

PREDICTORS OF DEPRESSED LEFT VENTRICULAR FUNCTION IN PATIENTS PRESENTING WITH ST-ELEVATION MYOCARDIAL INFARCTION

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ABSTRACT

Background: Left ventricular dysfunction after ST segment elevation myocardial infarction (STEMI) is the most important factor affecting morbidity and mortality. The primary objective of reperfusion therapy is to restore epicardial flow and to reperfuse the myocardial tissue. However, in some cases lack of microvascular reperfusion and, consequently, significant left ventricular (LV) dysfunction persist despite the rapid and sustained restoration of blood flow. Early in the course of STEMI, therapies that may harm patients who develop LV dysfunction, such as beta-blockers, are often administered.

Aim of the work: construct a predictive score of the risk of developing left ventricular dysfunction in patients with STEMI.

Patient and methods: This study was done in Cardiology department, Zagazig University on 100 patients; 78 males (78%) & 22 females (22%) with first acute STEMI underwent successful primary percutaneous intervention (pPCI). Within 72 hours of admission, all of the following parameters were reported: risk factors, blood pressure, heart rate, KILLIP class, need for mechanical ventilation (MV), cardiac enzymes, admission blood sugar, kidney function, lipid profile, electrocardiographic data (ST elevation in millimeters (mm), location of infarction, ST resolution, QRS width in milliseconds (ms), Selvester score), echocardiography (ejection fraction by Simpson's method, wall motion score index) and angiographic data (culprit artery, Total number of diseased vessels, severity of coronary artery disease, Gensini score and door to balloon time).

Results: LV dysfunction was associated with decreased renal function (creatinine >1.1 mg/dl, $p=0.029$), number of diseased vessels >2 ($p=0.034$), absence of complete ST segment resolution ($p<0.001$), wall motion score index > 2.05 ($p=0.008$), sum of ST segment elevation >13.5 mm ($p<0.001$), QRS width >98.5 ms ($p<0.001$), Selvester score >3.5 ($p<0.001$) Gensini score >95 ($p<0.001$), anterior location of infarction ($p=0.002$) and KILLIP class >1 ($p=0.003$). A prediction score using these variables stratified patients into low, intermediate and high-risk groups for LV dysfunction; positive likelihood ratios for LV dysfunction in these groups were 2.9, 2.1, and 2.4, respectively.

Conclusions: 10 key predictors of in-hospital LV dysfunction after STEMI were identified; a risk score based on these predictors helps to quickly identify patients presenting with STEMI who are at the highest risk for developing significant LV dysfunction and could guide optimal therapeutic choices.

Key words: ST-elevation myocardial infarction, left ventricular dysfunction, predictors, score, primary angioplasty.

INTRODUCTION

Although the incidence of ST-segment elevation myocardial infarction (STEMI) has decreased over the past decade, it remains a common and morbid diagnosis [1].

It is a life-threatening situation, rapid and correct decision making for life saving of patients in emergency room is very important [2].

Primary treatment for patients with acute STEMI is fibrinolysis or primary angioplasty. The use of primary PCI has improved the outcome of STEMI patients significantly [3].

Ischemic heart disease is the most common contributor to left ventricular dysfunction. The extent of left ventricular (LV) function varies considerably among patients with extensive coronary disease, and clinical and angiographic factors associated with LV impairment are poorly characterized. Specifically, whether clinical, demographic and angiographic characteristics differ among patients and are predictive of LV ejection fraction has not been determined [4].

Despite aggressive primary therapy, prognosis remains poor in patients with large

infarctions and severe left ventricular (LV) dysfunction [5].

Early in the course of ST-segment elevation myocardial infarction (STEMI), therapies that may harm patients who develop left ventricular (LV) dysfunction are often administered. Early identification, preferably in the emergency department (ED), of patients at highest risk for developing left ventricular (LV) dysfunction could serve to inform the use of certain therapies [6].

AIM OF THE WORK

The present study aimed to construct a predictive score of the risk of developing left ventricular dysfunction in patients with ST segment elevation myocardial infarction.

PATIENTS AND METHODS

This study was carried out in Zagazig University Hospitals; it was conducted from January 2012 to August 2013. It included (100) patients admitted with first acute ST segment elevation myocardial infarction (STEMI). Informed consent obtained from every patient on

participation of the study. Medical research and ethics committee approved the study.

