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ADULT CARDIOLOGY

Clinical and Biological Factors Affecting Left Ventricular End-Diastolic Pressure in ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Objective: To determine the clinical & biological factors affecting Left Ventricular End Diastolic Pressure(LVEDP) in ST-Segment Elevation Myocardial Infarction (STEMI) patients undergoing coronary angiography followed by Primary Percutaneous Coronary Intervention(PPCI).

Study Design: Quasi-Experimental study.

Place and Duration of Study: Armed Forces Institute of Cardiology/National Institute of Heart Diseases, Rawalpindi Pakistan, from Feb-Aug 2024.

Methodology: Ninety-six STEMI patients regardless of gender were enrolled through consecutive sampling. Baseline variables like Hs-Trop I, BMI, Ejection Fraction, Mean Arterial Pressure, and Killip Class were noted upon patient arrival at emergency room. LVEDP was measured using pigtail catheter during coronary-angiography and patients were classified non-randomly in two Groups: LVEDP<16mmHg (Group-A) and ≥16mmHg (Group-B). Coronary artery disease severity was assessed using the Gensini score. Clinical parameters and biological parameters were compared between Groups.

Results: Ninety-six patients [males: 66(68.8%), females: 30(31.2%)] with composite mean age 63.26±7.99 years were enrolled in study. For LVEDP, 51(53.1%) patients had values <16mmHg, while 45(46.9%) had LVEDP≥16mmHg. BMI was significantly higher in Group-B [30.00(24.00-34.00) kg/m² vs 24.00(22.00-26.00) kg/m²; p<0.001] and all the patients classified as Killip class III/IV had LVEDP≥16 mmHg (p<0.001). There was significant difference in mean values of Gensini score, EF, NT-proBNP, Hs Trop-I, MAP, TG and eGFR with comparatively higher mean values in Group-B patients except EF and eGFR (p<0.01). Correlation was significant between LVEDP and aforementioned clinical parameters except TRG (p<0.001).

Conclusion: LVEDP is strongly associated with higher BMI, advanced Killip class, and increased coronary artery severity. It also correlates with elevated cardiac biomarkers and reduced ejection fraction, identifying higher-risk individuals for adverse outcomes.

Keywords: Biological factors, Clinical factors, Left ventricular end-diastolic pressure, Primary percutaneous coronary intervention, ST-segment elevation myocardial infarction.

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INTRODUCTION

Cardiovascular diseases (CVDs) followed by coronary artery disease (CAD) and stroke remain the leading causes of death worldwide. CVDs accounted for 29.1% of reported deaths in Pakistan. Ischemic heart disease was the leading cause of premature death, ranking second overall. According to WHO data, 19% of deaths in Pakistan's lower-middle-income group were due to CVDs, with a higher mortality rate observed in females compared to males (age-standardized death rate per 100,000).[1,2] Acute coronary occlusion in CAD can lead to serious complications, including myocardial infarction (MI), heart failure (HF), arrhythmias, and sudden cardiac death.[3] Post-infarct remodeling, a key contributor to HF development, affects about 30% of patients following anterior wall MI and 17% after non-anterior wall MI.[4]

ST-segment elevation myocardial infarction (STEMI), a severe form of MI, has profound effects on both systolic and diastolic cardiac function. Following coronary occlusion, LVEDP begins to increase within 10-20 seconds, leading to myocardial wall motion abnormalities, decreased ejection fraction, and ischemic symptoms. However, the presentation of these events can vary based on collateral flow and ischemic preconditioning.[5]

LVEDP is widely recognized as a key marker of LV dysfunction and a strong predictor of adverse outcomes in patients with ischemic heart disease (IHD).[6,7] Cardiac catheterization remains the most reliable technique for assessing LVEDP and other hemodynamic parameters.[8] Given its prognostic value,

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it is crucial to explore factors that influence LVEDP in patients with STEMI, as this could improve outcome predictions and guide clinical management.

In STEMI patients, LVEDP is associated with LV function, heart failure, and mortality.[9] Identifying modifiable clinical parameters that contribute to elevated LVEDP in these patients may help in targeting interventions to reduce LVEDP and, consequently, improve patient outcomes. This study aims to identify the clinical and biological factors influencing LVEDP in patients with STEMI undergoing primary percutaneous coronary intervention (PPCI).

