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Expert Opinion Article

LVEDP: A Suggestive Predictor Of Mortality In Stemi Patients Undergoing Primary PCI

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INTRODUCTION

Left ventricular end-diastolic pressure (LVEDP) is directly related to left ventricular (LV) compliance and intravascular volume and pressure, and one of the main consequences of LV diastolic dysfunction is increased LV Filling pressures.

In the context of coronary artery disease, elevated filling pressures can be caused by acute ischemia, an infarction scar, or myocardial hibernation. [1,2,3,4,5]

Acute ischemia results in an upward and rightward shift of the diastolic pressure-volume curve, which may be due to various factors: calcium overload in cardiomyocytes resulting in late activation and incomplete relaxation, pericardial constriction due to increased atrial and ventricular volume, intra and interventricular asynchrony and papillary muscle dysfunction.[6,7,8]

Variables known to affect prognosis in ST-segment elevation myocardial infarction (STEMI) include age, associated co-morbidities, infarct size and location, left ventricular systolic function, ventricular arrhythmias, ischemic mitral regurgitation, and cardiogenic shock.The prognostic utility of left ventricular ejection fraction (LVEF) measured during or after STEMI is also well established.[9,10,11]

However, although diastolic dysfunction precedes the onset of systolic dysfunction in acute ischemia, the prognostic utility of diastolic indexes in patients with STEMI has rarely been examined.Moreover, although left ventricular end diastolic filling pressure (LVEDP), which reflects global ventricular compliance, is routinely measured during left heart catheterization, no studies have evaluated the implications of LVEDP in patients with STEMI undergoing primary percutaneous coronary intervention (PCI). [12,13,14,15,16,17]

We therefore, have examine the prognostic value of LVEDP and the interrelation between LVEDP and LVEF as assessed during the primary PCI procedure in patients with STEMI.

Aim and objectives-

- To predict mortality at 30 days and at 6 month among STEMI patients undergoing primary PCI on the basis of LVEDP.
- To find out correlation between LVEDP and EF in patients of STEMI undergoing primary PCI.

Material and methods

Study design: Hospital based observational study.
Setting: Department of Cardiology, S.M.S Medical College, Jaipur.
Study population:100 cases of STEMI patients undergoing primary PCI.
Study period:-12 months

Inclusion criteria:-

Patients with STEMI presenting to emergency department and to be taken up for primary PCI.

Exclusion criteria:-

- Previous myocardial infarction
- Previous coronary angioplasty
- Previous coronary grafting
- Chronic heart failure
- LV clot

All patients with STEMI and to be taken up for primary PCI were enrolled in this study.Detailed history and risk factors were recorded.Routine physical and biochemical examinations were done for all patients in study.LVEDP was recorded just before coronary angiogram. LVEDP was measured at the Z-point, which is identified on the LV pressure trace as the point at which the slope of the ventricular pressure upstroke changes, which coincides with the R wave on the electrocardiographic tracing.

STATISTICAL ANALYSIS-

Continuous is summarized in form of Mean±standard deviation.
Difference in the mean of two group is analyzed in form of student “t” test.
Continuous data is expressed in form of proportion.
Difference in proportion isanalyzed with chi square test.
Level of significance is analyzed at 95% confidence interval.
Pearson correlation is used to evaluate the linear correlation between LVEDP and LVEF.

RESULTS -

Outcomes were assessed at 30 day and 6 month after primary PCI.
Net adverse clinical events:-

- Death all cause cardiac and non cardiac

- Pumonary edema
- Cardiogenic shock
- Re infarction
- Stroke
- Major bleeding
- Stent thrombosis

- Baseline characteristic and LVEDP

	LVEDP(<18 mmHg) n=62	LVEDP(>18 mmHg) n=38	P value
Age (years)	56±8.2	60±10.1	0.07
Sex (m/f)	50(80.6)/12(19.3)	30(84.2)/8(15.7)	0.18
BMI(Kg/m²)	27±1.6	27.2±1.4	0.16
HTN	30(48)	16(42)	0.78
DM	10(16)	12(19)	0.05

Table 1

	LVEDP(<18mmHg) n=62	LVEDP(>18mmHg) n=38	P value
Smoking	30(48)	14(36)	0.68
Hb(g/dl)	14.6±1.4	14.2±1.6	0.78
Creatinine clearance(ml/min)	91.1±16.3	88.6±15.7	0.06
Door to ballon time(min)	68±18.6	72±20.1	0.04
Killip class(II-IV)	3(4.8)	6(15.7)	0.005
LVEF(%)	42±6.8	38±5.8	0.001

