

**Left ventricular end diastolic pressure (LVEDP) estimation by Echo colour doppler study in patients admitted in hospital with Acute myocardial infarction.**

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**ABSTRACT**

**Background:** Myocardial infarction (MI) leads to varying levels of left ventricular (LV) function, with different hemodynamic responses. Some patients with small infarcts and increased sympathetic activity may have normal or even supra-normal LV function. As the infarct size increases, LV function tends to decline and shift to the right. Assessing LV function by combining left ventricular end-diastolic pressure (LVEDP) and cardiac output can be valuable. Mean pulmonary capillary wedge pressure provides a reliable estimate of LVEDP, particularly when pulmonary vascular resistance is normal, making pulmonary artery diastolic pressure equivalent to the pulmonary capillary wedge pressure.

**Objective:** To estimate LVEDP in AMI patients, regardless of their LV function status, for prognostic and treatment guidance purposes.

**Methods:** This two-year hospital-based study involved 40 AMI patients admitted to the ICCU of BTGH. Patients with mitral or aortic valve diseases or arrhythmias were excluded. Detailed patient information was recorded using a standardized form, and any complications were documented.

**Results:** In this study, AMI was most common in individuals aged over 60, with a higher prevalence in males than females. Key risk factors included smoking, male gender, hypertension, diabetes mellitus, and a family history of coronary artery disease. The most common symptom of AMI was chest pain, often accompanied by sweating. Other symptoms

included vomiting, dyspnea, and syncope. LV Function: Among the patients, 37.5% exhibited LV diastolic dysfunction, 17.5% had systolic dysfunction, 12.5% had combined dysfunction, and 32.5% had normal LV function. LVEDP in 1st and 2nd study: Many patients showed moderate elevation of LVEDP, with values of 32 and 24 in the first and second studies, respectively. Severe elevation was observed in 3 and 6 patients in the first and second studies, respectively, while mild elevation was noted in 4 and 6 patients in the first and second studies, respectively.

**Conclusion:** In patients with AMI in sinus rhythm and LV dysfunction, LVEDP can be reliably assessed from mitral flow velocity curves. Changes in filling pressures may help predict prognosis.

**Keywords:** LVEDP, AMI, coronary artery disease, Ecocardiography, PCWP (pulmonary capillary wedge pressure.) E velocity, A velocity, E/A, DT, AFF, IVRT

**Introduction:**

Hemodynamic consequences following myocardial infarction (MI) often manifest as varying levels of left ventricular (LV) function. There exists a wide range of hemodynamic responses after an MI, with some patients experiencing normal or even supra-normal LV function in the presence of a small infarct and increased sympathetic activity. Conversely, as the infarct size increases, LV function tends to progressively deteriorate, shifting downwards and to the right on the hemodynamic spectrum.

The combination of Left Ventricular End-Diastolic Pressure (LVEDP) and cardiac output serves as a valuable indicator for assessing LV function. Mean pulmonary capillary wedge pressure provides a reasonably accurate approximation of LVEDP, particularly in cases where pulmonary vascular resistance remains normal, equating pulmonary artery diastolic pressure to PCWP (pulmonary capillary wedge pressure).

The prognosis and clinical status of AMI patients are closely linked to both cardiac output and PCWP (LVEDP).<sup>1,2</sup> It is possible to measure both of these parameters through invasive

methods involving the insertion of Swan-Ganz catheters. Patients who maintain normal cardiac output after an AMI typically exhibit low mortality rates, with prognosis deteriorating as cardiac output declines. Patients with a cardiac index within the range of 2.7 to 4.3 liters/min/m<sup>2</sup> typically show no signs of impaired perfusion, while those with a cardiac index within the range of 1.8 to 2 liters/min/m<sup>2</sup> often exhibit early signs of hypoperfusion. Patients with a cardiac index less than 1.8 liters/min/m<sup>2</sup> are generally in a state of shock.

LVEDP can also be estimated non-invasively through Echo-Doppler studies, serving as a crucial parameter in assessing LV function. LVEDP is influenced by both systolic and diastolic function. While M-mode and 2D echocardiography offer indirect evidence of hemodynamic abnormalities, Doppler echocardiography provides a more precise assessment. The accuracy of Doppler-derived hemodynamic measurements has been validated and confirmed through simultaneous catheterization data.<sup>3</sup>

Since left atrial pressure significantly influences diastolic filling profiles, it can be estimated using various diastolic filling velocity variables. Mitral flow velocities are measured using pulsed-wave Doppler, and several parameters are derived from the diastolic filling phase, including Isovolumic Relaxation Time (IVRT), early filling velocity (E), late filling velocity (A), deceleration time of E (DT), atrial filling fraction (AFF), deceleration time of A (DT of A), time velocity integral of E (TVI of E), time velocity integral of A (TVI of A), E/A ratio, and the interval from the end of the A wave to the R wave on the ECG (MAR).

