ORIGINAL ARTICLE

LEFT VENTRICULAR END-DIASTOLIC PRESSURE AND EXTENT OF CORONARY ARTERY DISEASE IN PATIENTS UNDERGOING PRIMARY PERCUTANEOUS CORONARY INTERVENTION

Bashir Ahmed Solangi[1], Khalil Ahmed Shaikh[1], Jehangir Ali Shah[1], Rajesh Kumar[1], Kamran Ahmed Khan[1], Mahesh Kumar Batra[1], Ambreen Nisar[1], Tahir Saghir[1], Nadeem Qamar[1]

[1]National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan

Objectives: This study aimed to assess the association of left ventricular end-diastolic pressure (LVEDP) with the extent and severity of coronary artery diseases (CAD) in individuals undergoing primary percutaneous coronary intervention (PCI) at a tertiary care cardiac center in Karachi, Pakistan.

Methodology: This descriptive cross-sectional study included consecutive patients undergoing primary PCI. LVEDP was assessed with the help of a multipurpose catheter. The Association of LVEDP with the extent and severity of CAD was assessed.

Results: LVEDP was stratified as ≤15 mmHg, 15-25 mmHg, and >25 mmHg. Out of 498 patients included in this study, 76.3% (380) were male, and mean age was 53.7±11.7 years. Mean LVEDP was 19.35±6.17 mmHg. Burden of diseases was found to be significantly associated with LVEDP level ($p<0.001$) with mean LVEDP of 18.5±5.6 mmHg, 19.5±6 mmHg, and 21.4±7.2 mmHg among patients with single, two and three-vessel disease respectively. Proportion of three-vessel diseases was 15.5% (37/239), 22.5% (47/209), and 36% (18/50) at LVEDP ≤15 mmHg, 15-25 mmHg, and >25 mmHg, respectively.

Conclusion: There was a strong inverse relationship between LVEDP and initial TIMI flow grade ($p=0.013$) and a positive relationship between LVEDP and total length of the lesion ($p=0.002$). In conclusion, increased LVEDP was found to be associated with increased burden and extent of coronary artery disease, poor initial TIMI flow grade, and longer length of lesion.

Keywords: STEMI, primary PCI, LVEDP, extent and severity of CAD

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INTRODUCTION

Usually, epicardial blood flow is normally reinstated in the infarct-related artery after primary percutaneous coronary intervention (PCI).[1] Microvascular injury caused by ST-segment–elevation myocardial infarction (STEMI) is frequently overlooked in day-to-day clinical practice, yet it increases the risk of mortality and cardiac failure.[2] Though cardiovascular magnetic resonance (CMR) imaging can detect microvascular injury after STEMI, it is impractical in the acute setting, and so is not effective for guiding clinical decision-making and pharmacological management.[3] In individuals with STEMI, the left ventricular end-diastolic pressure (LVEDP) is an important hemodynamics marker that has been demonstrated to be strongly associated with heart failure and death.[4, 5] It is feasible and can be easily measured during primary PCI. However, LVEDP is often omitted in routine clinical practice.[3]

There is a multifaceted pathophysiological mechanism behind the relationship between adverse events and LVEDP levels. It could be used as a surrogate measure for a high ischemia burden linked to an increased risk of immediate adverse events following acute coronary syndrome (ACS).[5] Even after successful PCI and flow restoration, impaired myocardial perfusion and coronary blood flow are associated with elevated left ventricular (LV) filling pressure.[6] Reduced oxygen delivery and decreased microvascular flow may have an adverse impact on infarct size and LV remodeling during the follow-up period after PCI.[7] Congestive heart failure has also been linked to an increased risk of procedure-related complications such as bleeding and contrast-induced nephropathy that adds to the

increased burden of morbidity and death in ACS patients. Neurohormonal and sympathetic activation can also be resultants of increased wall stress and LV filling pressures.[8,9] Even with a minor MI, increased LVEDP can be a reflection of an underlying cardiovascular disease. In acute MI, pre-existing hypertrophy or myocardial fibrosis can also lead to the progression and development of diastolic dysfunction.[5]

Several studies have established the prognostic role of LVEDP in acute MI settings,[4-6,8] however, only limited studies have explored the relationship of LVEDP with the severity and extent of coronary artery diseases.[8,10] Therefore, the objective of this study was to assess the association of LVEDP with extent and severity of CAD in individuals undergoing primary PCI at a tertiary care cardiac center of Karachi, Pakistan.

