vast part of these reports however, comes from the era of thrombolytic treatment of STEMI and was derived from non-uniform cohorts of patients regarding forms of MI, reperfusion treatment and pharmacotherapy.

Nowadays, in consequence of current standards of STEMI management, incorporating percutaneous coronary intervention (PCI) as a means of effective and safe reperfusion, together with dual antiplatelet treatment, we have witnessed a spectacular reduction in the rates of death, reinfarction, heart failure and strokes (4).

The purpose of this study was to assess the relationship between selected baseline and post-PCI ECG variables and the presence of LVSD 6 months after a first STEMI.

## Patients and methods

This prospective study was conducted at the Cardiology Department of Benha University Hospital and Nasr City Health Insurance Hospital, from 1 November 2022 to 30 June 2023. The study included a total of 191 patients who underwent primary PCI after STEMI.

An approval from the Research Ethics Committee of Benha Faculty of Medicine was obtained (MS 50-1-2023)

An informed written consent from all patients or first-degree relatives before participation was obtained.

**Inclusion criteria were** age >18 years, both sexes and patients with STEMI referred for primary PCI after diagnostic coronary angiography: Typical stenocardial pain with a duration of ≥30 min, time from the onset of symptoms to hospital admission <12 h, presence of electrocardiographic features suggestive of acute STEMI (ST-segment elevation of ≥0.2 mV in at least 2 adjacent leads for leads V1-V3 and/or ≥0.1 mV in at least 2 adjacent leads in the remaining leads, excluding lead aVR) (8).

**Exclusion criteria were a**ny previous myocardial infarction or coronary revascularization, presence of advanced acute or chronic heart failure (defined as class IV according to the Killip classification or class ≥III according to the New York Heart Association), ECG abnormalities that might become study confounders (i.e., left bundle branch block, isolated posterior myocardial infarction, isolated right ventricular myocardial infarction due to several reasons related to the differences in their presentation, diagnosis, and outcomes compared to other types of STEMI, permanent atrial fibrillation), severe valvular heart disease, cardiomyopathy, poorly controlled arterial hypertension (defined as blood pressure ≥180/110 mmHg on hospital admission), significant kidney dysfunction on hospital admission (defined as creatinine concentration exceeding 2 mg/dL).

**Grouping:** The patients were divided into two groups**: Group 1 (LVSD Group):** This group comprised patients who developed LVSD six months post-STEMI. LVSD was defined as left ventricular ejection fraction (LVEF) ≤40% on transthoracic echocardiography. **Group 2 (Non-LVSD Group):** This group included patients who did not develop LVSD six

months after the STEMI, with an LVEF >40%.

**All studied cases were subjected to the following: Detailed history taking, including** [personal history; age, gender, residence, occupation, and special habits, symptoms subjecting heart failure, past history of any medical condition or previous hospital admission, medical history of drugs taken by all of them.]. **Full clinical examination: General examination including** [general comment on patient conscious and mental state., Jaundice or pallor, vital signs: pulse, blood pressure, capillary filling time, respiratory rate and temperature, lower limb edema. body mass index (BMI), and waist circumferences.], **Systemic examination** [Cardiovascular examination included inspection for cyanosis, clubbing, and edema; palpation of the apex beat and heaves; auscultation of heart sounds and murmurs; and pulse assessment. Respiratory examination involved observation for distress and deformities, palpation of chest expansion, percussion for dullness, and auscultation of breath sounds]. **Routine laboratory investigations [**Random blood sugar, complete blood count, kidney, liver function testes and lipid profile]. 12 leads Electrocardiogram. Echocardiography.

**12 leads Electrocardiogram**

ECG recordings were performed at paper speed of 25 mm/s and 0.1 mV/mm calibration using 12 standard leads (I, II, III, aVR, aVL, aVF, V1–V6). Each ECG record was independently interpreted by two experienced cardiologists. In case of disagreement, a third cardiologist was consulted to determine the final interpretation. The following ECG parameters were evaluated: Heart rate, Location of MI, Number of leads with ST-segment elevation, Sum of ST-segment elevation in all leads, Maximum ST-segment elevation in a single lead, Presence of reciprocal ST-segment depression ≥0.1 mV on admission to hospital, Number of leads with pathological Q-waves (according to the 2007 universal definition of MI) (9). The degree of ischemia according to the Birnbaum–Sklarovsky classification (10). QRS complex width.

**Echocardiography**

Two-dimensional transthoracic echocardiography was performed in order to evaluate left ventricular systolic function using a Philips Sonos 7500 device (Philips, Andover, MA, USA) at two time points: before hospital discharge and after 6 months. Image acquisitions and measurements were performed according to the recommendations of the Echocardiography and the American Society of Echocardiography (11). This included assessment of left ventricular ejection fraction (LVEF), left ventricular end-diastolic and end-systolic diameters (LVEDd, LVESd), left ventricular end-diastolic and end-systolic volumes (LVEDV, LVESV) and wall motion score index (WMSI).

**Coronary angiography & primary PCI**

It was performed for all patients, PCI strategy: Invasive coronary angiography was performed in accordance with the American College of Cardiology (ACC)/American Heart Association (AHA) Guidelines for Coronary Angiography (12).

**Follow up:**

The primary study endpoint was the occurrence of LVSD 6 months after STEMI. LVSD was defined as LVEF _40% on transthoracic echocardiography. This cut-off value was previously shown to be associated with unfavorable prognosis. Additionally, LVEF _40% is used by the European Society of Cardiology guidelines for defining heart failure with reduced ejection fraction and post-infarct patients who benefit from therapy with a beta-blocker, angiotensin-converting enzyme inhibitor or mineralocorticoid receptor antagonist (4).

