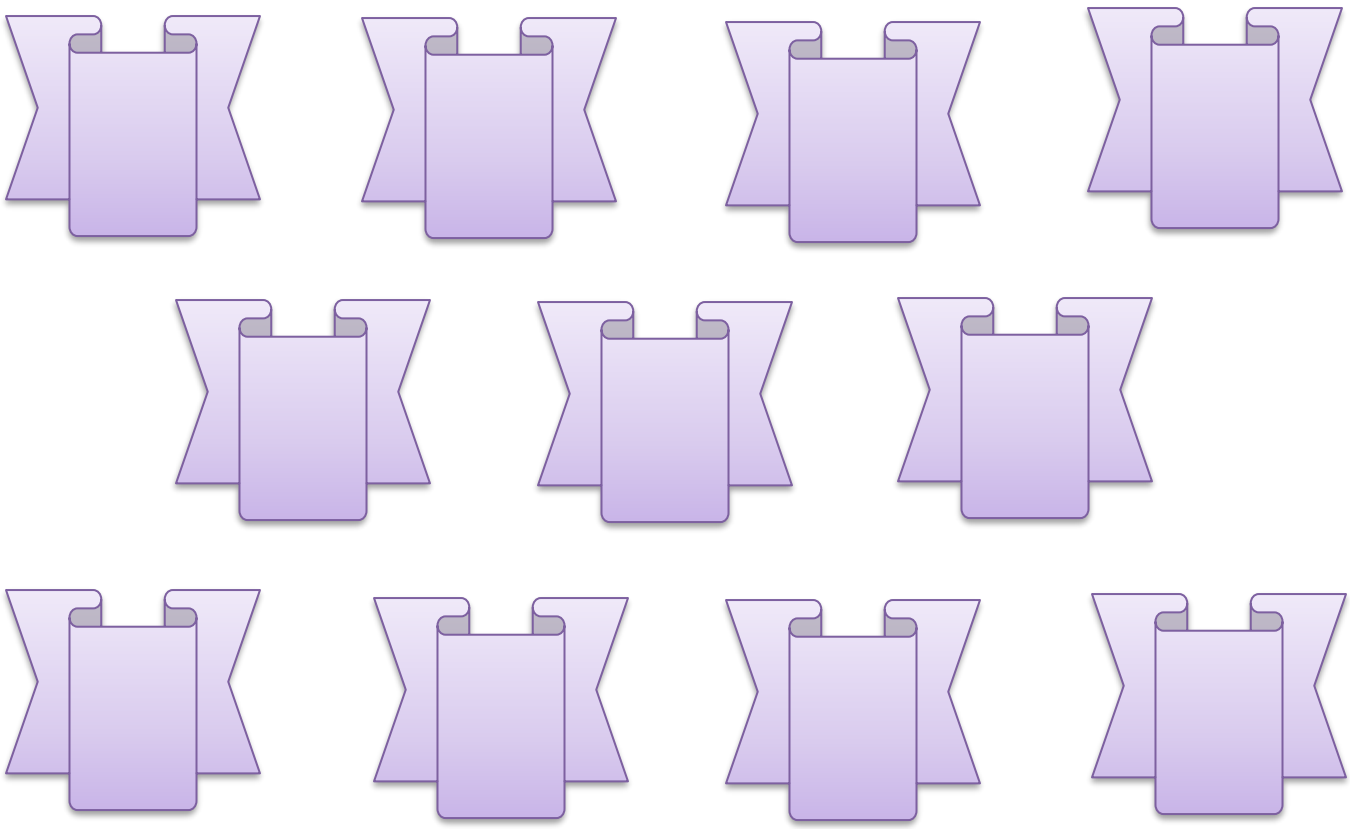
International Journal of Medical Arts

Volume 5, Issue 11, November 2023

https://ijma.journals.ekb.eg/



Print ISSN: 2636-4174

Online ISSN: 2682-3780

Available online at Journal Website
https://ijma.journals.ekb.eg/
Main Subject [Cardiology]

Original Article

Relation between Left Ventricular Wall Stress after Primary Percutaneous Coronary Intervention and Adverse Cardiovascular Events in Non-Diabetic Patients with Acute Anterior ST-Segment Elevation Myocardial Infarction

Islam Mohamed Moussa *[1], Mohamed Sayed Bashandy [1], Hamouda Abd-AlKhalek Al-Bahnasy [1], Ahmed Al-Habbaa [2]

[1] Department of Cardiology, Damietta Faculty of Medicine, Al-Azhar University, Damietta, Egypt
[2] Department of Cardiology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt

Article information

Received: 10-01-2023

Accepted: 08-10-2023

DOI: 10.21608/IJMA.2023.186447.1595.