Inclusion Criteria:

Patients with first acute STEMI. STEMI was diagnosed by elevation of cardiac biomarkers associated with symptoms of ischemia and a new ST elevation, presumed new onset left bundle branch block (LBBB) or pathological Q waves.

ST-segment elevation was defined as J-point elevation in 2 contiguous leads of 2 mm in leads V1, V2, or V3 and 1 mm in other leads. ST-segment depression 1 mm in leads V1 to V3, consistent with a posterior STEMI was also considered ST-segment elevation [6].

Those with previous history of coronary artery disease other than previous myocardial infarction were also included in the study group.

Exclusion criteria:

Patients with previous Myocardial infarction, Previous diagnosis of heart failure, Pre-existing significant valvular heart disease, Patients with old left bundle branch block (LBBB), those who did not do echocardiography during the index STEMI (within 72 hours of admission) or those whom it was difficult to delineate the endocardial border by Simpson's method, Patients with in-stent restenosis, Patients with previous CABG.

Grouping the patients: Patients were divided according to the ejection fraction (EF) by echocardiography in to 2 groups: **Group (I):** 36 patients with $EF \leq 40\%$, **Group (II):** 64 patients with $EF > 40\%$

All patients were subjected to all of the following:

A) Complete history taking.

B) Thorough physical examination including.

C) Resting 12-lead standard surface electrocardiogram:

STEMI was diagnosed according to the following: New ST segment elevation at J-point in ≥ 2 contiguous leads of ≥ 2 mm in leads V1, V2, or

V3 and ≥ 1 mm in other leads. ST-segment depression ≥ 1 mm in leads V1 to V3, consistent with a posterior STEMI, was considered as ST-segment elevation[6]. ST segment elevation, measured 20 ms after the J point. The height (in mm) of ST segment elevations was measured in leads I, aVL, and V1 through V6 for anterior infarction; leads II, III, aVF for inferior infarction and leads V5 to V6 for lateral. The sum of all measured ST segment elevations was expressed as STE. Resting ECG was repeated for all patients 90–120 minutes following first balloon inflation, from which the sum of ST segment elevations was measured again. The difference between both was then taken and divided by the initial one. The result then accepted as ST segment elevation resolution (STR) and expressed as a percentage.

$$STR = \frac{\text{Initial } (\Sigma \text{STE}) - (\Sigma \text{STE}) \text{ on the second ECG}}{\text{Initial } (\Sigma \text{STE})}$$

Patients were divided into three groups according to the degree of ST-segment resolution: $< 30\%$ (no resolution); 30% to 70% (partial resolution); and $> 70\%$ (complete resolution) [7].

QRS width was measured manually using calipers. The lead recording the widest QRS was used for the analysis. Development of atrial fibrillation or ventricular fibrillations during course of admission was reported for every patient.

Estimating infarction size using simplified Selvester QRS score was applied for discharge ECG. The simplified version of the Selvester QRS scoring system uses 50 weighted analysis of QRS complexes in ten leads (I, II, VL, VF and V1 to V6), totaling a maximum of 31 points. (Table 1). Each point being equivalent to approximately 3% of the left ventricular mass [8].

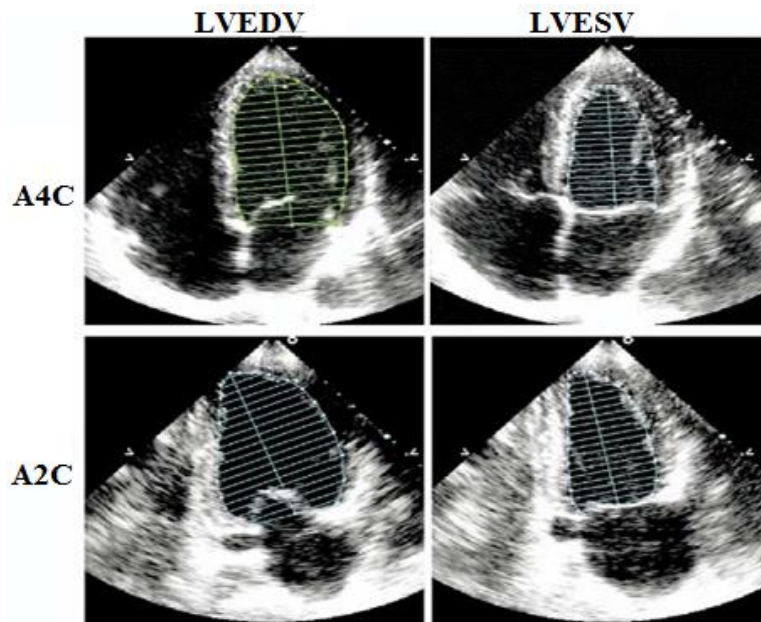


Figure 1: Method of calculation of biplane Simpson method [9].