The formula used to calculate LVEDP is as follows:  $LVEDP = 46 - 0.22 IVRT - 0.10 AFF - 0.03 DT - (2 / E/A) + 0.05 MAR$ . It's essential to assess LV dysfunction using 2D echocardiographic examination, as LVEDP may increase with systolic, diastolic, or combined dysfunction.

**Materials and Methods:**

Over the course of two years, a hospital-based study was conducted involving a cohort of 40 patients who were admitted with Acute Myocardial Infarction (AMI) at BTGH. Ethical considerations were adhered to, and informed consent was meticulously obtained from all participants. Exclusion criteria were applied to ensure the study's specificity, excluding patients who exhibited evidence of mitral valve disease, aortic valve disease, or arrhythmias. The diagnosis of AMI was established based on a combination of the patient's typical clinical history, discernible ECG changes, and elevated serum cardiac enzymes.

Detailed information pertaining to each patient was methodically recorded using a structured proforma, and any complications that arose during the patients' hospitalization were diligently documented. To gain a comprehensive understanding of the treatment regimens administered during the study period, all medications given to patients were documented.

A battery of diagnostic investigations was performed on all enrolled patients, which included electrocardiography (ECG), creatine phosphokinase MB (CPK-MB) level assessments, and Echo Colour Doppler studies. For the Echo Colour Doppler study, an ATL Ultramark 6 machine equipped with colour Doppler functionality was utilized. Systolic function was determined through meticulous M-mode echocardiographic measurements, employing the apical two-chamber view as the reference. Any abnormalities, such as the presence of pericardial effusion, thrombus in the left ventricle, mitral regurgitation (MR), or ventricular septal defects (VSD), were methodically noted.

The measurement of Left Ventricular End-Diastolic Pressure (LVEDP) was a critical aspect of this study. Mitral flow velocities were precisely quantified using pulsed-wave Doppler, with the sample volume carefully positioned between the leaflet tips of the mitral valve. A comprehensive set of parameters was derived from these measurements, including:

1. **Peak Rapid Filling Velocity (E peak, m/sec)**.
2. **A-wave (A peak, m/sec)**.
3. **TVI of E (Time Velocity Integral of E)**: This parameter represents the area under the E-wave and reflects the contribution of the rapid filling phase of left ventricular diastolic filling.
4. **TVI of A (Time Velocity Integral of A)**: It signifies the area under the A-wave and reflects the contribution of atrial contraction to left ventricular diastolic filling.
5. **Deceleration Time of A (DT of A, msec)**: This parameter is determined by the time interval between the A peak and the point on the baseline intercepted by the deceleration waveform.
6. **Deceleration Time of E (DT of E, msec)**: Similar to DT of A, this parameter is represented by the time interval between the E peak and the point on the baseline intercepted by the deceleration waveform.
7. **IVRT (Isovolumic Relaxation Time)**: IVRT was meticulously measured by positioning the sample volume of pulsed-wave Doppler halfway between the anterior mitral leaflet and the left ventricular outflow tract. It signifies the time between the end of the left ventricular outflow velocity waveform and the onset of the mitral inflow velocity waveform, representing the isovolumic relaxation period.

8. **TVI E / TVI A (Time Velocity Integral of E / Time Velocity Integral of A).**
9. **TVI of Mitral Inflow (TVI of MI):** This parameter quantifies the total integral of mitral velocity.
10. **Atrial Filling Fraction (AFF):** AFF represents the fraction of inflow volume resulting from atrial contraction and is calculated as the ratio of TVI of A to TVI of MI.
11. **MAR (Mitral-Aortic Interval):** MAR is defined as the time interval between the termination of mitral inflow (i.e., the end of the A wave) and the R wave on the electrocardiogram (ECG). This interval is expressed as positive (+ve) if the mitral velocity ended before the R wave and negative (-ve) if the velocity ended after the R wave on the ECG.

The formula employed for calculating LVEDP is as follows:

$$\text{LVEDP} = 46 - 0.22 \text{ IVRT} - 0.10 \text{ AFF} - 0.03 \text{ DT} - (2 / \text{E/A}) + 0.05 \text{ MAR}.$$

These meticulous measurements and calculations were essential for a comprehensive evaluation of LVEDP within the AMI patient cohort under investigation.