**Statistical analysis**

Statistical analysis was done by SPSS v26 (IBM Inc., Armonk, NY, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing unpaired Student's t- test. Qualitative variables were presented as frequency and percentage (%) and were analyzed utilizing the Chi-square test or Fisher's exact test when appropriate. A two tailed P value < 0.05 was considered statistically significant.

## Results

Demographic data (age, sex, weight, height, and time from symptom onset to PCI), Systolic, diastolic blood pressure, risk factors for CAD (BMI, smoking, hypertension, hyperlipidemia) and blood profile (Hb, WBCs, platelets, INR, ALT, and AST) were insignificantly different between the studied groups. The prevalence of Diabetes mellitus and FBG was significantly higher in LVSD group compared to non-LVSD group (P<0.05). **Table 1**

The prevalence of IRA: LAD/other, IRA TIMI 0 flow prior to PCI, IRA TIMI 3 flow post PCI, GP IIb/IIIa inhibitor usage, Glucose on admission, CK-MB, Cholesterol, Triglycerides, HDL, LDL, BNP on admission, BNP at discharge, Echocardiographic characteristics at discharge including LA, LVEDd, LVESd, LVEDV, LVESV, LVEF ≤40 %, and WMSI were significantly higher in LVSD group compared to non-LVSD group (P<0.001). The prevalence of multivessel CAD and stent implantation were insignificantly different between the two studied groups. eGFR and LVEF were significantly reduced in LVSD group compared to non-LVSD group (P<0.001). **Table 2**

Echocardiographic characteristics 6 months after discharge including LA, LVEDd, LVESd, LVEDV, LVESV, and WMSI, Electrocardiographic

characteristics at discharge including Heart rate, anterior location of STEMI, number of leads with ST-segment, sum of ST-segment elevation, maximal ST-segment elevation, number of leads with pathologic Q waves, and QRS duration and Electrocardiographic characteristics 6 months after discharge including Heart rate, number of leads with ST-segment elevation, sum of ST-segment elevation, maximal ST-segment elevation, number of leads with pathologic Q waves, and QRS duration were significantly higher in LVSD group compared to non-LVSD group (P<0.001). LVEF, prevalence of presence of reciprocal ST-segment depression ≥ 1mm and ST-segment resolution and the prevalence of ST-segment resolution (≥50%) was significantly reduced in LVSD group compared to non-LVSD group (P<0.001). **Table 3**

The results of the univariate logistic regression analysis shows that DM, IRA: LAD /other, IRA TIMI 0 flow prior to PCI, IRA TIMI 3 flow post PCI, GP IIb IIIa inhibitor usage, glucose on admission, CKMB, HR, most of echocardiographic data at discharge (LA, LVEDd, LVESd, LVEDV, LVESV, LVEF, LVEF ≤40 % at discharge and WMSI points) anterior location of STEMI, presence of reciprocal ST-segment, sum of ST-segment elevation, maximal ST-segment elevation, number of leads with pathologic Q waves and ST-segment resolution (≥50%) were significant predictors for the presence of LVSD 6 months after discharge. **Table 4**

The results of the multivariate logistic regression analysis shows that only IRA TIMI 3 flow post PCI, QRS duration and anterior location of STEMI were significant predictors for the presence of LVSD 6 months after discharge. **Table 5**

On multiple regression analysis, we found that only higher BMI, IRA TIMI 3 flow post PCI, higher glucose level on admission, QRS duration, anterior location of STEMI, and number of leads with pathologic Q waves were significant predictors for LVEF deterioration at 6 months after discharge. **Table 6**

**Table 1: Demographics, blood pressure, risk factors and blood profile for CAD of the studied groups**

| | Group 1 (LVSD group) (n=34) | Group 2 (non-LVSD group) (n=157) | P value |
|---|---|---|---|
| **Age (years)** | 52.56 ± 6.36 | 52.85 ± 17.68 | 0.882 |
| **Sex Male** | 25 (74%) | 116 (74%) | 0.965 |
| **Female** | 9 (26%) | 41 (26%) | |
| **Weight (Kg)** | 69 ± 7.68 | 67.5 ± 7.72 | 0.317 |
| **Height (m)** | 1.6 ± 0.05 | 1.6 ± 0.06 | 0.239 |
| **Time from symptom onset to PCI** | 255.7 ± 57.48 | 241.2 ± 59.16 | 0.195 |
| **Blood pressure** | | | |

| | | | |
|---|---|---|---|
| **SBP (mmHg)** | 131.5 ± 11.58 | 128.5 ± 13.21 | 0.222 |
| **DBP (mmHg)** | 72.6 ± 9.31 | 75.5 ± 10.28 | 0.141 |
| **Risk factors for CAD** | | | |
| **BMI (Kg/m2)** | 25.8 ± 3.04 | 24.9 ± 3.43 | 0.163 |
| **Smoking** | 23 (68%) | 79 (50%) | 0.066 |
| **Diabetes mellitus** | 16 (47%) | 37 (24%) | 0.01* |
| **Hypertension** | 14 (41%) | 59 (38%) | 0.695 |
| **Hyperlipidemia** | 16 (47%) | 52 (33%) | 0.123 |
| **Blood profile** | | | |
| **Hb (g/dl)** | 12.6 ± 1.42 | 12.7 ± 1.48 | 0.941 |
| **WBCs ($\times 10^9$/L)** | 7.9 ± 2.24 | 8 ± 2.06 | 0.822 |
| **Platelets ($\times 10^9$/L)** | 258.9 ± 41.64 | 264.8 ± 51.13 | 0.532 |
| **INR** | 0.9 ± 0.14 | 0.9 ± 0.15 | 0.749 |
| **ALT (U/L)** | 32.5 ± 8.68 | 33.6 ± 9.92 | 0.567 |
| **AST (U/L)** | 33.7 ± 7.15 | 32.8 ± 7.81 | 0.519 |
| **FBG (mg/dL)** | 117.1 ± 22.54 | 96.1 ± 13.47 | <0.001* |

PCI: Percutaneous coronary intervention, LVSD: Left ventricular systolic dysfunction, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, LVSD: Left ventricular systolic dysfunction, Hb: Hemoglobin, INR: International Normalized Ratio, WBCs: White blood cell count, ALT: Alanine transaminase, AST: Aspartate aminotransferase, FBG: Fasting blood glucose.