Table 2

	LVEDP(<18mmHg) n=62	LVEDP(>18mmHg) n=38	P value
Angiographic characteristic:			0.001
SVD	36	6	
DVD	18	20	
TVD	6	8	
LM	2	4	
PROX. LAD	11	18	
Procedural characteristic:			
No . Of vessels treated	1.0±0.2	1.0±0.2	0.57
No. of lesions treated	1.1±0.4	1.1±0.5	0.68
No. of stents implanted	1.5±0.9	1.5±0.8	0.19
TIMI flow			.003
0/1	9(14.5)	12(31)	
2/3	53(85.5)	26(69)	

Table 3

- Follow up study at 30 days:

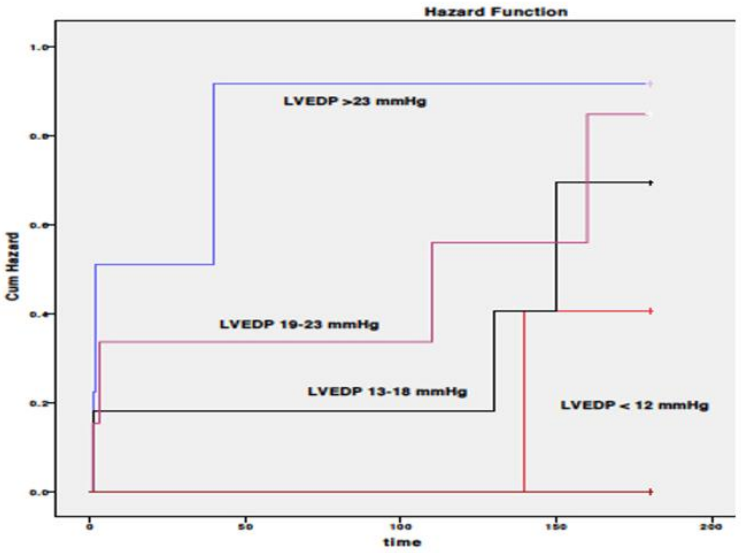
	LVEDP(<18mmHg) n=62	LVEDP(>18mmHg) n=38	P value
Death all cause Cardiac Non cardiac	- -	3 3 -	0.005
Pulmonary edema	-	2	0.04
Re infarction in same territory	-	3	0.05
Re infarction in other territory	-	-	-
Stroke	-	-	-
Bleeding	-	-	-
Stent thrombosis	-	3	0.001

Table 4

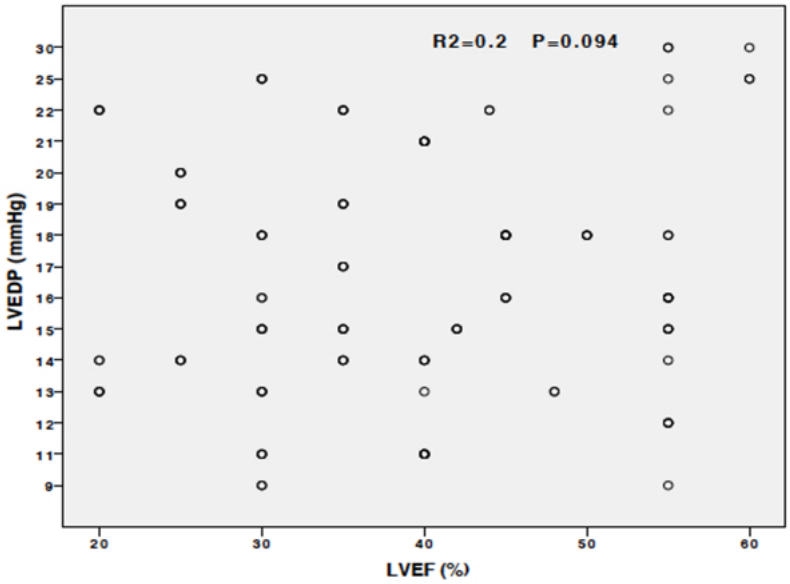
- Follow up study at 6 months:

	LVEDP(<18mmHg) n=62	LVEDP(>18mmHg) n=38	P value
Death all cause Cardiac Non cardiac	1 - 1	6 6 0	0.001
Pulmonary edema	1	3	0.05
Re infarction in same territory	-	3	0.04
Re infarction in other territory	-	2	0.04
Stroke	-	2	0.05
Bleeding	-	-	-
Stent thrombosis	-	3	0.001

Table 5



GRAPH 1



GRAPH 2

Of 100 patients enrolled in this study,LVEDP and EF were measured during the index event.Median LVEDP was measured which was 18 mmHg.

Baseline clinical,angiographic and procedural data were stratified by median LVEDP and devided in two groups.