METHODOLOGY

This descriptive cross-sectional study was conducted between June 2021 and December 2021 at the National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan. Informed consent was obtained from all the patients included in this study and approved by the ethical review board of the institution. In this study, we included consecutive patients who had undergone primary PCI for STEMI. Inclusion criteria were adult patients (age ≥ 18 years) of either gender presented at the emergency department within a 12-hour window period after typical chest pain. Exclusion criteria for the study were late presentation (> 12 hours after onset of symptoms), those with a history of cardiac surgery, who received conservative therapy/ thrombolytic agents, or procedures where the operator did not assess LVEDP for any reason. All the primary PCI procedures were performed as per the institutional protocol with the recommended pre- and post-procedure antiplatelet and anticoagulation therapies. LVEDP was assessed with the help of a multipurpose catheter.

Collected data consisted of demographic characteristics (gender and age), presenting characteristics (door to balloon time, total ischemic time, Killip class, hemodynamics), type of MI, risk profile (hypertension, diabetes, obesity, smoking, family history of CAD), and angiographic findings (number of diseased vessels, significant left main diseases, infarct-related artery, percentage stenosis in culprit artery, initial Thrombolysis in Myocardial Infarction (TIMI) flow grade), total length of lesion, and vessel diameter). Along with these, LVEDP was also assessed.

IBM SPSS version 21 was used for the analysis of data. LVEDP levels were stratified as ≤15 mmHg (normal), 15-25 mmHg (elevated), and >25 mmHg (severely elevated). Data were summarized as frequency (%) or mean ± standard deviation (SD) and compared among the three groups by conducting a Chi-square test/Likelihood ratio test or One-way analysis of variance (ANOVA) with p-value ≤ 0.05 as criteria for statistical significance.

RESULTS

The demographic distribution of 498 patients included in this study is as follows: 76.3% (380) were male, and mean age was 53.7±11.7 years. Mean LVEDP was 19.35±6.17 mmHg. More than half (55.4%) of the patients had single vessel disease (SVD), 24.1% had two-vessel disease (2VD), and the remaining 20.5% of the patients had three-vessel disease (3VD). Burden of diseases was found to be significantly associated with LVEDP level ($p<0.001$) with mean LVEDP of 18.5 ± 5.6 mmHg among patients with SVD, 19.5 ± 6 mmHg among patients with 2VD, and 21.4 ± 7.2 mmHg among patients with 3VD. The distribution of clinical and demographic characteristics of patients with LVEDP are presented in Table 1.

Distribution of LVEDP was ≤15 mmHg in 48%, 42% had LVEDP between 15 and 25 mmHg, while 10% had LVEDP > 25 mmHg. Distribution of LVEDP level was found to be associated with the burden of diseases ($p=0.005$), with 15.5% (37/239) having 3VD at LVEDP ≤15 mmHg, 22.5% (47/209) with 3VD at LVEDP between 15-25 mmHg, and 36% (18/50) with 3VD at LVEDP > 25 mmHg. Similarly, there was a strong inverse relationship between LVEDP and initial TIMI flow grade ($p=0.013$). The distribution of culprit left anterior descending artery (LVEDP) had a strong positive association, while the distribution of culprit right coronary artery (RCA) and left circumflex had an inverse relationship with LVEDP level ($p<0.001$). The distribution of angiographic findings stratified by left ventricular end-diastolic pressure (LVEDP) are presented in Table 2 and Figure 1.