METHODOLOGY

This Quasi Experimental study, was carried out from February-August 2024, after approval from Institutional Ethical Review Board (IERB) at Armed Forces Institute of Cardiology & National Institute of Heart Disease, Rawalpindi (IERB Ltr# 9/2/R&D/2024/304, Dated: 15th Feb,2024).

Sample size of 26 in each group was calculated by using WHO sample size calculator with 90% power of study, 5% margin of error and proportion of 21.8% and 71.8% patients in LVEDP<16 mmHg and LVEDP≥16 mmHg group, respectively.[5] However, we collected data from 96 patients.

Inclusion Criteria: STEMI patients who underwent coronary angiography followed by PPCI regardless of gender and age ranged between 20-75 years were included in the study.

Exclusion Criteria: Patients with known contrast allergy, acute renal failure, known cardiomyopathies, STEMI patients who had onset of symptoms >12 hours and at the time of presentation were asymptomatic and those who died immediately after arrival at hospital or during PPCI procedure were excluded from this study.

STEMI patients were diagnosed on the basis of 4th universal definition of MI,[10] and after written consent; demographic data was collected at the time of admission by employing consecutive sampling technique with non-random allocation of study participants. EF was estimated on transthoracic echocardiography at emergency room on Philips iE33 machine by Simpson's biplane method. Before PPCI, LVEDP was measured by using a Pigtail catheter advanced into the LV left ventricular cavity during coronary angiography. The pressure readings just prior to the peak of R-wave on the surface ECG at monitor were recorded as LVEDP. Based on LVEDP pressures, patients were further divided in to Group-A having LVEDP<16mmHg and Group-B with LVEDP≥16mmHg. Then, all the patients underwent Primary PCI with drug eluting stent.

According to the 1998 European Society of Cardiology report and its 2007 revision, an LVEDP greater than 16mmHg is indicative of LV diastolic dysfunction. As a result, this study used a cutoff value of 16mmHg for LVEDP, measured via left heart catheterization, to examine the factors linked to elevated LVEDP.[5] Severity of CAD was assessed by Gensini score,[11] under direct supervision of senior cath laboratory consultant.

Laboratory measurements: Blood samples for routine baseline investigations were taken before coronary angiography and high sensitive troponin-I (measured at 1, 3, 24, 32 hours) with peak value was also noted. Other complete blood picture (CBC), renal and liver function tests were performed. A separate fasting blood sample was taken to measure blood glucose, triglycerides, LDL, HDL, total cholesterol, uric acid, NT-Pro BNP, and estimated glomerular filtration rate (eGFR) was calculated by using CKD-EPI method.

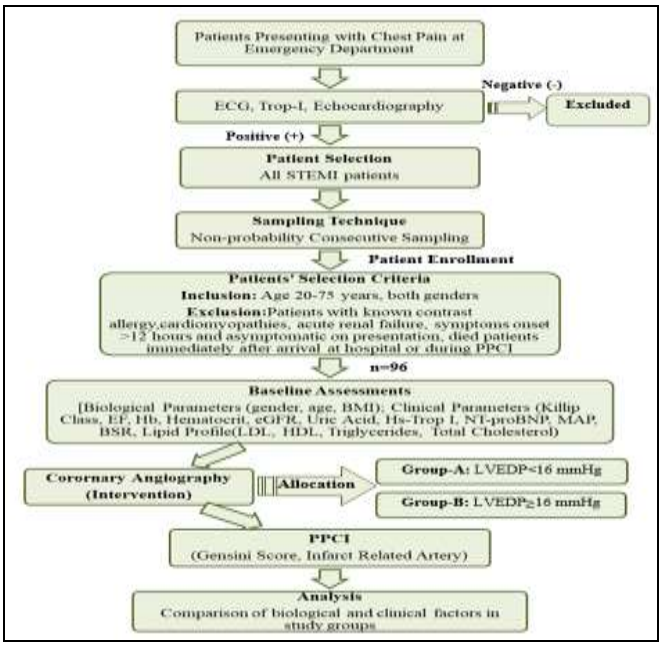

Figure: Patient Selection Flow chart for Quasi-experimental Study

Frequencies & percentages and mean & standard deviations were calculated for categorical and continuous data respectively. Data normality was checked by Kolmogorov-Smirnov test. Chi square test

and independent sample t-test/Mann Whitney-U test were applied to compare the frequency and mean/median difference between study Groups respectively. Pearson/Spearman correlation was used to find the correlation between LVEDP and clinical parameters. Findings were considered significant at *p*-value≤0.05.