**Table 2: Angiographic characteristics, biochemical and Echocardiographic characteristics in the studied groups**

| | Group 1 (LVSD group) (n=34) | Group 2 (Non-LVSD group) (n=157) | P value |
|---|---|---|---|
| **IRA: LAD/other** | 31 (91%) | 37 (24%) | <0.001* |
| **IRA TIMI 0 flow prior to PCI** | 26 (76%) | 51 (32%) | <0.001* |
| **IRA TIMI 3 flow post PCI** | 25 (74%) | 151 (96%) | <0.001* |
| **Multivessel CAD** | 20 (59%) | 101 (64%) | 0.545 |
| **Stent implantation** | 33 (97%) | 147 (94%) | 0.436 |
| **GP IIb/IIIa inhibitor usage** | 18 (53%) | 32 (20%) | <0.001* |
| **Biochemical characteristics** | | | |
| **eGFR (mL/min/1.73m2)** | 80.4 ± 4.78 | 85.2 ± 5.86 | <0.001* |
| **Glucose on admission (mg/dL)** | 161.3 ± 15.46 | 140.6 ± 12.15 | <0.001* |
| **CK-MB (IU/L)** | 549.2 ± 102.84 | 222.9 ± 71.94 | <0.001* |
| **Cholesterol (mg/dL)** | 222.9 ± 18.3 | 138.1 ± 10.17 | <0.001* |
| **Triglycerides (mg/dL)** | 225.1 ± 20.25 | 138.8 ± 10.59 | <0.001* |
| **HDL (mg/dL)** | 51.5 ± 4.86 | 44.8 ± 3.25 | <0.001* |
| **LDL (mg/dL)** | 149.7 ± 12.7 | 132.1 ± 7.57 | <0.001* |
| **BNP on admission (pg/mL)** | 99.1 ± 39.29 | 67.6 ± 22.51 | <0.001* |
| **BNP at discharge (pg/mL)** | 501.7 ± 144.58 | 103.9 ± 21.13 | <0.001* |
| **Echocardiographic** | | | |
| **LA (mm)** | 42.4 ± 2.56 | 38.7 ± 2.04 | <0.001* |
| **LVEDd (mm)** | 52.9 ± 2.44 | 48.8 ± 2.36 | <0.001* |
| **LVESd (mm)** | 37.1 ± 1.77 | 33.4 ± 2.36 | <0.001* |
| **LVEDV (mL)** | 118.8 ± 8.16 | 95.8 ± 8.54 | <0.001* |
| **LVESV (mL)** | 75.1 ± 5.03 | 53.2 ± 5.46 | <0.001* |
| **LVEF ≤40 % at discharge** | 27 (79%) | 24 (15%) | <0.001* |
| **LVEF (%)** | 36.3 ± 2.28 | 48.2 ± 4.26 | <0.001* |
| **WMSI (points)** | 1.9 ± 0.05 | 1.5 ± 0.09 | <0.001* |

IRA: Infarct-related artery, LAD: Left anterior descending artery, PCI: Percutaneous coronary intervention, CAD: Coronary artery disease, TIMI: Thrombolysis in myocardial infarction score, eGFR: Estimated Glomerular Filtration Rate, CK-MB: Creatine Kinase MB, HDL: High Density Lipoprotein Cholesterol, LDL: Low Density Lipoprotein Cholesterol, BNP: B-Type Natriuretic Peptide. LA: left atrium end-systolic diameter, LVEDd: left ventricular end-diastolic diameter, LVEDV: left ventricular end-diastolic volume, LVEF: left ventricular ejection fraction, LVESd: left ventricular end-systolic diameter, LVESV: left ventricular end-systolic volume, LVSD: left ventricular systolic dysfunction, WMSI: wall motion score index

**Table 3:** Echocardiographic characteristics of the studied groups 6 months after discharge, in relation to LVSD occurrence at discharge and in relation to LVSD occurrence 6 months after discharge

| | Group 1 (LVSD group) (n=34) | Group 2 (Non-LVSD group) (n=157) | P value |
|---|---|---|---|
| **LA (mm)** | 42.9 ± 2.04 | 39.9 ± 1.94 | <0.001* |
| **LVEDd (mm)** | 55.1 ± 1.87 | 48.6 ± 2.36 | <0.001* |
| **LVESd (mm)** | 41.3 ± 3.09 | 33.5 ± 1.71 | <0.001* |
| **LVEDV (mL)** | 146.9 ± 9.66 | 107.1 ± 9.68 | <0.001* |
| **LVESV (mL)** | 89.4 ± 7 | 54.8 ± 5.7 | <0.001* |
| **LVEF (%)** | 35.8 ± 1.74 | 49.4 ± 3.51 | <0.001* |
| **WMSI (points)** | 1.9 ± 0.07 | 1.4 ± 0.05 | <0.001* |
| **In relation to LVSD occurrence at discharge** | | | |
| **Heart rate (BPM)** | 82 ± 8.81 | 72.3 ± 7.93 | <0.001* |
| **Anterior location of STEMI** | 32 (94%) | 38 (24%) | <0.001* |
| **Number of leads with ST-segment elevation (n)** | 6.1 ± 0.82 | 4 ± 0.8 | <0.001* |
| **Sum of ST-segment elevation (mm)** | 13.9 ± 2.2 | 10.6 ± 0.91 | <0.001* |
| **Maximal ST-segment elevation (mm)** | 3.9 ± 0.92 | 2.7 ± 0.8 | <0.001* |
| **Number of leads with pathologic Q waves (n)** | 4 ± 0.83 | 2 ± 0.82 | <0.001* |
| **Presence of reciprocal ST-segment depression ≥ 1mm** | 13 (38%) | 137 (87%) | <0.001* |
| **QRS duration (ms)** | 100 ± 7.42 | 92.3 ± 4.89 | <0.001* |
| **In relation to LVSD occurrence 6 months after discharge** | | | |
| **Heart rate (BPM)** | 83.6 ± 6.15 | 74.9 ± 6.33 | <0.001* |
| **ST-segment resolution (%)** | 33.3 ± 21.86 | 73.4 ± 16.83 | <0.001* |
| **ST-segment resolution (≥50%)** | 13 (38%) | 119 (76%) | <0.001* |
| **Number of leads with ST-segment elevation (n)** | 5.6 ± 1.02 | 2 ± 1.46 | <0.001* |
| **Sum of ST-segment elevation (mm)** | 9.3 ± 3.06 | 2 ± 1.42 | <0.001* |
| **Maximal ST-segment elevation (mm)** | 2.8 ± 0.8 | 1 ± 0.3 | <0.001* |
| **Number of leads with pathologic Q waves (n)** | 5.3 ± 1.08 | 2.5 ± 1.11 | <0.001* |
| **QRS duration (ms)** | 93.6 ± 4.75 | 89.9 ± 10.19 | 0.04* |