Patients with LVEDP >18 mm Hg compared to those with LVEDP <18 mm Hg had more diabetes, pulmonary edema, higher Killip class and lower LVEF, higher door-to-balloon time, more culprit left anterior descending coronary artery disease, and were more likely to have baseline TIMI grade 0/1 flow on presentation.

There were no significant differences in number of lesions and vessels treated.

Of patients undergoing PCI, TIMI grade 3 flow was established slightly less frequently in patients with LVEDP >18 mm Hg compared to those with LVEDP <18 mm Hg (85.5% vs 69%, p 0.003).

Patients with LVEDP >18 mm Hg were also more likely to have increase in serum creatinine with decrease in creatinine clearance(p 0.06).

Patients with LVDEP >18mmHg Hg had significantly higher unadjusted rates of death, reinfarction,pulmonary edema,stent thrombosis at 30 days and 6 month.

Mortality was higher in those with an LVEDP > 18 mm Hg.(p=.0001)

Linear regression analysis demonstrated a very weak correlation between LVEDP and LVEF (R^2 0.02, p= 0.094;).

Stratified by medians, 6 month rates of death or reinfarction in patients with LVEF >40% and LVEDP< 18 mm Hg, LVEF >40% and LVEDP >18 mm Hg , LVEF< 40% and LVEDP< 18 mm Hg , and LVEF <40% and LVEDP >18 mm Hg were 7.4%, 9.1%, 12.3%, and 15.8%, respectively (p 0.01).

DISCUSSION-

The present study demonstrates that increased LVEDP is strongly associated with death, reinfarction,pulmonary edema, and stent thrombosis after primary PCI and is an independent predictor of death or reinfarction or stent thrombosis after primary PCI in patients with STEMI, even after adjustment for baseline LVEF.

One of the earliest signs of acute ischemia is diastolic dysfunction and LVEDP may be increased in the presence of ischemia with normal LVEF.[18,19,20,21,22,23]

Moreover, ischemia with subsequent impairment of myocardial contractility is associated with increased LVEDP because of an upward shift of the EDP point in the pressure–volume loop.Patients with increased LVEDP also frequently had other high-risk characteristics including diabetes, previous pulmonary edema and higher Killip class on presentation, and an occluded left anterior descending coronary artery infarct.

The prognostic utility of LVEDP measurement persisted after adjustment for baseline variables. As expected, systolic dysfunction was also more common in patients with an increased LVEDP. However, and perhaps surprisingly, there was only a weak inverse correlation between LVEF and LVEDP measured concomitantly. [24,25,26,27]

Thus, indexes of systolic and diastolic functions during STEMI in patients undergoing primary PCI provide independent and complementary prognostic information.

It has previously been shown that LVEF measured during the index procedure in patients with STEMI undergoing primary PCI is an important determinant of early and late mortality.Left ventriculography also provides potentially critical information on regional wall motion and may identify unexpected mechanical complications of STEMI. However, some operators do not perform left ventriculography during primary PCI because of concerns of delay to reperfusion, contrast nephropathy, hypotension, and ventricular arrhythmias. Measurement of LVEDP avoids these risks and, although not possessing all the prognostic utility of LVEF, is still an independent correlate of early and late death or reinfarction.There are several possible explanations for the association between increased LVEDP and subsequent death or reinfarction. Myocardial perfusion is driven by the diastolic pressure gradient between aortic and LV pressures, which determine endocardial capillary pressure. The fact that TIMI grade 3 flow was established less commonly in patients with increased LVEDP in this study is therefore not unexpected and likely contributed the poor prognosis in these patients.Moreover, myocardial perfusion is inversely proportional to LVEDP.

In consequence, even after successful reperfusion of the epicardial coronary artery, high LVEDP results in decreased perfusion pressure, impaired microvascular flow and subendocardial oxygen delivery, an sympathetic and neurohormonal activation resulting in pathologic myocardial remodeling.

Pulmonary edema and increased LVEDP have been found to be associated with contrast-induced nephropathy after PCI which is strongly associated with death and reinfarction.In the present study patients with high LVEDP also had a higher rate of increased creatinine after PCI.Some patients with increased LVEDP may have concomitant previous conditions associated with relaxation abnormalities and LV hypertrophy (e.g., hypertension and diabetes) and poorly tolerate ischemia and hemodynamic changes of STEMI.Nevertheless, this study does not provide evidence for causality for the association between LVEDP and outcome and should be interpreted as hypothesis generating.

CONCLUSION

LVEDP is useful and independent predictor of mortality and morbidity following primary percutaneous intervention irrespective of ejection fraction.

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