RESULTS

Out of the 96 patients enrolled in this study, 66(68.8%) were males, and 30(31.2%) were females and composite median age was 64.00(59.00-69.00) years. The median BMI was slightly higher than normal 25.00 kg/m²). Among clinical parameters, the median GENSINI score was 38.00(32.00-50.00) and ejection fraction was 50.00(40.00-55.00)%. For LVEDP, 51(53.1%) patients had values <16 mmHg, while 45(46.9%) had values >16mmHg. Majority of the patients belonged to Killip Class-I 49(51.0%) and LAD was the commonest infarct related artery followed by RCA [46(47.9%) and 34(35.4%) respectively]. (Table-I)

Table-II shows the association of LVEDP Groups with biological and clinical parameters of study participants. It can be noted that the mean BMI was significantly lower in LVEDP<16mmHg-Group (22.00(24.00-26.00)kg/m² vs 30.00(24.00-34.00) kg/m²; *p*<0.001) and all the patients who belonged to Killip class III/IV had LVEDP≥16 mmHg with significant *p*-value (*p*<0.001). There is a statistically significant difference in median values of Gensini score, EF, NT-proBNP, Hs-Trop I, MAP, TG and eGFR with comparatively higher median values in patients having LVEDP≥16 mmHg except EF and eGFR (*p*<0.001). Other parameters like BSR, LDL, HDL, TC, HCT, Hb, and uric acid showed no significant differences between the Groups (*p*>0.05). However LVEDP≥16 mmHg-Group showed lower median values except LDL and mean uric acid.

Table-III findings reveal significant correlations between LVEDP and several key clinical parameters, including Gensini score, EF, NT-proBNP, Hs Trop I, MAP, and eGFR, all of which demonstrate strong associations with LVEDP (*p*<0.001). These findings

Table-I: Biological and Clinical Parameters of Study Sample (n=96)

Variables		Frequency (%)
Biological Parameters		
Gender	Male	66(68.8)
	Female	30(31.2)
Age(years) Median(IQR)		64.00(59.00-69.00)
Body mass index(kg/m2) Median(IQR)		25.00(23.00-25.00)
Clinical Parameters		
LVEDP	<16mmHg	51(53.1)
	≥16mmHg	45(46.9)
Killips Class	I	49(51.0)
	II	21(21.9)
	III	17(17.7)
	IV	9(9.4)
Infarct Related Artery	LAD	46(47.9)
	LCx	14(14.6)
	RCA	34(35.4)
	LMS	2(2.1)
		Median(IQR)/(Mean±SD)
Gensini score		38.00(32.00-50.00)
Ejection Fraction(%)		50.00(40.00-55.00)
NT-proBNP(pg/ml)		400.50(231.00-6735.00)
High Sensitive Troponin I(ng/L)		4450.00(2876.75-10747.50)
Mean arterial pressure(mmHg)		88.00(76.00-103.75)
Blood Sugar Random(mg/dl)		119.50(90.00-200.00)
Low Density Lipoprotein (mg/dl)		111.00(100.00-128.00)
Triglycerides(mg/dl)		237.50(21.00-321.00)
High Density Lipoprotein(mg/dl)		39.00(37.00-40.00)
Total Cholesterol(mg/dl)		208.00(193.00-249.00)
Hematocrit(%)		41.00(39.00-43.00)
Hemoglobin (g/dl)		13.00(12.02-14.00)
eGFR (ml/min/1.73m²)		73.00(67.25-77.75)
Uric acid (mg/dl)		6.64±0.99

NT-proBNP=N-terminal pro B-type Natriuretic Peptide,eGFR=estimated Glomerular Filtration Rate, LVEDP= Left Ventricular End Diastolic Pressure, LMS=Left Main Stem, LAD=Left Anterior Descending, LCx=Left Circumflex, RCA=Right Coronary Artery

suggest that higher LVEDP is closely linked to more severe CAD, reduced cardiac function, myocardial injury, higher blood pressure, and impaired kidney function. Other parameters, such as lipid levels, HCT, Hb and uric acid, showed weak and insignificant correlations with LVEDP, indicating a lesser impact on this cardiac measure (p>0.05).