*Corresponding author

Email: drbrahadeesh7@gmail.com

Citation: Moussa IM, Bashandy MS, Al-Bahnasy HA, Al-Habbaa A. Relation between Left Ventricular Wall Stress after Primary Percutaneous Coronary Intervention and Adverse Cardiovascular Events in Non-Diabetic Patients with Acute Anterior ST-Segment Elevation Myocardial Infarction. IJMA 2023 November; 5 [11]: 3867-3874. doi: 10.21608/IJMA.2023.186447.1595.

ABSTRACT

Background: Acute ST-segment elevation myocardial infarction [STEMI] results in left ventricular adverse remodeling [LVR]**.** Increased left ventricular wall stress [LVWS] after Myocardial infarction [MI] initiates this process. Predicting the risk of future major adverse cardiovascular events [MACE] after STEMI has been a subject of great interest. There is a lack of imaging-based data for risk stratifying post-STEMI patients' clinical outcomes at this time. As a result, improvements in echocardiography are urgently needed to identify objectively measurable echocardiographic markers for improved risk stratification.

The Aim of the work: This study aimed to study the relation between echocardiography-derived LVWS in non-diabetic patients presented with first acute anterior STEMI who underwent primary percutaneous coronary intervention with the MACE.

Patients and Methods: The current study was a prospective cohort study that took place between January 2022 and November 2022 and included 78 non-diabetic patients who presented with acute anterior STEMI treated by primary PCI. LVWS was calculated within 72 hours by pre-discharge echocardiogram using volume-based formulas. Patients were divided into two groups based on their three months follow-up data following primary PCI; Group I: MACE-negative, and Group II: MACE-positive.

Results: MACE-positive patients [n=18] had significantly higher end-systolic wall stress [ESWS] levels 94.95 ± 31.27 vs 77.20 ± 24.37 in MACE-negative patients [P value = 0.013*]. A receiver operating characteristics [ROC] curve was performed for the ESWS [KPa] as a predictor for MACE. The findings revealed an area under the curve of 0.674, the cut-off value for predicting MACE = 85.35 [KPa], with 66.97% sensitivity, 65% specificity, 86.7% Negative predictive value, 36.4 % positive predictive Value [P value = 0.026].

Conclusion: Echocardiography-based left ventricular systolic wall stress is a possibly useful prognostic tool for risk-stratifying non-diabetic STEMI patients early after MI and predicting MACE.

Keywords: Left Ventricular Wall Stress; Percutaneous Coronary Intervention; Acute Anterior ST-Segment Elevation.

INTRODUCTION

Even though early percutaneous catheter intervention [PCI] was used to try to save the heart at risk, changes in myocardial wall stress [MWS] cause ventricular dilatation and dysfunction after an acute ST-segment elevation myocardial infarction [STEMI]. Whether this is temporary or has bad effects that last for a long time [1]. Myocardial remodeling caused by MI has been implicated in approximately two-thirds of the 5 million annual cases of heart failure [HF] [2].

MWS is a parameter that is tied to the size and pressure of the ventricles and is the opposite of wall thickness. MWS can't be measured directly, but a formula based on Laplace's law can be used to estimate its number [3]. Risk stratification is an essential component of the care of patients with acute MI. Prognostic information is critical for appropriate triage and resource allocation to provide MI patients with the appropriate intensity and location of care [4].

At the moment, there aren't enough imaging-based data to help risk-stratify post-STEMI patients for clinical results, such as follow-up after they leave the hospital [1]. Because of this, there is a pressing need for improvements in echocardiography to find echocardiographic parameters that can be measured in an objective way to help with risk stratification [5].

So, this study aimed to show the relation between echocardiography-derived LVWS in non-diabetic patients presented with first acute anterior STEMI who underwent primary PCI with the MACE.

PATIENTS AND METHODS

The current study was a prospective cohort study that took place between January 2022 and November 2022 and included 78 non-diabetic patients who presented with acute anterior STEMI treated by primary PCI at Al-Azhar University Hospital in New-Damietta and El-Nasr Specialized Hospital in Port-Saeid. After getting approval from the ethics committee at Al-Azhar University and signed permission from each patient, we chose the patients based on the following criteria:

The Inclusion criteria: Patients with first acute anterior STEMI underwent primary PCI within 12 hours of chest pain onset.