Table 1: Distribution of clinical and demographic characteristics of patients stratified by disease burden

Characteristics	Disease Burden			P-value
	Single Vessel	Two Vessel	Three Vessel	
Total (N)	**276 (55.4%)**	**120 (24.1%)**	**102 (20.5%)**	-
Gender				
Male	75.4% (208)	75% (90)	80.4% (82)	0.551a
Female	24.6% (68)	25% (30)	19.6% (20)	
Age (years)	52.35 ± 12.72	55.09 ± 9.5	55.89 ± 10.81	0.014c
Door to balloon time (minutes)	56.17 ± 23.33	60.39 ± 25.17	62.79 ± 31.37	0.056c
Total ischemic time (hours)	6.52 ± 2.16	6.65 ± 1.98	7 ± 2.25	0.152c
Heart Rate (bpm)	82.2 ± 18.1	90.9 ± 17.2	86.7 ± 20.6	<0.001c
Systolic blood pressure (mmHg)	119 ± 18.5	121.5 ± 20	117.9 ± 18.8	0.318c
Diastolic blood pressure (mmHg)	75 ± 10.2	76.1 ± 10.7	74.2 ± 11.1	0.391c
Killip Class				
I	81.2% (224)	75.8% (91)	74.5% (76)	0.498b
II	15.2% (42)	19.2% (23)	17.6% (18)	
III	1.8% (5)	2.5% (3)	2% (2)	
IV	1.8% (5)	2.5% (3)	5.9% (6)	
Type of myocardial infarction (MI)				
Anterior	54.7% (151)	55.8% (67)	51% (52)	0.394b
Inferio-posterior	3.3% (9)	9.2% (11)	6.9% (7)	
Inferior	23.2% (64)	23.3% (28)	21.6% (22)	
Inferior plus RV infarction	9.8% (27)	6.7% (8)	11.8% (12)	
Isolated posterior	5.4% (15)	3.3% (4)	6.9% (7)	
Lateral	3.6% (10)	1.7% (2)	2% (2)	
Mechanical ventilation	1.8% (5)	4.2% (5)	7.8% (8)	0.027b
Co-morbid conditions				
Hypertension	62.7% (173)	70% (84)	81.4% (83)	0.002a
Diabetes mellitus	38.8% (107)	50.8% (61)	49% (50)	0.041a
Current smoker	27.2% (75)	36.7% (44)	32.4% (33)	0.153a
Family history of CAD	23.2% (64)	8.3% (10)	8.8% (9)	<0.001a
Obesity	6.5% (18)	5% (6)	2.9% (3)	0.383b
Alcohol	1.4% (4)	0% (0)	0% (0)	0.093b
LVEDP (mmHg)	18.5 ± 5.6	19.5 ± 6	21.4 ± 7.2	<0.001c
≤15 mmHg	54% (149)	44.2% (53)	36.3% (37)	0.005a
15-25 mmHg	38.8% (107)	45.8% (55)	46.1% (47)	
>25 mmHg	7.2% (20)	10% (12)	17.6% (18)	
LVEF (%)	42.3 ± 9.1	41 ± 8.9	40.4 ± 9.3	0.140c

a=Chi-square test, b=Likelihood ratio test, c=One-way ANOVA, RV=right ventricular, CAD=coronary artery diseases, LVEDP= left ventricular end-diastolic pressure, LVEF=left ventricular ejection fraction

Table 2: Distribution of demographic and angiographic findings stratified by left ventricular end-diastolic pressure (LVEDP)

Characteristics	LVEDP			P-value
	≤15 mmHg	15-25 mmHg	>25 mmHg	
Total (N)	**239 (48%)**	**209 (42%)**	**50 (10%)**	-
Gender				
Female	72.8% (174)	78.5% (164)	84% (42)	0.15a
Male	27.2% (65)	21.5% (45)	16% (8)	
Age (years)	53.4 ± 11.66	53.43 ± 11.27	56.35 ± 13.83	0.259c
Door to balloon time (minutes)	55.92 ± 21.84	60.66 ± 26.75	62.24 ± 35.76	0.084c
Total ischemic time (hours)	6.61 ± 1.76	6.53 ± 2.38	7.33 ± 2.62	0.057c
0 to 4	5.4% (13)	10% (21)	4% (2)	0.047b
5 to 8	72.8% (174)	68.4% (143)	56% (28)	
9 to 12	21.3% (51)	20.1% (42)	38% (19)	
>12	0.4% (1)	1.4% (3)	2% (1)	
Heart Rate (bpm)	77 ± 14.5	88.7 ± 16.8	110.1 ± 18.1	<0.001c
Systolic blood pressure (mmHg)	120.2 ± 16.2	121.8 ± 19.7	105.3 ± 21.6	<0.001c
Diastolic blood pressure (mmHg)	75.6 ± 9.4	76.3 ± 10.6	67.7 ± 12.6	<0.001c
Killip Class				
I	95.8% (229)	74.2% (155)	14% (7)	<0.001b
II	3.8% (9)	23.4% (49)	50% (25)	
III	0.4% (1)	1.4% (3)	12% (6)	
IV	0% (0)	1% (2)	24% (12)	
Type of myocardial infarction (MI)				
Anterior	32.2% (77)	72.2% (151)	84% (42)	<0.001b