LA: left atrium end-systolic diameter, LVEDd: left ventricular end-diastolic diameter, LVEDV: left ventricular end-diastolic volume, LVEF: left ventricular ejection fraction, LVESd: left ventricular end-systolic diameter, LVESV: left ventricular end-systolic volume, LVSD: left ventricular systolic dysfunction, WMSI: wall motion score index, BPM: beats per minute, LVSD: left ventricular systolic dysfunction, PCI: percutaneous coronary intervention, STEMI: ST-segment elevation myocardial infarction

**Table 4:** Univariate logistic regression analysis for prediction of the presence of LVSD 6 months after discharge

| | Coefficient | SE | P | OR | 95% CI |
|---|---|---|---|---|---|
| **Age (years)** | 0.032 | 0.030 | 0.293 | 1.032 | 0.9727 to 1.096 |
| **Sex** | -0.717 | 0.51720 | 0.165 | 0.487 | 0.1770 to 1.3443 |
| **BMI (Kg/m$^2$)** | 0.079 | 0.056 | 0.163 | 1.082 | 0.9683 to 1.2103 |
| **Smoking** | -0.724 | 0.399 | 0.069 | 0.484 | 0.2212 to 1.0606 |
| **HTN** | -0.150 | 0.385 | 0.696 | 0.860 | 0.4040 to 1.8308 |
| **DM** | -1.902 | 0.426 | **<0.001*** | 0.149 | 0.0647 to 0.3441 |
| **Hyperlipidemia** | -0.584 | 0.383 | 0.127 | 0.557 | 0.2629 to 1.1806 |
| **Time from symptom onset to PCI** | 0.0042 | 0.003 | 0.195 | 1.004 | 0.9978 to 1.0107 |
| **IRA: LAD /other** | -3.511 | 0.633 | **<0.001*** | 0.029 | 0.0086 to 0.1032 |
| **IRA TIMI 0 flow prior to PCI** | -1.910 | 0.438 | **<0.001*** | 0.148 | 0.0626 to 0.3498 |
| **IRA TIMI 3 flow post PCI** | 2.203 | 0.569 | **<0.001*** | 9.060 | 2.966 to 27.6662 |
| **Multivessel CAD** | 0.233 | 0.386 | 0.546 | 1.262 | 0.5922 to 2.6916 |
| **Stent implantation** | -0.808 | 1.066 | 0.448 | 0.445 | 0.0551 to 3.6016 |
| **GP IIb IIIa inhibitor usage** | -1.480 | 0.396 | **0.002*** | 0.227 | 0.1046 to 0.4951 |
| **Glucose on admission (mg/dL)** | 0.129 | 0.024 | **<0.001*** | 1.138 | 1.0847 to 1.1950 |
| **Cholesterol (mg/dL)** | 1.020 | 322.48 | 0.645 | 0.786 | 0.0673 to 3.6456 |
| **TG (mg/dL)** | 0.002 | 0.001 | 0.056 | 1.002 | 0.9999 to 1.0055 |
| **HDL (mg/dL)** | 0.0191 | 0.010 | 0.063 | 1.019 | 0.9989 to 1.0402 |
| **LDL (mg/dL)** | 0.004 | 0.003 | 0.256 | 1.004 | 0.9969 to 1.0118 |
| **CKMB (U/L)** | 0.021 | 0.004 | **<0.001*** | 1.021 | 1.0141 to 1.0294 |
| **eGFR (mL/min/1.73 m$^2$)** | 0.008 | 0.011 | 0.487 | 1.008 | 0.9850 to 1.0322 |
| **BNP on admission (pg/mL)** | 0.009 | 0.006 | 0.150 | 1.009 | 0.9967 to 1.0217 |
| **BNP at discharge (pg/mL)** | 0.031 | 0.006 | **<0.001*** | 1.032 | 1.0189 to 1.0453 |
| **HR (beat/min)** | 0.148 | 0.029 | **<0.001*** | 1.159 | 1.0942 to 1.2287 |
| **SBP (mmHg)** | 0.017 | 0.014 | 0.222 | 1.018 | 0.9892 to 1.0478 |
| **DBP (mmHg)** | -0.028 | 0.0191 | 0.142 | 0.972 | 0.9365 to 1.0095 |
| **LA at discharge (mm)** | 0.753 | 0.132 | **<0.001*** | 2.125 | 1.6397 to 2.7547 |
| **LVEDd at discharge (mm)** | 0.868 | 0.162 | **<0.001*** | 2.383 | 1.7335 to 3.2764 |
| **LVESd at discharge (mm)** | 0.872 | 0.161 | **<0.001*** | 2.392 | 1.7435 to 3.2831 |
| **LVEDV at discharge (ml)** | 0.407 | 0.085 | **<0.001*** | 1.502 | 1.2706 to 1.7772 |
| **LVESV at discharge (ml)** | 0.172 | 0.026 | **<0.001*** | 1.188 | 1.1282 to 1.2519 |
| **LVEF at discharge (%)** | -0.355 | 0.057 | **<0.001*** | 0.700 | 0.6260 to 0.7844 |
| **LVEF ≤40 % at discharge** | -3.062 | 0.478 | **<0.001*** | 0.046 | 0.0183 to 0.1195 |
| **WMSI points at discharge** | 1.172 | 0.234 | **<0.001*** | 1.180 | 1.1298 to 1.2535 |
| **QRS duration (ms)** | 0.001 | 0.005 | 0.799 | 1.001 | 0.9900 to 1.0131 |
| **Anterior location of STEMI** | -3.914 | 0.752 | **<0.001*** | 0.020 | 0.0046 to 0.0872 |
| **Presence of reciprocal ST-segment** | 2.403 | 0.426 | **<0.001*** | 11.065 | 4.7971 to 25.524 |
| **Number of leads with ST-segment** | 0.125 | 0.981 | 0.342 | 2.001 | 0.8700 to 1.054 |
| **Sum of ST-segment elevation (mm)** | 1.650 | 0.31823 | **<0.001*** | 5.208 | 2.7913 to 9.7179 |
| **Maximal ST-segment elevation (mm)** | 1.698 | 0.303 | **<0.001*** | 5.465 | 3.0140 to 9.9108 |
| **Number of leads with pathologic Q waves** | 3.246 | 0.715 | **<0.001*** | 25.698 | 6.3194 to 104.5011 |
| **ST-segment resolution (≥50%)** | 1.621 | 0.391 | **<0.001*** | 5.058 | 2.3139 to 11.059 |