Exclusion criteria: 1] History of previous ischemic heart disease [IHD] or MI in the previous 3 months. 2] Diabetes mellitus [DM]. 3] Patients with Aortic stenosis. 4] Previous cardiac surgery 3 months before presentation. 5] Patients with poor echocardiographic window.

Patients were divided into two groups based on their three months follow-up data following primary PCI; **Group I:** MACE-negative, and **Group II:** MACE-positive, LV wall stress was calculated by Echocardiography.

Data collection: All patients were subjected to a full medical history, which included a History of hypertension [HTN], DM, IHD, chronic kidney disease, dyslipidemia, and smoking. Clinical examination was done on each patient with special attention to the cardiovascular system. A standard 12 lead Electrocardiogram [ECG] for each patient was done. Routine laboratory tests were done including complete blood count, liver functions, renal functions, lipid profile, and virology.

Trans-thoracic echocardiography

All patients underwent a targeted echo-cardiographic examination within 72 hours of the initial PCI procedure. utilizing GE vivid echocardiography equipment. To rule out procedural or infarct-related complications, this study assessed residual segmental wall motion abnormalities at rest and estimated left ventricular ejection fraction [LVEF] using modified biplane Simpson's method. and calculate myocardial wall stress as the following:

[a] Systolic blood pressure was estimated to be used in wall stress calculation using a mercury column sphygmomanometer during the echo study.

[b] Patient lay in the left lateral position with an ECG cable connected during the study.

[c] Apical 5 champers view with continuous wave Doppler on the aortic valve to exclude any pressure gradient across the aortic valve.

[d] Apical 4 chamber and 2 chamber views were used to calculate LVEF % using the 2D modified Simpson's biplane method.

[e] The previous step yielded the LV volumes in systole and diastole.

[f] Pulsed-wave Doppler on the mitral valve in an apical 4 chamber view to estimate [E] wave velocity, and tissue Doppler on the mitral annulus [septal and lateral] to calculate the mean [E'], End-diastolic pressure based on Nagueh's formula and E/E' echocardiographic parameters [pulmonary capillary wedge pressure] [PCWP].

$$\mathbf{PCWP = 1.24 \times [E/E'] + 1.9.}\ ^{[6]}$$

Where E = early mitral inflow velocity by pulsed wave Doppler on mitral valve and $\boldsymbol{E'}$=mean of tissue Doppler of the mitral septal and lateral annulus.

[g] M-Mode on the parasternal view [long or short-axis view at the level of LV] was used to calculate: LVEDD: Left ventricle end-diastolic diameter in centimeters, IVSd: inter-ventricular septal thickness at diastole's end, PWd: posterior wall diameter at diastole's end.

[h] These parameters were used to calculate the LV myocardial mass by the following formula:

$$\mathbf{LV\ mass = 0.8\{1.04[([LVEDD + IVSd + PWd]^3 - LVEDD^3)]\} + 0.6}\ ^{[1]}$$

[i] LV Mass result was used to calculate myocardial volume using this equation.

$$\mathbf{Myocardial\ volume = \frac{LV\ mass}{Myocardial\ Density}}.$$

The clinically accepted value of myocardial tissue density is 1.055 g/ml [1, 7].

[j] Echocardiographically recorded volume and pressure parameters were used to determine the LVWS, which is expressed as a stress value in kiloPascals [KPa]. The following formula was previously used in clinical research and was developed from LaPlace's law by **Mirsky et al.** [8, 9]

$$\mathbf{LV\ Wall\ stress} = \frac{P}{\left[\frac{V_{lum}+V_{myo}}{V_{lum}}\right]^{2/3}-1}\ ^{[1]}$$

Coronary angiography and primary per-cutaneous coronary intervention: The following information was obtained from all patients: Culprit artery, number of diseased vessels, reperfusion success, angiographic thrombus burden, PCI-related complications recorded such as arrhythmia, tachyarrhythmia or brady-arrhythmias, acute heart failure, Coronary artery dissection, no-reflow, and cardiac arrest.

Follow-up: The patients were followed up during their hospital stay and three months after PCI for detection of heart failure, arrhythmias, reinfarction, stroke, and death.

Statistical analysis: With the aid of the IBM SPSS software package version 20.0, data were fed into the computer and evaluated. [IBM Corp, Armonk, NY]. Numbers and percentages were used to describe qualitative data. Mean and standard deviation was used to describe quantitative data. The cut-off points in a continuously distributed measurement that most accurately predicts whether a condition is present was identified using the receiver operating characteristics [ROC] curve. The sensitivity and specificity of the test in determining the diagnosis for each value of the measures are computed before ROC curves are produced.