Inferio-posterior	7.9% (19)	3.8% (8)	0% (0)	
Inferior	37.7% (90)	10% (21)	6% (3)	
Inferior plus RV infarction	12.6% (30)	7.2% (15)	4% (2)	
Isolated posterior	6.3% (15)	4.3% (9)	4% (2)	
Lateral	3.3% (8)	2.4% (5)	2% (1)	
Mechanical ventilation	0.4% (1)	1.9% (4)	26% (13)	0.001b
Co-morbid conditions				
Hypertension	63.6% (152)	69.4% (145)	86% (43)	0.008a
Diabetes mellitus	42.7% (102)	40.7% (85)	62% (31)	0.021a
Current smoker	27.6% (66)	32.1% (67)	38% (19)	0.286a
Family history of CAD	19.7% (47)	14.4% (30)	12% (6)	0.208a
Obesity	6.3% (15)	4.8% (10)	4% (2)	0.700b
Alcohol	0.4% (1)	1% (2)	2% (1)	0.540b
Total length of lesion (mm)	25.6 ± 10.3	27.6 ± 10.9	27.4 ± 9.5	0.002c
Average vessel diameter	3.2 ± 0.3	3.3 ± 0.3	3.3 ± 0.3	0.064c
Final TIMI flow in culprit vessel				
0	61.9% (148)	64.6% (135)	74% (37)	0.013b
I	4.2% (10)	4.3% (9)	0% (0)	
II	7.9% (19)	8.6% (18)	18% (9)	
III	25.9% (62)	22.5% (47)	8% (4)	

a=Chi-square test, b=Likelihood ratio test, c=One-way ANOVA, RV=right ventricular, CAD=coronary artery diseases, LVEDP= left ventricular end diastolic pressure, TIMI=Thrombolysis in Myocardial Infarction