**Table 5**: Multivariate logistic regression analysis for prediction of the presence of LVSD at 6 months after discharge

| | Coefficient | SE | P | OR | 95% CI |
|---|---|---|---|---|---|
| **Age (years)** | -0.005 | 0.040 | 0.900 | 0.995 | 0.9207 to 1.0753 |
| **Sex** | -0.301 | 0.816 | 0.712 | 0.7403 | 0.1496 to 3.6636 |
| **BMI (Kg/m$^2$)** | 0.193 | 0.194 | 0.318 | 1.2134 | 0.8304 to 1.7730 |
| **Smoking** | -0.222 | 0.777 | 0.775 | 0.8011 | 0.1746 to 3.6751 |
| **HTN** | 0.202 | 0.194 | 0.299 | 0.6701 | 0.0549 to 8.1811 |
| **DM** | -1.318 | 1.265 | 0.297 | 0.2677 | 0.0224 to 3.1947 |
| **Hyperlipidemia** | 0.487 | 1.435 | 0.734 | 1.6274 | 0.0978 to 27.080 |
| **Time from symptom onset to PCI** | 0.002 | 0.004 | 0.946 | 1.003 | 0.9920 to 1.0087 |
| **IRA: LAD /other** | -2.068 | 1.399 | 0.139 | 0.126 | 0.0081 to 1.9624 |
| **IRA TIMI 0 flow prior to PCI** | 0.230 | 1.322 | 0.861 | 1.259 | 0.0944 to 16.805 |
| **IRA TIMI 3 flow post PCI** | 2.184 | 0.670 | **0.001*** | 8.887 | 2.3879 to 33.077 |
| **Multivessel CAD** | -0.625 | 0.545 | 0.252 | 0.535 | 0.1839 to 1.5590 |
| **Stent implantation** | -1.263 | 1.179 | 0.284 | 0.283 | 0.0281 to 2.8493 |
| **GP IIb IIIa inhibitor usage** | -0.523 | 0.554 | 0.345 | 0.593 | 0.2002 to 1.7563 |
| **Glucose on admission (mg/dL)** | 0.004 | 0.009 | 0.648 | 1.004 | 0.9863 to 1.0223 |
| **Cholesterol (mg/dL)** | 0.002 | 0.011 | 0.791 | 1.002 | 0.9813 to 1.0251 |
| **TG (mg/dL)** | 0.237 | 0.015 | 0.245 | 0.674 | 0.2310 to 6.1833 |
| **HDL (mg/dL)** | 0.381 | 0.194 | 0.052 | 1.464 | 0.9997 to 2.1457 |
| **LDL (mg/dL)** | 0.055 | 0.043 | 0.203 | 1.057 | 0.9704 to 1.1513 |
| **CKMB (U/L)** | 0.035 | 0.095 | 0.709 | 1.036 | 0.8590 to 1.2503 |
| **eGFR (mL/min/1.73 m$^2$)** | 0.106 | 0.062 | 0.091 | 1.111 | 0.9833 to 1.2572 |
| **BNP on admission(pg/mL)** | 0.063 | 0.127 | 0.618 | 1.065 | 0.8297 to 1.3689 |
| **BNP at discharge (pg/mL)** | 0.1005 | 0.116 | 0.387 | 1.105 | 0.8805 to 1.3885 |
| **HR (beat/min)** | -2.283 | 1.323 | 0.084 | 0.101 | 0.0076 to 1.3638 |
| **SBP (mmHg)** | 0.016 | 0.0148 | 0.276 | 1.016 | 0.9871 to 1.0463 |
| **DBP (mmHg)** | -0.183 | 0.098 | 0.063 | 0.832 | 0.6862 to 1.0103 |
| **LA at discharge (mm)** | 0.331 | 0.365 | 0.365 | 1.392 | 0.6796 to 2.8531 |
| **LVEDd at discharge (mm)** | 0.296 | 0.171 | 0.084 | 1.345 | 0.9602 to 1.8840 |
| **LVESd at discharge (mm)** | 0.704 | 0.366 | 0.054 | 2.023 | 0.9872 to 4.1468 |
| **LVEDV at discharge (ml)** | 0.212 | 0.129 | 0.101 | 1.236 | 0.9592 to 1.5949 |
| **LVESV at discharge (ml)** | 0.422 | 0.243 | 0.463 | 0.276 | 0.6892 to 1.2349 |
| **LVEF at discharge (%)** | -0.032 | 0.127 | 0.674 | 0.456 | 0.5493 to 1.0682 |
| **LVEF ≤40 % at discharge** | -0.010 | 0.037 | 0.769 | 0.989 | 0.9193 to 1.0642 |
| **WMSI points at discharge** | 0.032 | 0.030 | 0.292 | 1.0326 | 0.9727 to 1.0963 |
| **QRS duration (ms)** | 0.211 | 0.081 | **0.009*** | 1.235 | 1.0535 to 1.4499 |
| **Anterior location of STEMI** | 0.498 | 0.084 | **<0.001*** | 1.646 | 1.3956 to 1.9424 |
| **Presence of reciprocal ST-segment** | 0.0967 | 0.079 | 0.2210 | 1.101 | 0.9435 to 1.2862 |
| **Number of leads with ST segment** | -0.084 | 0.115 | 0.464 | 0.919 | 0.7338 to 1.1518 |
| **Sum of ST-segment elevation (mm)** | 0.055 | 0.091 | 0.544 | 1.056 | 0.8840 to 1.2635 |
| **Maximal ST-segment elevation (mm)** | 0.064 | 0.069 | 0.344 | 1.066 | 0.9335 to 1.2182 |
| **Number of leads with pathologic Q waves** | -0.044 | 0.090 | 0.627 | 0.956 | 0.8009 to 1.1434 |
| **ST-segment resolution (≥50%)** | 0.073 | 0.100 | 0.464 | 1.076 | 0.8836 to 1.3115 |