RESULTS

Table 1 shows the demographic and clinical data of the studied patients. The mean age of our study population was 54.41 ± 10.12 years in Group I and 54.33 ± 9.57 years in Group II. Males represented 91% of our study population. In terms of the anthropometric measurements, we found no significant difference in height, weight, BMI, or BSA between the two groups [P value = 0.24, 0.24, 0.24, and 0.92 respectively]. The incidence of HTN, DM, Dyslipidemia, and positive family history was almost equal in both groups and the differences were not significant statistically [P value 0.3, 0.8, 0.4, 1 respectively]. However, the incidence of COVID-19 infection was significantly higher in group II than in group I [P value = 0.02].

As regards the Killip class, in group I, 59 patients [98.3%] presented with Killip class I, only one patient [1.7%] presented with Killip class II, and no patients were presented with either Killip class III or IV. While, in group II, 12 patients [66.7%] presented with Killip class I, 2 patients [11.1%] presented with Killip class II, only one patient [1.3%] presented with Killip class III, and 3 patients [16.7%] were presented with Killip class IV. This difference between the two groups was statistically significant [P-value <0.001].

In our study, 22 patients [36.7%] in group I had balloon angioplasty, 38 patients [63.3%] had direct stenting, while in group II, 14 patients [77.8%] had balloon angioplasty, 4 patients [22.2%], had direct stenting with statistically

significant difference between 2 groups regarding procedural technique [P-value=0.002].

In terms of thrombus burden, 12 patients [15.4%] had a heavy thrombus burden in our study population, in group I, they were 5 patients [8.3 %], while in group II, they were 7 patients [38.9%] with statistically significant difference between 2 groups regarding thrombus burden [P = 0.005]. As regards the PCI complications, it was significantly higher in group II [50%] than in group I [10%] [P =0.001] [Table 2].

We compared the Echocardiography data between the 2 groups in table [3]. and we found no statistically significant difference between the two groups [P > 0.05] except for LV volumes in diastole [ml]; in which the mean LVEDV in group I was found to be 81.83 ml ± 31.83 ml SD, which is lower than that of group II, which was 97.33 ml ± 25.65 ml SD [P-value 0.032] [table 3].

As regards the In-hospital complications; 6 patients [7.7%] had in-hospital complications, and all were in group II. In terms of length of hospital stay [LOH] 69 patients [88.5%] had short hospital stay ≤72 in our study population. In group I they were 58 patients [96.7%], while in group II, they were 11 patients [61%]. 9 patients [11.5%] had long hospital stays> 72 in our study population. In group I they were 2 patients [3.3%], while in group II, they were 7 patients [38.9. %] with a statistically high significant difference between 2 lengths of hospital stay [P = 0.001].

In our study populations the mean ESWS was 81.30 KPa ± 26.97 KPa SD. In group I, it was 77.20 KPa ± 24.37 KPa SD, while in group II it was 94.95 KPa ± 31.27 KPa SD with a statically significant difference between 2 groups regarding ESWS, [P-value 0.013*] [Figure 1].

The mean EDWS in our study population was 13.46 KPa ± 5.86 KPa SD. In group I, it was 12.88 KPa ± 5.91 KPa SD, while in group II it was 15.38 KPa ± 5.39 KPa SD with no statically significant difference between 2 groups regarding EDWS, [P-value 0.114]. A receiver operating characteristic [ROC] curve was performed for the ESWS and EDWS [KPa] as a predictor for MACE [Figure 2].

For ESWS: the findings revealed an area under the curve of 0.674, the cut-off value for predicting MACE = 85.35 [KPa], with 66.97% sensitivity, 65% specificity, 86.7% Negative predictive value, 36.4 % positive predictive Value [P value 0.026]. For EDWS: The findings revealed an area under the curve of 0.635, the cut-off value of >13.94 [KPa] with 61.11% sensitivity, 58.33% specificity, 83.3 % Negative predictive value, 30.6 % positive predictive value [P-value = 0.084] [Table 4].

Pearson's Correlation analysis was done between LVWS [ESWS and EDWS] and length of hospital stay, and we found that no statistically significant correlation between LVWS and length of hospital stay [P = 0.2, and 0.9 respectively]. Also, Pearson's Correlation analysis revealed no statistically significant correlation between LVWS and in-hospital complication [P = 0.2, and 0.7 respectively].