Visual semi quantitative assessment of regional wall motion and thickening for WMSI was performed. We used the 16-segment model recommended by the ASE [10]. The score for each segment was graded according to the following system: normal, 1; hypokinesia, 2;

akinesia, 3; dyskinesia, 4. The total wall motion score (WMS) was obtained by adding the score for each segment. The WMSI was calculated by dividing the total wall motion score by 16, as shown in Figure 2.

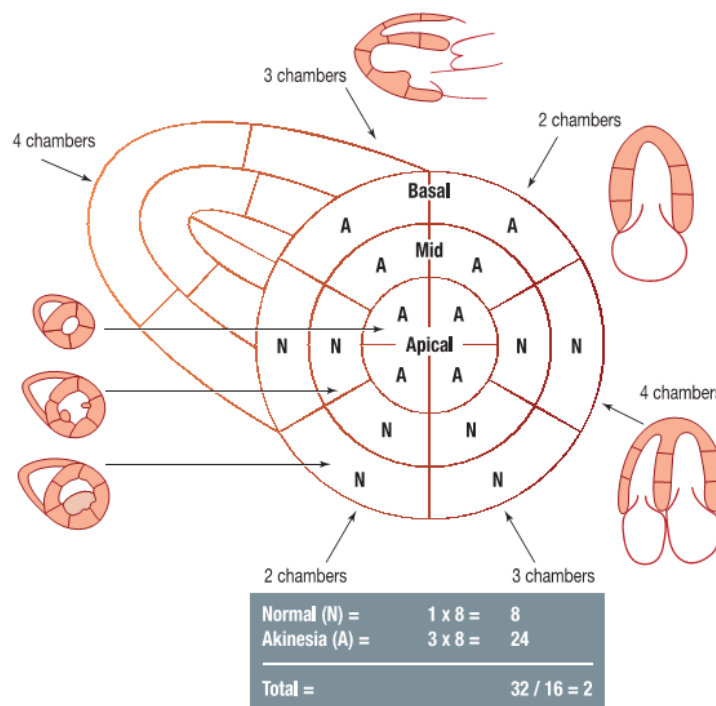


Figure 2: Wall motion score index calculation. If a patient has eight normal segments and eight akinetic segments the wall motion score would be calculated as 32 and the wall motion score index would be 32/16 = 2 [11].

F) Coronary Angioplasty:

All patients transferred directly to cath lab in a timely fashion in accordance with guidelines,

the door to balloon time (DBT) was calculated. All patients received chewable aspirin 300 mg and clopidogrel 600 mg before the procedure.

Coronary angiography was done in the standard fashion; the common femoral artery was punctured using seldinger's technique. Coronary angiography was performed in multiple projections for adequate analysis of target lesions. Export aspiration catheter for thrombus aspiration then drug eluting stent (Endeavor resolute integrity) was put either after balloon dilatation or directly only in IRA [12].

Total number of vessels having lesions as well as number of total occlusions other than

culprit one were calculated. Coronary lesions other than culprit one were considered significant if any stenosis of $\geq 70\%$ in at least one major epicardial coronary artery. For left main (LM), lesion $\geq 50\%$ was considered significant. The extent of coronary atherosclerosis was assessed by the modified Gensini score [13]. In this score, angiographic coronary artery disease extension points calculated from stenosis score \times segment score in 8 coronary segments [14]. Table 2

Table 2: Modified Gensini risk score [14].

Segment	Score
LM	5
LAD	20
LCX	20
RCA	20
D ₁	10
OM ₁	10
PDA	10
S ₁	5

Stenosis Percentage	Score
1-49	1
50-74	2
75-99	3
100	4

Statistical analysis

All statistical data were processed using the IBM SPSS 19 software. Data were expressed as mean \pm standard deviation (SD). Student t-test, and chi-square test were used to compare the variables. Correlations between the ejection fraction and other parameters were analyzed. A stepwise multivariate analysis was done for independent variables. Odds ratios and 95% confidence intervals were also calculated. A P value of less than 0.05 was considered significant. A prediction score was developed to provide a schema for the assessment of a patient's likelihood of having depressed LV function using

variables readily accessible during patient admission. Key predictors from the multivariate logistic regression as well as likely significant confounders based on clinical knowledge were included in the prediction score. Cutoffs were established with the design of maximizing the sensitivity of the prediction score. Fit was assessed using a receiver-operating characteristic curve.