BMI: body mass index, HTN: hypertension, DM: diabetes mellites, PCI: Percutaneous coronary intervention, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate, BNP: B-type natriuretic peptide; CK-MB: maximal activity of isoenzyme MB of creatinine kinase, eGFR: estimated glomerular filtration rate, HDL: high-density-lipoprotein, IRA: infarct-related artery, LAD: left anterior descending artery, LDL: low-density-lipoprotein, LVSD: left ventricular systolic dysfunction, TIMI: thrombolysis in myocardial infarction score, LA: left atrium end-systolic diameter, LVEDd: left ventricular end-diastolic diameter, LVEDV: left ventricular end-diastolic volume, LVEF: left ventricular ejection fraction, LVESd: left ventricular end-systolic diameter, LVESV: left ventricular end-systolic volume, LVSD: left ventricular systolic dysfunction, WMSI: wall motion score index, STEMI: ST-segment elevation myocardial infarction, SE: standard error, OR: odds ratio, CI: confidence interval, *: statistically significant as P value <0.05.

**Table 6:** Multiple regression analysis for prediction of LVEF deterioration at 6 months after discharge

| | **Coefficient** | **SE** | **t** | **P** | **$r_{partial}$** | **$r_{semipartial}$** |
|---|---|---|---|---|---|---|
| **Age (years)** | -0.097 | 0.071 | -1.350 | 0.179 | -0.099 | 0.097 |
| **Sex** | 0.719 | 1.276 | 0.563 | 0.574 | 0.041 | 0.040 |
| **BMI ($Kg/m^2$)** | -0.430 | 0.079 | -5.402 | **<0.001*** | -0.364 | 0.3649 |
| **Smoking** | 1.786 | 1.382 | 1.293 | 0.198 | 0.095 | 0.093 |
| **HTN** | 0.028 | 1.965 | 0.014 | 0.989 | 0.001 | 0.001 |
| **DM** | -2.873 | 2.808 | -1.023 | 0.308 | -0.075 | 0.073 |
| **Hyperlipidemia** | 2.616 | 2.141 | 1.221 | 0.224 | 0.090 | 0.088 |
| **Time from symptom onset to PCI** | -0.010 | 0.008 | -1.312 | 0.191 | -0.096 | 0.094 |
| **IRA: LAD /other** | -0.080 | 0.061 | -1.305 | 0.1933 | -0.094 | 0.082 |
| **IRA TIMI 0 flow prior to PCI** | -1.95 | 1.302 | -1.501 | 0.135 | -0.108 | 0.095 |
| **IRA TIMI 3 flow post PCI** | -4.962 | 1.365 | -3.634 | **<0.001*** | -0.255 | 0.223 |
| **Multivessel CAD** | -1.271 | 0.937 | -1.357 | 0.176 | -0.098 | 0.093 |
| **Stent implantation** | 1.753 | 1.955 | 0.896 | 0.371 | 0.065 | 0.061 |
| **GP IIb IIIa inhibitor usage** | -0.119 | 0.066 | -1.791 | 0.074 | -0.129 | 0.122 |
| **Glucose on admission (mg/dL)** | -0.172 | 0.026 | -6.391 | **<0.001*** | -0.420 | 0.421 |
| **Cholesterol (mg/dL)** | 0.019 | 0.026 | 0.736 | 0.462 | 0.053 | 0.032 |
| **TG (mg/dL)** | 0.024 | 0.025 | 0.963 | 0.336 | 0.0701 | 0.0395 |
| **HDL (mg/dL)** | -0.055 | 0.075 | -0.734 | 0.464 | -0.054 | 0.032 |
| **LDL (mg/dL)** | 0.017 | 0.030 | 0.569 | 0.570 | 0.042 | 0.024 |
| **CKMB (U/L)** | 0.020 | 0.025 | 0.805 | 0.422 | 0.058 | 0.034 |
| **eGFR (mL/min/1.73 $m^2$)** | 0.010 | 0.047 | 0.209 | 0.835 | 0.015 | 0.009 |
| **BNP on admission (pg/mL)** | -0.020 | 0.010 | -1.99 | 0.054 | -0.144 | 0.0873 |
| **BNP at discharge (pg/mL)** | -0.024 | 0.026 | -0.897 | 0.370 | -0.065 | 0.039 |
| **HR (beat/min)** | 0.058 | 0.039 | 1.471 | 0.142 | 0.106 | 0.095 |
| **SBP (mmHg)** | 0.027 | 0.021 | 1.307 | 0.193 | 0.095 | 0.057 |
| **DBP (mmHg)** | 0.061 | 0.041 | 1.482 | 0.140 | 0.107 | 0.101 |
| **LA at discharge (mm)** | -0.091 | 0.123 | -0.736 | 0.463 | -0.054 | 0.032 |
| **LVEDd at discharge (mm)** | -0.111 | 0.111 | -1.005 | 0.316 | -0.074 | 0.043 |