Russo *et al.*,[12] and Lumori *et al.*[13] showed that higher BMI worsened LV diastolic function, independent of LV mass and other risk factors. Their study demonstrated that increasing BMI worsens LV diastolic function. Russo *et a*l.[12] statistics reported a decrease in E/A ratio from 0.87 (normal-weight) to 0.81 (obese), a reduction in E′ velocity from 7.5cm/s - 7.0cm/s, and an increase in the E/E′ ratio from 9.9-11.1 (p<0.01), indicating higher LV filling pressures in obese individuals while, Lumori *et al.*[13] reported 1.12 aOR of BMI for LVEDP. Similarly, our study found significantly higher BMI in patients with elevated LVEDP [30.00(24.00-34.00) vs 24.00(22.00-26.00) kg/m², p<0.001]. This may help explain the heightened risk of heart failure seen in individuals who are overweight or obese, as both conditions contribute to diastolic dysfunction, increasing the likelihood of heart failure. A study conducted in Pakistan by Suleman *et al.*[14] documented insignificant association between BMI and LVEDP which may be due to difference in study settings, study design variations,

Table-II: Association of LVEDP-Groups with Biological and Clinical Parameters of Study Sample (n=96)

Variables		LVEDP <16 mmHg (Total=51)	LVEDP ≥16 mmHg (Total=45)	*p*-value
Biological parameters				
Gender Frequency(%)	Male	37(72.5)	29(64.4)	0.53
	Female	14(27.5)	16(35.6)	
Age(years) Median(IQR)		65(60.00-69.00)	63.00(56.00-70.00)	0.54
Body mass index(kg/m2) Median(IQR)		24.00(22.00-26.00)	30.00(24.00-34.00)	<0.001
Clinical parameters				
Killip class Frequency(%)	I	45(88.2)	4(8.9)	<0.001
	II	6(11.8)	15(33.3)	
	III	-	17(37.8)	
	IV	-	9(20.0)	
Infarct Related Artery Frequency(%)	LAD	10(19.6)	36(80.0)	<0.001
	LCx	11(21.6)	3(6.7)	
	RCA	30(58.8)	4(8.9)	
	LMS	-	2(4.4)	
Median(IQR)/Mean±SD				
Gensini score		32.00(28.00-38.00)	60.00(39.00-89.00)	<0.001
Ejection Fraction(%)		55.00(50.00-56.00)	40.00(35.00-48.00)	<0.001
NT-proBNP(pg/ml)		248.00(189.00-341.00)	6740.00(1902.00-100000.00)	<0.001
High sensitive Troponin I(ng/L)		3417.00(2000.00-4500.00)	12000.00(5000.00-15383.00)	<0.001
Mean arterial pressure(mmHg)		79.00(73.00-89.00)	100.00(89.00-112.00)	<0.001
Blood sugar random(mg/dl)		149.00(90.00-202.00)	110.00(89.00-178.00)	0.19
Low density lipoprotein(mg/dl)		110.00(97.00-124.00)	117.00(103.00-134.00)	0.07
High density lipoprotein (mg/dl)		39.00(38.00-40.00)	38.00(37.00-40.00)	0.17
Triglycerides(mg/dl)		234.00(201.00-279.00)	268.00(216.00-379.00)	0.018
Total cholesterol(mg/dl)		209.00(193.00-245.00)	204.00(193.00-258.00)	0.75
Hematocrit(%)		41.00(39.00-44.00)	40.00(39.00-43.00)	0.29
Hemoglobin(g/dl)		13.00(12.30-14.50)	13.00(12.00-14.00)	0.30
eGFR(ml/min/1.73m²)		76.00(72.00-79.00)	69.00(62.00-73.00)	<0.001
Uric acid(mg/dl)		6.59±0.81	6.70±1.17	0.62

NT-proBNP=N-terminal pro B-type Natriuretic Peptide; eGFR=Estimated Glomerular Filtration Rate, LMS=Left Main Stem, LAD=Left Anterior Descending Artery, LCx=Left Circumflex Artery, RCA=Right Coronary Artery