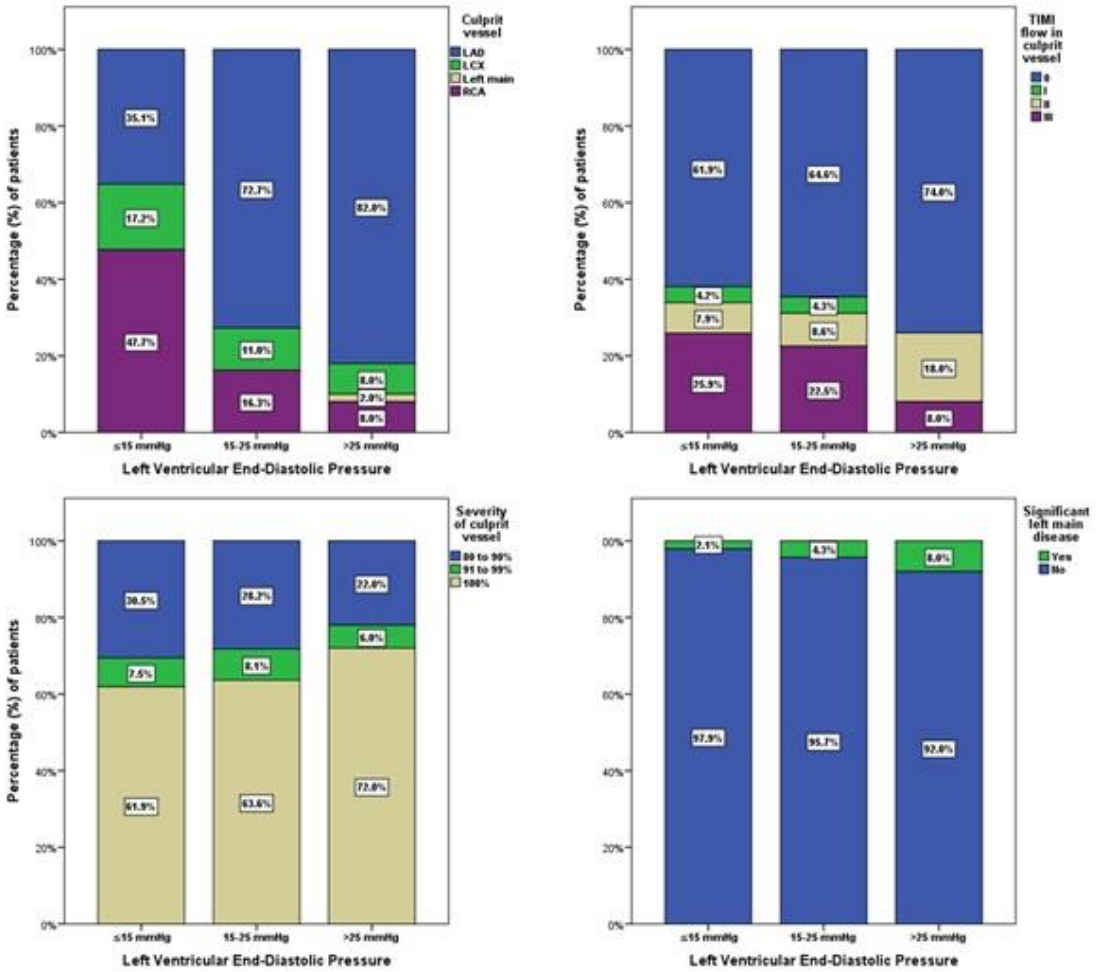

Figure 1: Distribution of culprit artery (A), initial Thrombolysis in Myocardial Infarction (TIMI) flow grade (B), severity of diseases (C), and significant left main disease (D) stratified by left ventricular end-diastolic pressure (LVEDP)

LAD=left anterior descending artery, RCA=right coronary artery, LCx=left circumflex artery

DISCUSSION

The prognostic role of LVEDP is well established, but how it relates to the burden and distribution of diseases still needs to be well elucidated. Therefore, in this study, we evaluated the association of LVEDP with the extent and severity of CAD. We observed that the burden of diseases was found to be significantly associated with LVEDP level ($p<0.001$) with mean LVEDP of 18.5 ± 5.6 mmHg among patients with SVD, 19.5 ± 6 mmHg among patients with 2VD, and 21.4 ± 7.2 mmHg among patients with 3VD. Proportion of 3VD was 15.5% (37/239) at LVEDP ≤15 mmHg, 22.5% (47/209) at LVEDP 15-25 mmHg, and 36% (18/50) at LVEDP > 25 mmHg. Similarly, there was a strong inverse relationship between LVEDP and initial TIMI flow grade ($p=0.013$). An increased proportion of culprit left anterior descending artery (LAD) was found to be associated with increased LVEDP, while there was an inverse association between the proportion of culprit right coronary artery (RCA) and left circumflex with LVEDP level. Also, there was a positive relationship between LVEDP and the lesion length.

Although very limited data are available in the context of the association between LVEDP and disease burden, research led by Du LJ et al.[10] evaluated the association of LVEDP with the severity and extent of CAD on left heart catheterization of 912 patients. Elevated LVEDP was reported in patients with CAD and compared to subjects without CAD, and it was found to be an independent predictor of the presence of CAD. Similar to our observations, a positive correlation between the number of involved vessels and LVEDP was observed, and LVEDP was also significantly associated with the Gensini score. In another study by Lin FY et al.[11] showed that on coronary CT angiography, increased LVEDP is linked to the severity and burden of disease in both obstructive and non-obstructive CAD cases.