| **LVESd at discharge (mm)** | -0.082 | 0.114 | -0.715 | 0.476 | -0.053 | 0.031 |
| **LVEDV at discharge (ml)** | -0.051 | 0.029 | -1.769 | 0.079 | -0.130 | 0.076 |
| **LVESV at discharge (ml)** | -0.130 | 0.042 | -3.092 | 0.055 | -0.223 | 0.133 |
| **LVEF at discharge (%)** | 0.195 | 0.064 | 3.061 | 0.086 | 0.221 | 0.132 |
| **LVEF ≤40 % at discharge** | 1.553 | 0.704 | 2.207 | 0.0.63 | 0.161 | 0.095 |
| **WMSI points at discharge** | -12.107 | 2.694 | -4.494 | 0.076 | -0.315 | 0.194 |
| **QRS duration (ms)** | -1.906 | 0.303 | -6.300 | **<0.001*** | -0.421 | 0.293 |
| **Anterior location of STEMI** | 2.242 | 0.702 | 3.194 | **0.002*** | 0.229 | 0.149 |
| **Presence of reciprocal STsegment** | -2.343 | 1.195 | -1.960 | 0.052 | -0.143 | 0.091 |
| **Number of leads with ST-segment** | -0.025 | 0.053 | -0.472 | 0.638 | -0.035 | 0.022 |
| **Sum of ST-segment elevation (mm)** | 1.766 | 0.206 | -8.568 | 0.054 | -0.528 | 0.471 |
| **Maximal ST-segment elevation(mm)** | -1.702 | 0.385 | -4.413 | 0.056 | -0.305 | 0.242 |
| **Number of leads with pathologic Q waves** | -1.348 | 0.320 | -4.217 | **<0.001*** | -0.297 | 0.196 |
| **ST-segment resolution (≥50%)** | 0.621 | 1.000 | 0.621 | 0.535 | 0.046 | 0.029 |

## Discussion

In the current study, demographic data including (age, sex, weight, height, and time from symptom onset to PCI) were insignificantly different between the studied groups. Systolic and diastolic blood pressure were insignificantly different among the studied groups. Risk factors for CAD (BMI, smoking, hypertension, hyperlipidemia) were insignificantly different among the studied groups. The prevalence of diabetes mellitus was significantly higher in LVSD group compared to non-LVSD group (P=0.01).

In agreement with our results, a study was carried out to assess the performance of ten ECG parameters regarding the prediction of LVSD after a first STEMI. They analyzed 249 patients (74.7% males) treated with primary PCI. They reported similar demographic data as the current study, also at baseline, patients who presented with LVSD after 6 months of follow-up showed a higher prevalence of diabetes mellitus (4).

In terms of the blood, kidney and lipide profiles, Hb, WBCs, platelets, INR, ALT, and AST were insignificantly different among the studied groups. FBG was significantly higher in LVSD group compared to non-LVSD group (P<0.001). eGFR, was significantly reduced in LVSD group compared to non-LVSD group (P<0.001). Glucose on admission, CK-MB, Cholesterol, Triglycerides, HDL, LDL, BNP on admission and BNP at discharge were significantly higher in LVSD group compared to non-LVSD group (P<0.001).

Supporting our results, reported that eGFR (CKD-EPI equation) [mL/min/1.73 m2] was significantly higher in patients with LVSD at 6 Months than patients without LVSD at 6 Months [80.3 (72.8–88.1) vs. 86.5 (75.0–96.6), respectively, P = 0.036]. Moreover, glucose on admission, CK-MB, Cholesterol, Triglycerides, HDL, LDL, BNP on admission and BNP at discharge were significantly higher in LVSD group compared to non-LVSD group (P<0.001 for each) (4).

In the current work, the prevalence of IRA: LAD/other, IRA TIMI 0 flow prior to PCI, IRA TIMI 3 flow post PCI and GP IIb/IIIa inhibitor usage were significantly higher in LVSD group compared to non-LVSD group (P<0.001). The prevalence of multivessel CAD and stent implantation were insignificantly different between the two studied groups.