DISCUSSION

This study highlights key factors affecting LVEDP in STEMI patients, including BMI, Killip class, Gensini score, EF, NT-proBNP, Hs Trop-I, triglycerides, MAP, and eGFR. It demonstrates a close association between LVEDP and severe CAD, reduced cardiac function, myocardial injury, elevated blood pressure, and impaired kidney function. The study found that a higher percentage of patients in Group-B (LVEDP>16 mmHg) had LAD as the culprit vessel compared to Group-A. Therefore, LVEDP is crucial for evaluating heart filling during diastole and assessing the severity of heart dysfunction.

or other confounding factors, highlighting the complexity of this relationship. Regarding gender, both studies indicated no significant difference. This suggests that the effects of BMI on LVEDP are independent of gender. This highlights that BMI, rather than gender, may play a more clinically significant role in influencing LV diastolic dysfunction.

Table-III: Correlation Between Clinical Parameters and LVEDP (n=96)

Clinical Parameters	Correlation Coefficient (r)	*p*-value
Gensini Score	0.632	<0.001
EF(%)	-0.784	<0.001
NT-proBNP(pg/ml)	0.685	<0.001
Hs Trop-I(ng/L)	0.600	<0.001
MAP(mmHg)	0.449	<0.001
eGFR(ml/min/1.73m²)	-0.529	<0.001
BSR(mg/dl)	-0.227	0.026
LDL(mg/dl)	0.134	0.192
TG(mg/dl)	0.161	0.117
HDL(mg/dl)	-0.134	0.194
TC(mg/dl)	-0.004	0.969
HCT(%)	-0.100	0.332
Hb(g/dl)	-0.060	0.565
Uric Acid(mg/dl)	0.076	0.464

EF=Ejection Fraction; NT-proBNP=N terminal-Pro BNP; Hs Trop-I=High sensitive troponin-I; MAP=Mean Arterial Pressure; BSR=Blood Sugar Random; LDL=Low-Density Lipoprotein; HDL=High-Density Lipoprotein; TG=Triglycerides; TC=Total Cholesterol; HCT=Hematocrit; Hb=Hemoglobin; eGFR=Estimated Glomerular Filtration Rate

Our study reported significant association of LVEDP and Killip classification, which measures heart failure severity. Specifically, patients with elevated LVEDP were predominantly classified in Killip Class-II and higher, indicating more severe heart failure. In contrast, those with lower LVEDP levels were primarily in Killip Class-I, reflecting better cardiac function. This aligns with previous research conducted by Zhou *et al*.[5] that reported 30.8% of patients with LVEDP≥16 mmHg in Killip Class-II or higher, compared to only 20.7% in the lower LVEDP-Group (p=0.022). These findings can help clinicians accurately identify patients at higher risk for adverse outcomes based on their LVEDP readings. For instance, recognizing patients with elevated LVEDP may prompt earlier intervention strategies, such as optimizing diuresis or adjusting heart failure management protocols.

For infarct related artery, the work of Ndrepepa *et al*.[15] is comparable where in a cohort with LVEDP>19mmHg, 50.1% of patients had infarcts in LAD, while 31.1% were related to RCA, and 16.6% involved the LCx. Conversely, in patients with LVEDP<19mmHg, RCA was the predominant infarct-related artery, accounting for 44.2%, followed by LAD at 36.3% and LCx at 17.7%. These results demonstrate that LAD-related infarcts tend to cause extensive myocardial damage and cardiac strain. Similar to this, current study infarctions involving LAD were more prevalent in patients with LVEDP≥16 mmHg, whereas those with lower LVEDP (<16mmHg) were more likely to have infarctions in RCA. This signifies that LAD supplies a significant portion of the LV; occlusions in this artery can lead to extensive myocardial damage, likely explaining the higher LVEDP observed in these patients.

Additionally, existing study patients with elevated LVEDP exhibited higher levels of biomarkers such as Hs-Trop I and NT-proBNP, which are indicators of cardiac injury and stress. This correlation suggests that elevated LVEDP is associated with more extensive myocardial damage. Relationship of LVEDP with infarct location and biomarker levels emphasizes the importance of early identification and management of patients with elevated LVEDP. Our findings are consistent with existing literature that links higher levels of cardiac biomarkers to poorer outcomes in heart disease.[16] This suggests that monitoring LVEDP and related biomarkers could serve as vital tools for assessing the extent of cardiac muscle strain and guiding treatment strategies in STEMI patients.