A study led by Ndrepepa G et al.[8] observed that individuals with STEMI who had elevated LVEDP during primary PCI were highly vulnerable to cardiac death during the follow-up period of up to 8 years following reperfusion. Larger myocardial ischemia was found to be correlated with elevated LVEDP, with 17.0% initial area at risk at 1st LVEDP tertile and 38% in the 3rd LVEDP tertile. Also, there was a significant positive correlation between LVEDP and scintigraphic infarct size, with a significant inverse relationship with myocardial salvage after 1 to 2 weeks of primary PCI. LVEDP measures acute LV pump performance, contractility, filling, compliance, and extent of ischemic injury.[3] LV cavity pressure may be susceptibly altered by the intramyocardial vessels due to the proximity of myocardial and vascular compartments.[12] External compressive forces may be transmitted on the microcirculation due to increased LV cavity pressure, resulting in decreased perfusion pressure and increasing endocardial capillary pressure, hence affecting the territory of the culprit artery and leading to impaired microvascular perfusion.[12,13] Correlation between LVEDP and zero-flow pressure also corroborate this theory.[14] Additional aspects that may alter LVEDP in STEMI include vasodilatory drugs, such as nitrate, and intravascular volume status, which may reduce LVEDP momentarily.[3,15,16] A recent analysis published by Ammar A et al.[17] reported elevated LVEDP as an independent predictor of contrast-induced nephropathy.

Myocardial infarction affects diastolic function in the same way as it affects systolic function, but this effect is not fully elucidated. Myocardial infarction and ischemia influence both the passive filling and active relaxation physiologic phases of diastole. Following a myocardial infarction, active relaxation phases of diastole get delayed, and changes can be observed in the stiffness of the left ventricular. Such alterations are dependent on the remodeling and extent and severity of ischemia. Dilation is a counteraction of increased wall stiffness due to fibrosis and interstitial edema. Hence, alterations in diastolic function are linked to an increased risk of adverse outcomes. Furthermore, patients with co-morbid conditions associated with poor diastolic function have even higher rates of adverse events after MI.[18] Infarct size and myocardial salvage are the two key determinates of LV remodeling and survival after STEMI; hence, the association of these parameters with LVEDP could describe why elevated LVEDP levels are linked to increased risk of long term adverse cardiovascular events including cardiac mortality.[8] A study by Saito et al.[19] observed decreased LV end-systolic volume index and LVEF recovery for the patients with elevated baseline LVEDP, indicating the LVEDP's role in subsequent congestive heart failure development and LV remodeling. Hence, in the course of percutaneous coronary intervention, if coronary stenosis is severe, LVEDP should also be measured, which can provide additional insights into the diastolic function and earliest detection of underlying diastolic dysfunction and appropriate management can not only prevent the development of congestive heart failure but also will help in improving overall outcomes of the STEMI patients. Hence, to prevent adverse outcomes, decreasing the length of the procedure, limiting the use of contrast agents, and initiating vasodilator drugs can be preventive measures of operator in patients with elevated LVEDP.

Our study has several limitations that need to be addressed. Firstly, it is important to acknowledge the observational nature of our study, which was conducted at a single center with a relatively small sample size. These factors may restrict the generalizability of our study findings. Consequently, larger-scale investigations are warranted to corroborate the association between LVEDP and the burden and severity of disease in STEMI patients.

CONCLUSION

In conclusion, increased LVEDP was found to be associated with increased burden and extent of coronary artery disease, poor initial TIMI flow grade, and longer length of lesion. Elevated LVEDP was also found to be associated with anterior wall MI. Hence, LVEDP should be routinely measured during primary PCI. It can help in the early detection of underlying diastolic dysfunction and appropriate management to prevent the development of subsequent congestive heart failure.

AUTHORS' CONTRIBUTION

BAS, KAS, JAS and RK: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. KAK, MKB, AN, TS, and NQ: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

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Address for Correspondence:

Dr. Bashir Ahmed Solangi, Associate professor of Adult Cardiology, National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan.
Email: bashir1981.ba@gmail.com