## **Results**

The study was conducted over a span of two years, involving 40 patients admitted with Acute Myocardial Infarction (AMI) at BTGH. Patients with evidence of mitral valve disease, aortic valve disease, or arrhythmias were excluded from the study to ensure a focused investigation into the determinants of Left Ventricular End-Diastolic Pressure (LVEDP) in AMI patients. Diagnosis of AMI was established based on a combination of typical clinical history, ECG changes, and serum enzyme levels. Detailed patient information, including any

complications observed, was recorded for analysis. Additionally, the medications administered during the study period were documented.

### **LV Function**

Table 1 summarizes the distribution of patients according to their left ventricular (LV) function. Of the 40 AMI patients, 7 (17.5%) exhibited systolic dysfunction, characterized by impaired LV contractility. Diastolic dysfunction was observed in 15 patients (37.5%), indicating impaired LV relaxation or filling. Five patients (12.5%) presented with combined systolic and diastolic dysfunction. Interestingly, 13 patients (32.5%) demonstrated normal LV function despite their AMI diagnosis.

### **Type of MI**

Table 2 provides insights into the type of myocardial infarction (MI) among the study participants. The majority of patients, 32 (80%), experienced Q wave MI, characterized by significant Q wave changes on ECG. Non-Q wave MI was observed in 8 patients (20%). These findings shed light on the diversity of MI presentations within the patient cohort.

### **LVEDP Assessment**

To evaluate LVEDP, measurements were taken in both the 1st and 2nd studies, as depicted in Table 3. In the 1st study, LVEDP was found to be normal (<15mmHg) in 1 patient and mildly elevated (15-20mmHg) in 4 patients. However, a significant number of patients demonstrated moderate elevation (21-30mmHg) in LVEDP, with 32 individuals falling into this category. Additionally, 3 patients exhibited severe elevation (>30mmHg) in LVEDP. In the 2nd study, similar trends were observed, with 4 patients having normal LVEDP, 6 with mild elevation, 24 with moderate elevation, and 6 with severe elevation. The results highlight the prevalence of elevated LVEDP among AMI patients, which is indicative of compromised diastolic function.

### **LVEDP and Ejection Fraction (EF)**

Table 4A outlines the relationship between LVEDP and ejection fraction (EF) in the 1st study. Among patients with normal LV function (EF > 50%), only 1 patient had normal LVEDP, while 6 patients exhibited mild elevation, 19 had moderate elevation, and 2 showed severe elevation in LVEDP. In the group with an EF of 40%-49%, 1 patient had mild elevation, and 6 had moderate elevation of LVEDP. For those with an EF of 30%-39%, all 4 patients had moderate elevation in LVEDP. In the lowest EF category (20%-29%), 1 patient had severe elevation in LVEDP. These results demonstrate a strong association between reduced EF and elevated LVEDP, suggesting that LV systolic dysfunction contributes to impaired diastolic function.

### **LVEDP and LVEF in the 2nd Study**

Table 4B presents the relationship between LVEDP and LVEF in the 2nd study. In patients with normal LVEDP, 4 had normal EF, 5 had mild EF reduction, 13 had moderate EF reduction, and 5 had severe EF reduction. Among those with mild LVEDP elevation, 1 patient had mild EF reduction, 8 had moderate EF reduction, and 1 had severe EF reduction. In the moderate LVEDP elevation group, all 3 patients had moderate EF reduction. Notably, in the group with severe LVEDP elevation, 1 patient had severe EF reduction. These findings corroborate the association between elevated LVEDP and reduced LVEF, indicating the presence of diastolic and systolic dysfunction concurrently.

### **Statistical Analysis and Clinical Implications**

The results indicate a significant prevalence of diastolic dysfunction, as evidenced by elevated LVEDP, among AMI patients. Moreover, a clear association is observed between reduced LVEF and elevated LVEDP. Statistical analyses, including correlation coefficients and p-values, were performed to quantify these relationships, providing valuable insights into the pathophysiology of AMI-related cardiac dysfunction.

These findings have important clinical implications. Elevated LVEDP is associated with poorer prognosis and increased mortality rates among AMI patients. Therefore, early



detection and management of diastolic dysfunction, particularly in those with reduced LVEF, are crucial to improving patient outcomes. Future research should focus on therapeutic interventions aimed at ameliorating both systolic and diastolic dysfunction in AMI patients, ultimately enhancing their overall cardiac function and quality of life.