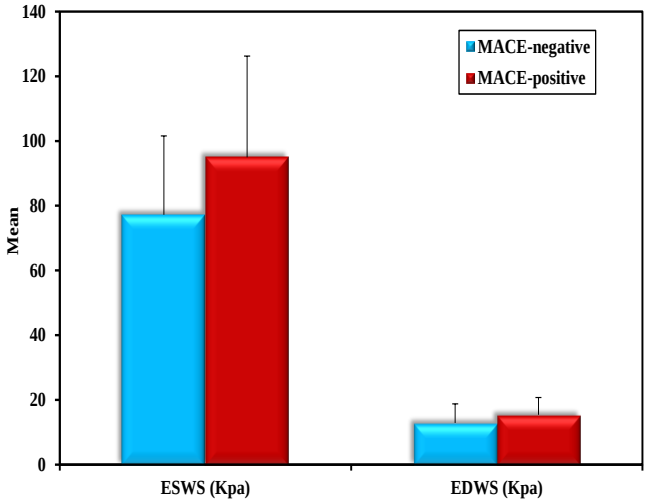


Figure [1]: Comparison between the two studied groups regarding ESWS and EDWS

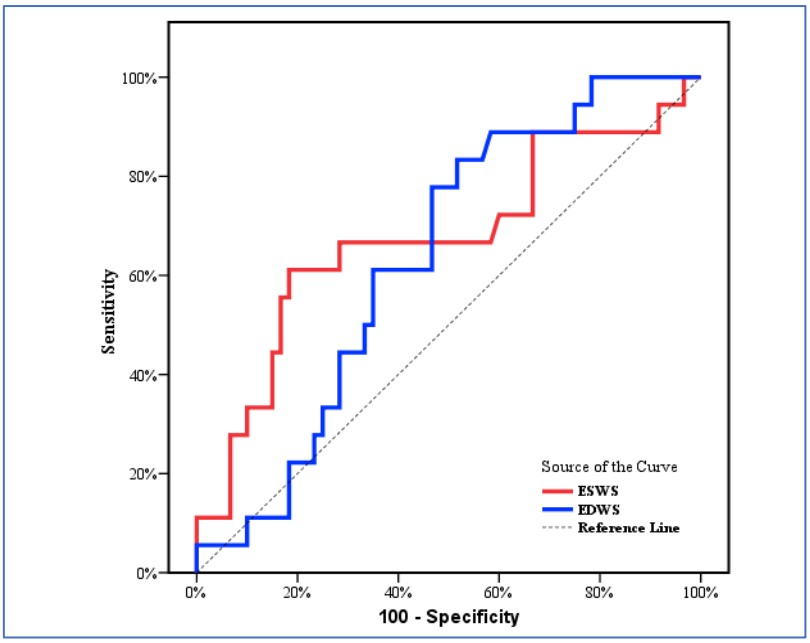


Figure [2]: ROC curve for ESWS and EDWS to predict MACE

Table [1]: Demographic and clinical characteristics of the study groups

Demographic data	Total [n = 78]	MACE-negative [n = 60]	MACE-positive [n = 18]	*P value*
Sex. N [%]				
Male	71 [91%]	56 [93.3%]	15 [83.3%]	**0.343** [a]
Female	7 [9%]	4 [6.7%]	3 [16.7%]	
Age [years]				
Min. – Max.	29.0 – 80.0	29.0 – 80.0	38.0 – 69.0	**0.971** [b]
Mean ± SD.	54.41 ± 10.12	54.43 ± 10.35	54.33 ± 9.57	
Measurements. [Mean ± SD.]				
Height [cm]	169.76 ± 7.77	170.32 ± 7.45	167.89 ± 8.71	**0.247** [b]
Weight [kg]	86.27 ± 14.39	85.97 ± 14.98	87.28 ± 12.56	**0.247** [b]
BSA [m^2]	2.01 ± 0.19	2.01 ± 0.18	2.01 ± 0.20	**0.957** [b]
BMI [kg/m^2]	29.97 ± 5.0	29.65 ± 5.06	31.07 ± 4.76	**0.293** [b]
Comorbidities and family history. N [%]				
HTN	27 [34.6%]	19 [31.7%]	8 [44.4%]	**0.318**[c]
Smoking	55 [70.5%]	42 [70.0%]	13 [72.2%]	**0.856** [c]
Dyslipidemia	10 [12.8%]	9 [15%]	1 [5.6%]	**0.438**[a]
Post covid	6 [7.7%]	2 [3.3%]	4 [22.2%]	**0.002***[a]
Family history	14 [17.9%]	11 [18.3%]	3 [16.7%]	**1** [a]
Clinical presentation				
SBP [Mean ± SD].	122.74 ± 18.07	121.35 ± 14.57	127.39 ± 26.70	**0.369** [b]
HR [Mean ± SD].	82.08 ± 15.88	81.20 ± 15.40	85.0 ± 17.54	**0.482** [b]
Killip class				
1	71 [91.0%]	59 [98.3%]	12 [66.7%]	**0.001***[c]
2	3 [3.8%]	1 [1.7%]	2 [11.1%]	
3	1 [1.3%]	0 [0.0%]	1 [5.6%]	
4	3 [3.8%]	0 [0.0%]	3 [16.7%]	
Symptoms to door [Mean ± SD]	3.87 ± 2.61	3.63 ± 2.41	4.67 ± 3.14	**0.225** [b]
Door to balloon [Mean ± SD.]	5.62 ± 2.85	5.45 ± 2.70	6.17 ± 3.33	**0.477** [b]