RESULTS

The patients were divided according to ejection fraction (EF) by echocardiography in to 2 groups: Group (I): 36 patients with EF $\leq 40\%$, Group (II): 64 patients with EF $> 40\%$.

Table 3: Demographic and clinical data in the study groups (I&II) according to ejection fraction.

Variable	Group I (n) = 36	Group II (n) = 64	P-Value
Age ($\chi \pm SD$)	54.1 \pm 10.2	51.7 \pm 8.1	0.234 NS
Gender n (%)	Female	10 (27.8%)	0.2 NS
	Male	26 (72.2%)	
HTN n (%)	15 (41.7 %)	34 (53.1%)	0.2 NS
Known DM n (%)	22 (61.1%)	37 (57.8%)	0.7 NS
Smoker n (%)	14 (38.9 %)	30 (46.9%)	0.4 NS
Dyslipidemia n (%)	21 (58.3%)	40 (62.5%)	0.6 NS
CAD history n (%)	10 (27.8%)	17 (26.6%)	0.89 NS
SBP ($\chi \pm SD$)	130.03 \pm 16.9	131.61 \pm 20.69	0.680 NS
DBP ($\chi \pm SD$)	80.42 \pm 10.72	82.58 \pm 13.1	0.375 NS
Heart rate ($\chi \pm SD$)	76.03 \pm 13.84	73.34 \pm 14.22	0.360 NS
KILLIP class n (%)	I	19 (52.8%)	0.003*
	II	17 (47.2%)	
Need for MV n (%)	19 (52.8%)	2 (3.1%)	0.001**

n= Number, χ =mean, SD=standard deviation, MV= mechanical ventilation, NS= non- significant, * significant, ** highly significant

Table 4: Electrocardiographic data in the study groups (I&II) according to ejection fraction.

Variable	Group I (n) = 36	Group II (n) = 64	P-Value
Max. ST elevation (mm) ($\chi \pm SD$)	17 \pm 4.7	12.6 \pm 4.4	0.00 **
QRS width (ms) ($\chi \pm SD$)	104 \pm 7.5	88.23 \pm 14.7	0.00 **
Selvester score ($\chi \pm SD$)	5.4 \pm 1.6	2.9 \pm 1.5	0.00 **
Location of infarction n (%)	Anterior	25 (69.5%)	0.002*
	Inferior	11 (30.6%)	
	Antero-lateral	0 (0%)	
STR n (%)	Complete	2 (5.6%)	0.00**
	Partial	30 (83.3%)	
	Absent	4 (11.1%)	
AF n (%)	0 (0%)	0 (0%)	-
VF n (%)	4 (11.1%)	2 (3.1%)	0.1 NS

STR= ST segment resolution, ms= milliseconds, AF= atrial fibrillation, VF= Ventricular fibrillation.

Table 5: Laboratory data in the study groups (I&II) according to ejection fraction.

Variable	Group I (n) = 36	Group II (n) = 64	P-Value
	$\chi \pm SD$	$\chi \pm SD$	
Peak CK-MB (ng/ml)	166.5 \pm 71.2	186.12	0.15 NS
Troponin (ng/ml)	2.98 \pm 2.5	2.7 \pm 2.35	0.63 NS
Creatinine (mg/dl)	1.2 \pm 0.5	0.95 \pm 0.34	0.007*
Cholesterol (mg/dl)	176.4 \pm 48.8	185.9 \pm 48.7	0.35 NS
TG (mg/dl)	118.9 \pm 54.99	144.8 \pm 111.2	0.19 NS
HDL (mg/dl)	38.9 \pm 7.8	38.9 \pm 7.96	0.96 NS
LDL (mg/dl)	108.62	108.3 \pm 32.2	0.96 NS
RBS (mg/dl)	200.48 \pm 91.3	195.9 \pm 85.6	0.81 NS