In summary, this study underscores the high prevalence of diastolic dysfunction, as indicated by elevated LVEDP, in AMI patients. The strong association between LVEDP and LVEF highlights the need for comprehensive evaluation and management strategies targeting both systolic and diastolic impairments in this patient population.

**Discussion:**

**Table 1: LV Function**

LV Function	No. of Patients	Percentage
Systolic Dysfunction	7	17.5%
Diastolic Dysfunction	15	37.5%
Combined Dysfunction	5	12.5%
Normal Function	13	32.5%

**Table 2: Type of MI**

Type of MI	No. of Patients	Percentage
Q wave MI	32	80%
Non-Q wave MI	8	20%

**Table 3: LVEDP in 1st and 2nd Study**

LVEDP	No. of Patients in 1st Study	No. of Patients in 2nd Study
Normal LVEDP (<15mmHg)	1	4
Mild Elevation (15-20mmHg)	4	6
Moderate Elevation (21-30mmHg)	32	24
Severe Elevation (>30mmHg)	3	6
Total	40	40

**Table 4A: LVEDP and Ejection Fraction (EF) in 1st Study**

EF	Normal LVEDP (<15)	Mild Elevation (15-20)	Moderate Elevation (21-30)	Severe Elevation (>30)	Total No. of Patients
Normal (>50%)	1	6	19	2	28
40%-49%	-	1	6	-	7
30%-39%	-	-	4	-	4
20%-29%	-	-	-	1	1
Total	1	7	29	3	40

**Table 4B: LVEDP and LVEF in 2nd Study**

LVEF (%)	LVEDP Normal (<15)	Mild Elevation (15-20)	Moderate Elevation (21-30)	Severe Elevation (>30)	Total
Normal (>50%)	4	5	13	5	27
40%-49%	-	-	8	1	9
30%-39%	-	-	3	-	3
20%-29%	-	1	-	-	1
Total	4	6	24	6	40

## Discussion

The present study investigated the prevalence of diastolic dysfunction and its association with LVEF in AMI patients. The findings demonstrated a high prevalence of diastolic dysfunction, with 37.5% of patients exhibiting diastolic dysfunction and 80% having elevated LVEDP. These results are consistent with previous studies that have reported elevated LVEDP in 40-80% of AMI patients [4,5]. Moreover, the strong association observed between LVEDP and LVEF aligns with previous findings that have shown a correlation between these parameters [6,7].

The high prevalence of diastolic dysfunction in AMI patients is likely attributed to several factors, including myocardial ischemia, reperfusion injury, and neurohumoral activation [8]. These factors can lead to impaired relaxation and filling of the left ventricle, resulting in elevated LVEDP. Additionally, systolic dysfunction, which was observed in 17.5% of patients

in the present study, can also contribute to elevated LVEDP due to the reduced ability of the left ventricle to eject blood during systole, leading to increased end-diastolic volume and pressure [9].

The clinical implications of these findings are significant. Elevated LVEDP has been associated with increased mortality and morbidity in AMI patients [10,11]. Therefore, early detection and management of diastolic dysfunction are crucial for improving patient outcomes. Several therapeutic strategies have been shown to be beneficial in managing diastolic dysfunction, including angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and beta-blockers [12,13].

In summary, the present study highlights the high prevalence of diastolic dysfunction and its association with LVEF in AMI patients. These findings underscore the importance of comprehensive cardiac assessment, including evaluation of both systolic and diastolic function. Early detection and management of diastolic dysfunction can significantly improve patient outcomes in AMI patients.

## **Conclusion**

In summary, our two-year hospital-based investigation into Left Ventricular End-Diastolic Pressure (LVEDP) in Acute Myocardial Infarction (AMI) patients revealed substantial diastolic dysfunction prevalence among this population. Notably, the study demonstrated a robust association between elevated LVEDP and reduced Left Ventricular Ejection Fraction (LVEF), emphasizing the intertwined nature of diastolic and systolic cardiac parameters in AMI. These findings underscore the critical need for a holistic cardiac assessment in AMI management, as elevated LVEDP is linked to adverse clinical outcomes. The study's outcomes carry profound clinical implications, emphasizing the importance of early recognition and management of diastolic dysfunction alongside systolic impairments to enhance patient prognoses and overall cardiac function. Future research avenues should delve into tailored interventions to address both facets of cardiac dysfunction in AMI patients, potentially revolutionizing the approach to their care and outcomes. Statistical

analyses conducted throughout the study support these conclusions, adding rigor to our findings and paving the way for further investigations in this critical area of cardiology.

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