SD: Standard deviation**. IQR**: Inter Quartile Range. **a:** Fisher Exact test. **b:** independent t-test. **c:** Chi-Square test. ***:** Statistically significant at $p \leq 0.05$. **HTN:** Hypertension. **BSA**: Body surface area. **BMI:** Body mass index.

Table 2: Comparison between the 2 groups regarding Angiographic and primary PCI data

	Total [n = 78]	MACE-negative [n = 60]	MACE-positive [n = 18]	P value
Coronary dominance				
Right	65 [83.3%]	52 [86.7%]	13 [72.2%0	0.164 [a]
Left	13 [16.7%]	8 [13.3%]	5 [27.8%]	
Number of diseased vessels				
Single	51 [65.4%]	42 [70%]	9 [50%]	0.168 [b]
2 vessels	17 [21.8%]	13 [21.7%]	4 [22.2%]	
Multi-vessels	10 [12.8%]	5 [8.3%]	5 [27.8%]	
Procedure technique				
Balloon angioplasty	36 [46.2%]	22 [36.7%]	14 [77.8%]	**0.002* [b]**
Direct stenting	42 [53.8%]	38 [63.3%]	4 [22.2%]	
Number. of stents				
PTCA				
No stents	3 [3.8%]	3 [5%]	0 [0%]	0.437 [b]
1	56 [71.8%]	44 [73.3%]	12 [66.7%]	
2	19 [24.4%]	13 [21.7%]	6 [33.3%]	
TIMI Flow [Pre]				
0	51 [65.4%]	36 [60%]	15 [83.3%]	0.132 [b]
1	14 [17.9%]	11 [18.3%]	3 [16.7%]	
2	12 [15.4%]	12 [20%]	0 [0%]	
3	1 [1.3%]	1 [1.7%]	0 [0%]	
TIMI Flow [Post]				
0	1 [1.3%]	1 [1.7%]	0 [0%]	0.253 [b]
1	7 [9%]	4 [6.7%]	3 [16.7%]	
2	19 [24.4%]	13 [21.7%]	6 [33.3%]	
3	51 [65.4%]	42 [70%]	9 [50%]	
Thrombus burden				
No	66 [84.6%]	55 [91.7%]	11 [61.1%]	**0.005* [a]**
Yes	12 [15.4%]	5 [8.3%]	7 [38.9%]	
PCI complication				
No	63 [80.8%]	54 [90%]	9 [50%]	**0.001* [a]**
Yes	15 [19.2%]	6 [10%]	9 [50%]	

a: Fisher exact test. b: Chi square test

Table [3]: Comparison of pre-discharge echocardiogram data with 72 hours of Primary PCI between the two groups