HDL: high density lipoprotein, LDL: Low density lipoprotein, CK-MB: creatine kinase myocardial band, RBS: random blood sugar

Table 6: Echocardiographic data the in study groups

Variable	Group I (n) = 36	Group II (n) = 64	P-Value
	($\chi \pm SD$)	($\chi \pm SD$)	
EDV mL	103 \pm 18	83 \pm 18	<0.001**
ESV mL	66 \pm 13	37 \pm 11	<0.001**
Biplane EF %	35.6 \pm 5.19	54.4 \pm 5.6	0.00**
WMSI	2.23 \pm 0.2	1.5 \pm 0.22	0.00**

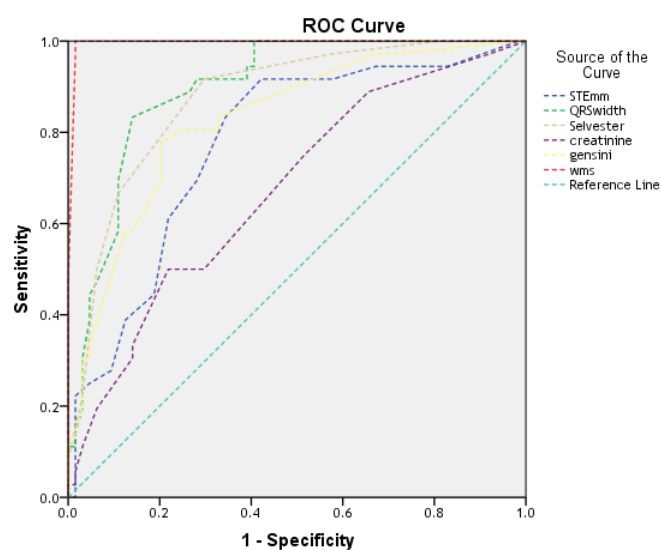
EDV= end diastolic volume, ESV= end systolic volume, EF= ejection fraction, WMSI= wall motion score index, mL= milliliter

Table 7: Angiographic data in the study groups (I&II)

Variable	Group I (n) =36	Group II (n) = 64	P-value	
Number of totally diseased vessels	Single	6 (16.7%)	39 (60.9%)	0.00 **
	Two	14 (38.9%)	20 (31.2%)	
	Three	11 (30.6%)	3 (4.7%)	
	Four	4 (11.1%)	2 (3.1%)	
	Six	1 (2.8%)	0 (0%)	
Culprit artery	LAD	25 (69.4)	19 (29.7%)	0.002*
	LCX	3 (8.3%)	13 (20.3%)	
	OM2	0 (0%)	1 (1.6%)	
	RCA	8 (22.2%)	31 (48.4%)	
No. of totally occluded vessels including culprit	One	34 (94.4%)	62 (96.9%)	0.5 NS
	Two	2 (5.6%)	2 (3.1%)	
Other significant vessels	27 (75%)	15 (23.4%)	0.00**	
Culprit vessel severity ($\chi \pm SD$)	99.1 \pm 1.7	97.23 \pm 12.5	0.375 NS	
Gensini score ($\chi \pm SD$)	136.11 \pm 40.5	87.9 \pm 31.31	0.00**	
DBT ($\chi \pm SD$)	80.7 \pm 22.23	81.02 \pm 21.71	0.939 NS	

χ = mean, SD= standard deviation, LAD= left anterior descending, LCX= left circumflex, RCA= right coronary artery, OM= obtuse marginal, DBT= door to balloon time, Significant vessel= left main > 50 % or non-left main lesion 70%.

A ROC curve was then constructed for significant continuous quantitative variables and area under the curve AUC, cut off point for every variable was determined as follows: figure 3

**Figure 3:** Significant quantitative continuous variables.

By multivariate analysis, we found four significant independent predictors of depressed left ventricle function (EF \leq 40%). They were creatinine level (p= 0.029), total number of affected vessels $>$ 2 (p= 0.034), complete STR $>$ 70% (p= 0.00) and WMSI (p=0.008).

A score then constructed containing both significant independent and dependent predictors.