A Chinese study reported EF lower in patients with high LVEDP, measuring 57.43±7.53% in comparison to 61.28±5.78% in patients with LVEDP<16 mmHg (p<0.001).[5] Similarly, our study found a more pronounced reduction in EF among patients with LVEDP≥16 mmHg compared to lower LVEDP [40.00(35.00-48.00)% vs 55.00(50.00-56.00%]; p<0.001) respectively. The substantial drop in EF reflects the severity of LV dysfunction but also features prognostic implications of these measures. Clinicians should prioritize close monitoring of LVEDP and EF in STEMI patients, as these parameters can inform treatment decisions and ultimately improve patient outcomes.

MAP was significantly higher in our patients with elevated LVEDP. This elevated afterload places additional strain on the heart, particularly during MI. Patients with LVEDP≥16 mmHg had a median MAP of 100.00 mmHg, compared to 79.00mmHg in those with LVEDP<16 mmHg (p<0.001). Burak *et al*.[17] reported elevated systolic (164±26mmHg) and

diastolic (91±14mmHg) pressures in patients with high LVEDP, reinforcing our findings. This critical link between elevated MAP and LVEDP, underscores the need for effective blood pressure management in STEMI patients to reduce cardiac strain, thereby, close monitoring and control of BP are essential for optimizing cardiac performance in this high-risk population.

Additionally, Gensini score was strongly correlated with LVEDP. This suggests that as CAD worsens, LVEDP increases, reflecting greater heart dysfunction.[16] Similarly, Solangi *et al*.[19] demonstrated association of higher LVEDP with severe CAD, where mean LVEDP increasing from 18.5±5.6mmHg in single-vessel disease (SVD) to 21.4±7.2mmHg in three-vessel disease (3VD). The proportion of 3VD also rose from 15.5% at LVEDP ≤15 mmHg to 36% at LVEDP>25 mmHg, indicating that higher LVEDP is linked to a greater CAD burden. Parallel to this, our study also demonstrated a positive relationship between LVEDP and Gensini score, and a negative relationship with EF, consistent with Suleman *et al*.[14] who reported a lower mean EF in patients with LVEDP >20 mmHg (58.7±6.9% vs 61.2±4.3%). Additionally, higher NT-proBNP levels in the elevated LVEDP-Group, from 45.9±17.9 pg/ml-700.6±12.5 pg/ml, further highlight its significant influence. Elevated NT-proBNP levels reflect the heart's compensatory response to increased pressure and strain on the ventricle by promoting diuresis and vasodilation, making NT-pro BNP a valuable diagnostic and prognostic marker in patients with elevated LV filling pressures.[19]

Overall, our study highlights the critical role of LVEDP in predicting cardiac function and outcomes in STEMI patients. Significant associations between LVEDP and clinical parameters such as Gensini score, EF, NT-proBNP, Hs Trop-I, and MAP reflect the severity of myocardial injury and heart dysfunction. These findings reinforce the prognostic value of LVEDP as a marker of disease severity and cardiac strain, aligning with existing literature and emphasizing its potential as a guide for therapeutic interventions.

LIMITATIONS OF STUDY

The study was limited to STEMI patients, leaving the status of LVEDP in NSTEMI and unstable angina patients unexplored. Additionally, LVEDP was measured only during the index procedure, without post-PPCI follow-up or data on the long-term status of patients with elevated LVEDP. This lack of follow-up leaves gaps in understanding the progression of patients with initially elevated LVEDP.

CONCLUSION

In patients undergoing PPCI for STEMI, elevated LVEDP (≥16mmHg) is significantly associated with higher BMI, advanced Killip class, and more severe CAD. Additionally, elevated LVEDP correlates with increased levels of cardiac biomarkers (Hs Trop-I, NT-proBNP), higher MAP, and worse myocardial function (lower EF). These findings suggest that clinical and biological factors, such as increased BMI and markers of myocardial stress, can predict elevated LVEDP, helping identify patients at higher risk for adverse outcomes following PCI.

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Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

MWS & MBS: Data acquisition, data analysis, critical review, approval of the final version to be published.

SF & IA: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

RH & MMKS: Study concept, data acquisition, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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