Echocardiography	Total [n = 78]	MACE negative [n = 60]	MACE-positive [n = 18]	P value [a]
LA diameter [cm]	4.50 ± 6.31	4.85 ± 7.17	3.34 ± 0.44	0.134
Aortic root diameter [cm]	3.73 ± 4.42	3.97 ± 5.01	2.93 ± 0.58	0.177
SBP	122.74 ± 18.07	121.35 ± 14.57	127.39 ± 26.70	0.369
PCWP	13.62 ± 4.60	13.59 ± 4.92	13.71 ± 3.43	0.740
LV volume systole [ml]	53.01 ± 21.45	50.17 ± 18.32	62.50 ± 28.23	0.112
LV volume diastole [ml]	85.41 ± 31.06	81.83 ± 31.83	97.33 ± 25.65	0.032*
LVEF%	44.95 ± 10.56	45.78 ± 10.34	42.17 ± 11.10	0.204
E [m/s]	0.60 ± 0.19	0.61 ± 0.20	0.56 ± 0.15	0.290
E' [m/s]	0.07 ± 0.02	0.07 ± 0.02	0.06 ± 0.02	0.315
e' Septal [m/s]	0.06 ± 0.02	0.06 ± 0.02	0.06 ± 0.03	0.820
e' Lateral [m/s]	0.08 ± 0.03	0.08 ± 0.03	0.07 ± 0.02	0.090
LVEDD [cm]	4.92 ± 0.66	4.91 ± 0.62	4.94 ± 0.81	0.833
IVSd [cm]	1.01 ± 0.26	1.01 ± 0.28	1.01 ± 0.21	0.659
PWd [cm].	0.92 ± 0.19	0.93 ± 0.19	0.90 ± 0.17	0.552
LV Mass [g]	271.32 ± 867.11	302.66 ± 988.14	166.85 ± 42.63	0.943
Myocardial volume [ml]	258.40 ± 825.82	288.25 ± 941.08	158.90 ± 40.60	0.943

a: independent t test. **SBP**: Systolic blood pressure. **PCWP:** Pulmonary capillary wedge pressure. **LVEF:** Left ventricular ejection fraction. **E**: early mitral inflow velocity by pulsed wave Doppler on mitral valve. **E'**=mean of tissue Doppler of the mitral septal and lateral annulus. **LVEDD**: Left ventricular end-diastolic diameter. **IVSd**: Iner-ventricular septal dimension during diastole. **PWd**: Posterior wall dimension during diastole.

Table [4]: Validity [AUC, sensitivity, specificity] for ESWS and EDWS to predict MACE

	AUC	p	95% C. I	Cut off	Sensitivity	Specificity	PPV	NPV
ESWS	0.674	0.026*	0.516 – 0.831	>85.35	66.67	65.0	36.4	86.7
EDWS	0.635	0.084	0.506 – 0.764	>13.94	61.11	58.33	30.6	83.3

AUC: Area Under a Curve. **P value**: Probability value. **CI**: Confidence Intervals. **NPV**: Negative predictive value. **PPV:** Positive predictive value

DISCUSSION

This study demonstrates a positive independent association between the echocardiography-derived LVWS obtained within 72 hours after PCI and the occurrence of MACE including congestive heart failure, recurrent MI, stroke, and death. The ESWS was substantially higher in the MACE-positive group than in the MACE-negative group. On ROC curve analysis at ESWS cut-off value < 85.35 [KPa] the NPV was 86.7%, with a sensitivity of 66.67% and specify 65.0%, this was concordant with an earlier study suggested the use of LVWS as a diagnostic tool to predict a patient's prognosis following PCI.

In the study conducted by **Kattel *et al.*** [1], Patients with acute STEMI requiring urgent PCI and a high ESWS were studied in 2021. linked to a higher rate of adverse outcomes in patients who received primary PCI for STEMI. They discovered that an ESWS of over 62.5 [KPa] was linked to an 8-fold higher mortality rate and poorer outcomes compared to a lower ESWS of under 62.5 [KPa].

Another study that supports our findings was conducted by **Clerfond *et al.*** [10] on 169 patients with STIMI, Mean systolic wall stress was substantially higher in patients with HF before and after discharge, according to their calculations [P < 0.001].

In contrast to a prospective study by **Mosleh *et al.*** [5] on 81 patients presenting with STEMI and requiring primary PCI, we did not find a statistically significant association between EDWS and MACE. However, patients with high EDWS were associated with significantly more MACE outcomes [P = 0.032]. The difference between our study and the one by **Mosleh *et al.*** [5] could be attributed to the fact that our participants were not diabetic. Diastolic velocities and the flow profile via the mitral valve determine how quickly EDWS occurs. Previous studies have shown that diabetes mellitus is connected with an increased risk of diastolic heart failure and diastolic dysfunction [11, 12].

There was no correlation between LVEF% and MACE. This suggests that ESWS may be a useful feature for predicting MACE following MI. The reasoning behind this is that picture foreshortening and geographical differences in LV contractility have less of an impact on calculating ESWS than calculating LVEF [5].

The timing of the echocardiography may also explain why ESWS was a predictor of MACE in our STEMI sample whereas LVEF was not. The echocardiography was done no more than three days after the STEMI. Therefore, it may be too soon to see a drop in LVEF that will be permanent, and a fall in LVEF noticed at this stage may be attributable in part to a stunned myocardial that might recover and not be a prediction of future MACE.