Relative significance for each predictor given based on the significant predictor with the least value, each one of quantitative predictors was given the score if it was equal or above the cutoff point. A total score of 42 was then calculated (table 8)

Table 8: Score for prediction of depressed left ventricle function (EF \leq 40%)

Variable	Score
STE mm $>$ 13.5 mm	+3
QRS width $>$ 98.5 ms	+6
Selvester score $>$ 3.5	+4
Creatinine $>$ 1.1 mg/dl	+1
No. of Total diseased vessels $>$ 2	+3
Gensini score $>$ 95	+4
WMSI $>$ 2.05	+11
Anterior infarction	+3
KILLIP $>$ I	+1
Complete STR $<$ 70	+6
Total score	+42

To maximize clinical applicability, cut point of 21 for prediction score was set. At cut point of $>$ 21 (with higher scores representing a higher

likelihood of having depressed left ventricle function), test sensitivity was 97.2% and specificity was 98.4%. Table 9

Table 9: Consistency of the predictive score at cut off point 21

Sensitivity	specificity	+VE predictive	-VE predictive	accuracy	KAPPA AGREEMENT	P
97.2%	98.4%	97.2%	98.4%	98%	0.957	0.00**

A 3 tiered score was then constructed to divide patients in to 3 categories:

- Low risk (score 0-14) with likelihood ratio of 2.9
- Intermediate risk (score 15-28) with likelihood ratio of 2.1
- high risk (score 29-42) with likelihood ratio of 2.4

DISCUSSION

The present study showed no statistical significant difference between the two groups as regard age and sex, hypertension HTN, diabetes mellitus DM, smoking and dyslipidemia. This was concordant with **Bhave et al. [6]**.

Regarding age, our study was discordant with **Ali et al. [15]** who stated that older age may predict LV dysfunction and heart failure in patients treated with thrombolysis for STEMI. This discrepancy could be explained by larger sample volume included in his study, higher mean age and different modalities of revascularization.

Regarding sex, our result was discordant with **Shacham et al. [16]**. In their study, they

concluded that female patients demonstrated worse systolic and diastolic LV function, despite receiving similar treatment as male patients. This discrepancy could be explained by older females in their study and the co-morbidities they had in their study.

Regarding diabetes mellitus and smoking, our study was discordant with **Mateus et al. [17]**. They stated that a history of diabetes might be related to the likelihood of impaired LV systolic function. In addition, smoking increased the risk of left ventricular dysfunction compared to non- or ex-smokers. This discrepancy might be attributed to different point to define LV dysfunction (they used EF of 45 %).

Our study showed no significant difference between the two groups regarding history of coronary artery disease (CAD); this was concordant with **Świątkiewicz et al. [18]** but was discordant with **Bhave et al. [6]**. In their study, a history of coronary artery disease was associated with a 3.1-fold increase in the odds of having depressed LV function ($p=0.014$).

Our study showed no significant difference between the two groups regarding systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR). This was concordant with **Hamdan et al. [19]**.

Regarding SBP, our result was discordant with **Jia et al. [20]**. In their study, SBP was an independent predictor of LV dysfunction in a multivariate regression analysis. Larger sample volume (850) might explain this discrepancy.

Regarding heart rate, our result was discordant with **Honda et al. [21]**. In 200 patients with myocardial infarction, heart rate on admission was a significant independent predictor of depressed LV function before discharge.

Regarding KILLIP class, KILLIP class II was higher in group I ($EF \leq 40\%$) ($p= 0.003$). In a meta-analysis of three angioplasty trials on 2,654 acute myocardial infarction patients conducted by **DeGeare et al. [22]**, their results were concordant with ours. They found that higher KILLIP class was associated with lower left ventricular ejection fraction ($p < 0.0001$)

Regarding need for mechanical ventilation (MV), we found significant difference between the 2 groups ($p= 0.001$) being more frequently encountered in group I. This was concordant with **Bhave et al. [6]** in which the need for mechanical ventilation was the strongest predictor of depressed LV function.

We found significant difference between the two groups regarding location of infarction ($p= 0.0002$). Anterior infarction was more frequent in group I while inferior and anterolateral infarctions were more in group II. This was concordant with **Świątkiewicz et al. [18]**. This could be explained by the fact that anterior MI patients experience more pronounced post-infarction LV remodeling and dysfunction [23].

In our study, maximum ST segment elevation was significantly higher in group I with negative correlation with EF ($r= -0.398$, $p < 0.001$). This finding was in line with a previous study **Bhave et al. [6]**. This goes with the fact that the height of ST segment elevations may be a surrogate for the size of the myocardial territory being injured [6].