A study reported that patients who presented with LVSD after 6 months of follow-up showed a higher prevalence of left anterior descending artery (LAD) as the IRA and TIMI 0 flow before PCI, but less frequent TIMI 3 flow post PCI. Interestingly, patients with LVEF ≤ 40% at the time of discharge from hospital, but not 6 months after STEMI (n = 39), when compared with those presenting with LVEF ≤40% both at hospital

discharge and LVSD 6 months after STEMI (n = 45), had a lower proportion of the LAD as the IRA (31 vs. 42; p = 0.058), more frequent TIMI 3 flow in the IRA following PCI (38 vs. 34; p = 0.002) and lower values of cardiac biomarkers, including maximal concentration of cardiac troponin I (50.0 vs. 50.0 ng/mL; p = 0.039), maximal activity of CK-MB (354 vs. 555 U/L; p < 0.001) and BNP concentration on hospital discharge (177.3 vs. 439.3 pg/mL; p < 0.001) (4).

Echocardiographic characteristics at discharge including LA, LVEDd, LVESd, LVEDV, LVESV, LVEF ≤40 %, and WMSI were significantly higher in LVSD group compared to non-LVSD group (P<0.001). LVEF was significantly reduced in LVSD group compared to non-LVSD group (P<0.001). Echocardiographic characteristics 6 months after discharge including LA, LVEDd, LVESd, LVEDV, LVESV, and WMSI were significantly higher in LVSD group compared to non-LVSD group (P<0.001). LVEF was significantly reduced in LVSD group compared to non-LVSD group (P<0.001).

In harmony with our findings, a study reported that echocardiographic parameters such as LA, LVEDd, LVESd, LVEDV, LVESV, LVEF ≤40%, and WMSI at discharge and six months post-discharge were significantly elevated in the LVSD group, with a notably lower LVEF, compared to the non-LVSD group (P<0.001 for all). Furthermore, within 6 months of STEMI, a significant increase in median values of LVEF from 44% to 46% could be noted, leading to a decline in the percentage of patients with LVEF ≤40% from a baseline value of 33.7% to 20.9% after 6 months (p < 0.001) (4).

In the present study, electrocardiographic characteristics at discharge including heart rate, anterior location of STEMI, number of leads with ST-segment, sum of ST-segment elevation, maximal ST-segment elevation, number of leads with pathologic Q waves, and QRS duration were significantly higher in LVSD group compared to non-LVSD group (P<0.001). The prevalence of reciprocal ST-segment depression ≥ 1mm was significantly reduced in LVSD group compared to non-LVSD group (P<0.001). Electrocardiographic characteristics 6 months after discharge including heart rate, number of leads with ST-segment elevation, sum of ST-segment elevation, maximal ST-segment elevation, number of leads with pathologic Q waves, and QRS duration were significantly higher in LVSD group compared to non-LVSD group (P<0.001). ST-segment resolution and the prevalence of ST-segment resolution (≥50%) were significantly reduced in LVSD group compared to non-LVSD group (P<0.001).

Interestingly, a study observed that in post-PCI ECG assessments, reduced ST-segment resolution, and longer QRS duration were linked to LVSD after six

months. They noted a trend where increased QRS duration at admission and post-PCI correlated with a higher LVSD incidence (admission OR: 1.59, 95% CI: 0.80–3.17, p=0.180; post-PCI OR: 3.42, 95% CI: 1.76–6.66, p<0.001). Moreover, they found significantly lower LVEF values in the longest QRS duration tercile both at baseline and post-PCI, compared to the lower and middle terciles (4).

The univariate logistic regression analysis identified diabetes mellitus, infarct-related artery (IRA) characteristics, GP IIb/IIIa inhibitor usage, admission glucose, CKMB, heart rate, echocardiographic parameters at discharge, and electrocardiographic features as significant predictors for LVSD six months post-discharge. However, the multivariate logistic regression analysis refined these predictors to IRA TIMI 3 flow post PCI, QRS duration, and the anterior location of STEMI. Additionally, multiple regression analysis further pinpointed high BMI, elevated glucose level on admission, QRS duration, anterior location of STEMI, and the number of leads with pathologic Q waves as key factors for LVEF deterioration six months after discharge.

Our results were compatible with a study conducted univariate regression analysis to explore potential predictors of LVSD six months post-STEMI and found a significant association with most electrocardiographic parameters at hospital admission and post-PCI, except for baseline QRS duration. Notably, reciprocal ST-segment depression ≥ 1mm at baseline suggested a reduced risk of LVSD. Angiographic predictors included LAD involvement as the IRA and GP IIb/IIIa inhibitor use, while TIMI 0 flow before PCI and TIMI 3 flow post-PCI were linked to a lower LVSD risk. Predictive biochemical variables were admission glucose, peak cardiac troponin I, CK-MB activity, and discharge BNP levels, with diabetes mellitus as the sole clinical predictor. A subsequent multivariate logistic regression analysis pinpointed the anterior location of STEMI, longer post-PCI QRS duration, and impaired post-PCI IRA flow as independent predictors of LVSD six months after STEMI (4).

Another study was done and revealed that three parameters related to ST-segment elevation were analyzed: the number of leads with ST-segment elevation, the sum of ST-segment elevations and the amplitude of maximum ST-segment elevation in a single lead. All of them were associated with LVR occurrence in the univariate regression models. Moreover, multivariate models, including all data obtained on hospital admission or during primary PCI (performed just after admission), showed that the number of leads with ST-segment elevation and the sum of ST-segment elevations were electrocardiographic predictors of LVR and LVEDV increase after 6 months of STEMI. When analyzed as tertiles, it showed that a particularly high prevalence of LVR (exceeding 30%)

occurred in the second and third tertile, corresponding to >3 leads with ST-segment elevations and a sum of ST-segment elevations >5 mm (13).

## Conclusion

The study conclusively demonstrates that specific baseline and post-PCI electrocardiographic parameters, including elevated heart rate, anterior location of STEMI, and extensive ST-segment elevation are significant predictors of LVSD six months after a first STEMI. These findings show the critical importance of early, comprehensive electrocardiographic evaluation in patients experiencing a first STEMI for identifying those at increased risk of developing LVSD, facilitating early intervention and potentially improving long-term cardiac outcomes.

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