Acute distension of the viable myocardium and the Frank-Starling mechanism, along with the amplification of chronotropic and inotropic activity by adrenergic receptor stimulation, may preserve the pump function despite the sudden loss of contractile tissue [13].

Conclusion: Left ventricular wall stress [LVWS] determined from echocardiography has the potential to be a valuable predictive tool for risk-stratifying Non-diabetic STEMI patients soon after MI and predicting unfavorable cardiovascular events.

Financial and non-financial relations and activities of interest: None

REFERENCES

1. Kattel S, Bhatt H, Gurung S, Karthikeyan B, Sharma UC. Elevated myocardial wall stress after percutaneous coronary intervention in acute ST elevation myocardial infraction is associated with increased mortality. Echo-cardiography. 2021 Aug;38[8]:1263-1271. doi: 10.1111/echo.15131.

2. Jenča D, Melenovský V, Stehlik J, Staněk V, Kettner J, Kautzner J, Adámková V, Wohlfahrt P. Heart failure after myocardial infarction: incidence and predictors. ESC Heart Fail. 2021;8[1]: 222-237. doi: 10.1002/ehf2.13144.

3. Uchiyama N, Yuasa T, Miyata M, Horizoe Y, Chaen H, Kubota K, *et al.* Correlation of Right Ventricular Wall Stress With Plasma B-Type Natriuretic Peptide Levels in Patients With Pulmonary Hypertension. Circ J. 2019 May 24; 83[6]:1278-1285. doi: 10.1253/circj.CJ-18-1155.

4. Smulders KRR, Demandt JPA, Vlaar PJ. Early risk assessment in patients with suspected NSTE-ACS; a retrospective cohort study. Am J Emerg Med. 2022 Oct;60:106-115. doi: 10.1016/j.ajem.2022.07.053.

5. Mosleh W, Elango K, Shah T, Chaudhari M, Gandhi S, Kattel S, *et al.* Elevated end-diastolic wall stress after acute myocardial infarction predicts adverse cardiovascular outcomes and longer hospital length of stay. Echocardiography. 2018 Nov;35[11]:1721-1728. doi: 10.1111/echo.14136.

6. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quiñones MA. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol. 1997 Nov 15;30[6]:1527-33. doi: 10.1016/s0735-1097[97]00344-6.

7. Gheorghe AG, Fuchs A, Jacobsen C, Kofoed KF, Møgelvang R, Lynnerup N, Banner J. Cardiac left ventricular myocardial tissue density, evaluated by computed tomography and autopsy. BMC Med Imaging. 2019 Apr; 19[1]:29. doi: 10.1186/s12880-019-0326-4.

8. Mirsky I. Left ventricular stresses in the intact human heart. Biophys J. 1969 Feb;9[2]: 189-208. doi: 10.1016/S0006-3495[69]86379-4.

9. Mirsky I, Parmley WW. Assessment of passive elastic stiffness for isolated heart muscle and the intact heart. Circ Res. 1973; 33[2]:233-43. doi: 10.1161/01.res.33.2.233.

10. Clerfond G, Bière L, Mateus V, Grall S, Willoteaux S, Prunier F, Furber A. End-systolic wall stress predicts post-discharge heart failure after acute myocardial infarction. Arch Cardiovasc Dis. 2015 May;108[5]:310-20. doi: 10.1016/j.acvd.2015.01.008.

11. Patil VC, Patil HV, Shah KB, Vasani JD, Shetty P. Diastolic dysfunction in asymptomatic type 2 diabetes mellitus with normal systolic function. J Cardiovasc Dis Res. 2011 Oct; 2[4]:213-22. doi: 10.4103/0975-3583.89805.

12. Ernande L, Bergerot C, Rietzschel ER, De Buyzere ML, Thibault H, Pignonblanc PG, *et al.* Diastolic dysfunction in patients with type 2 diabetes mellitus: is it really the first marker of diabetic cardiomyopathy? J Am Soc Echocardiogr. 2011 Nov;24[11]:1268-1275. e1. doi: 10.1016/j.echo.2011.07.017.

13. Alter P, Koczulla AR, Nell C, Figiel JH, Vogelmeier CF, Rominger MB. Wall stress determines systolic and diastolic function--Characteristics of heart failure. Int J Cardiol. 2016 Jan 1;202:685-93. doi: 10.1016/j.ijcard.2015.09.032.

International Journal of Medical Arts

https://ijma.journals.ekb.eg/
Print ISSN: 2636-4174
Online ISSN: 2682-3780