Regarding ST segment resolution, complete STR $> 70\%$ was significantly higher in group II ($p < 0.001$) while partial and absent STR were higher in group I ($p < 0.001$, $p= 0.006$ respectively).

Our result was concordant with that of **Matetzky et al. [24]**. They reported 28% left ventricular dysfunction rate in patients without ST resolution versus 19 % in with ST resolution.

Our results were discordant to **Kosuge et al. [25]** in which clinical significance of ST resolution in 129 patients with anterior wall MI was evaluated and they found that ST resolution after recanalization does not consistently predict myocardial salvage in patients with anterior MI. This discrepancy may be due to their inclusion of anterior MI only which is known to have a larger area of myocardium affected and hence lower likelihood of ST resolution and improved LV function.

Regarding QRS width, QRS width was higher in-group I more than II ($p < 0.001$) with significant negative correlation with EF ($r= -0.538$, $p < 0.001$). This was concordant with **Murkofsky et al. [26]**. They showed that a prolongation of the QRS duration (> 0.10 s) on a standard 12-lead ECG was associated with lower LV EF as determined by radionuclide gated blood pool imaging. This goes with the fact that increased QRS duration is associated with more advanced remodeling in patients with CAD and poor LV function [27].

On the other hand, in the sight of the Valsartan in acute myocardial infarction (VALIANT) trial, **Yerra et al. [28]** found that a prolonged QRS duration at baseline was associated with reduced systolic function. However, following adjustment for other variables, this association lost significance.

Regarding Selvester score, it was significantly higher in group I with a strong negative association with EF ($r= -0.611$, $p < 0.001$). This result was in agreement with **Ghayal et al. [29]**. They concluded that the ECG Selvester score was a better predictor of infarct size and reduced LVEF than cardiac biomarkers in patients undergoing primary PCI for STEMI. However, this was in disagreement with **Adler et al. [30]** in which they evaluated 51 patients with first anterior wall myocardial infarction who had received reperfusion therapy and pre discharge resting technetium - 99m - sestamibi scan. Overall, the correlation between the QRS score and the myocardial perfusion defect extent ($\rho = 0.249$; $p = 0.08$) and ejection fraction ($\rho = -0.229$; $p = 0.11$) was poor.

In the present study, creatinine level was significantly higher in-group I ($p= 0.007$) with significant negative correlation with EF ($r= -0.296$, $p= 0.003$). This was consistent with a previous study [6]. In their study, a baseline creatinine level of 1.0 to 1.5 mg/dl was associated with 2.4-fold increase in the odds of having depressed LV function compared to patients whose creatinine levels were <1.0 mg/dl.

Our result was discordant with a more recent study by **Ng et al. [31]** in which creatinine showed no significant difference among 2648 STEMI patients divided in to three groups as regard LVEF ($p=0.5$). This might be explained by different modality in assessing renal impairment and different grouping as regard LVEF.

In our study, we found no significant difference between both groups regarding maximum creatine kinase (CK-MB) level. ($p=0.15$). This was in line with a study of **Grande et al. [32]** but was discordant with **Turer et al. [33]** in which correlation between peak CK-MB and both infarction size and EF was investigated. They concluded that CK-MB values have significant correlation with both of them ($r = -0.30$, $P = 0.035$). This discrepancy might be attributed to small sample size in our study compared to them and different modality in assessment of EF.

Regarding troponin level, we found no significant difference between our groups ($p=0.63$). This result was concordant with a previous study [6].

However, our results were in disagreement with that of **Chia et al. [34]** in which they found that a troponin I (Tn I) after 72 hours with threshold >55 ng/ml was 90% sensitive for large infarct size ($\geq 10\%$) and low LVEF ($\leq 40\%$) ($p=0.001$). This discrepancy might be explained by their larger sample size (378 patients), different method in assessing LV function.

We found no significant difference between the two groups regarding admission random blood sugar ($p=0.81$). This result was in concordant with a previous study [6] but was in disagreement with that of **Terlecki et al. [35]** in which there was a significant difference as regard LV function between those with normoglycemia versus patients with hyperglycemia being lower in the latter group ($p=0.0022$). This might be due to higher number of patients included in their study, their lower value of defining hyperglycemia (140 mg/dl versus 200mg/dl in our study).

Regarding dyslipidemia, we found no significant difference between the 2 groups as regard LV function (cholesterol $p= 0.35$, TG $p=$

0.19, HDL $p= 0.96$, LDL $p= 0.96$). This was concordant with previous studies [6,19].

On the other hand, in a study conducted by **Mateus et al. [17]** about impact of cardiovascular risk factors on LV function in STEMI patients, dyslipidemia was less common in patients with depressed LV function (35.4 % vs. 56.9 %, $p = 0.01$). This discrepancy might be higher percentage of dyslipidemic patients in depressed LV function group.

In our study, there was significant difference between the two groups regarding number of diseased vessels ($p < 0.001$) being higher in-group I with depressed LV function ($p < 0.001$). This result was concordant with a study of **Mateus et al. [17]** and a recent study of **Altmann et al. [36]**, this goes with the fact that increased number of affected vessels impairs perfusion of larger myocardial area and hence impair function [37].

On the contrary, our results were in disagreement with that of **Hamdan et al. [19]**. In their study, there was no significant difference regarding number of diseased vessels among STEMI patients stratified by EF. This discrepancy may be attributed to different way in stratifying patients as regard EF in to 3 groups (normal to mild, moderate and severe) with nearly comparable number of patients in all groups.

In our study, Culprit left anterior descending (LAD) was frequently encountered in group I ($EF \leq 40\%$) while culprit left circumflex (LCX) and right coronary artery (RCA) were more in group II ($EF \geq 40\%$). This result was concordant with that of **Altmann et al. [36]**, this goes with the fact that LAD supplies large myocardial area and its affection impairs perfusion of larger myocardial area and hence impair function [37].

In our study, there was significant difference between the two groups regarding Gensini score ($p < 0.001$) with strong negative correlation with EF and high statistical significance ($r = -0.53$, $p < 0.001$). This was concordant with **Jia et al. [20]**. This goes with the fact of higher Gensini score is associated with more severe coronary artery disease, lower ST segment resolution and lower LV function [38].

There was significant difference between the two groups regarding wall motion score index (WMSI) being higher in group I ($p < 0.001$) with strong negative correlation of high significance with EF ($p < 0.001$, $r = -0.99$). This was concordant with **Galasko et al. [39]** in which 174 AMI were included and they found that WMSIs of 0.6, 0.8, and 1.1 corresponded best to EFs 45%, 40%, and 35%, respectively a greater degree of wall motion

abnormality must take place before a lower EF occurs.

Our study demonstrated that decreased renal function (creatinine > 1.1 mg/dl), increased total number of diseased vessels > 2, absence of complete ST segment resolution, wall motion score index > 2.05, increased sum of ST segment elevation > 13.5 mm, QRS width > 98.5 ms, Selvester score > 3.5, Gensini score > 95, anterior location of infarction and KILLIP class > 1 are significant predictors of depressed LV function in patients presenting with STEMI.

A dichotomous prediction score created, with the goal of having high sensitivity for depressed LV function using a cutoff point 21, was able to separate patients into 2 groups: very low risk for LV dysfunction and intermediate to high risk for LV dysfunction. Using a 3-tiered score categorization, discrimination among low-, intermediate-, and high-risk patients was possible.

Of 100 patients included in our study, 53 were considered as low risk patients (score 0-14) with likelihood of 2.9, 26 were considered moderate risk (score 15-28) with likelihood ratio of 2.1 and 21 considered as high risk patients (score 29-42) with likelihood ratio of 2.4.

Study limitation:

- 1- Small sample size of our study. This could limit the generalizability of our findings.
- 2- The results obtained from a single medical center (Zagazig University hospital)
- 3- Selection bias may present because of exclusion of few patients who did not do echocardiography during their index hospitalization.
- 4- Lack of long term follow up of left ventricle function so that we could not detect possible LV function recovery with time.
- 5- Our study applied on STEMI only, excluding NSTEMI and unstable Angina.
- 6- We did not take in to consideration SYNTAX score in assessing severity of coronary artery disease.

CONCLUSION

Our findings may aid in the clinical management of patients with STEMI in the early stages of their hospitalization. Studies have shown that early beta-blockers administration, a therapy long considered the standard of care in the emergency department for STEMI, decreases the risk for arrhythmia and re-infarction but at the cost of a significant increase in the incidence of cardiogenic shock. Therefore,

A high prediction score for LV dysfunction may identify patients who are likely to have more

harm than benefit from early aggressive therapy. In contrast, patients with low prediction scores could be targeted as better candidates for such therapy.

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