

Echocardiographic Evaluation of Left Ventricular Diastolic Function after Percutaneous Coronary Intervention in Patients with Acute STEMI

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ABSTRACT

BACKGROUND

Although percutaneous coronary intervention (PCI) is an excellent therapy for coronary artery disease, the effects of PCI on left ventricular diastolic function have not been systematically investigated in patients of Acute ST Elevation Myocardial Infarction (STEMI) in our population. The aim of this study was to investigate the reversibility of these diastolic abnormalities and improvement in left ventricular diastolic function by using echocardiographic diastolic parameters in patients with Acute STEMI in the setting of PCI and thus determine the effects of improved myocardial perfusion on impaired left ventricular diastolic abnormalities.

METHODS

A total of 100 consecutive patients admitted to Intensive Coronary Care Unit (ICCU), was included in the study. Echocardiography was done before PCI and 48 hours after PCI, to evaluate the indices of LV diastolic function in these patients.

RESULTS

The mean age of the patients was 52.04 ± 9.49 years, and majority of patients were males (84%). All had mild to moderate degree of left ventricular diastolic dysfunction. Mitral E wave velocity ($63.41 \text{ cm/s} \pm 19.93$ before treatment versus $71.51 \text{ cm/s} \pm 9.56$, 48 hours after treatment), the peak velocity of late filling due to atrial contraction (mitral A wave velocity) ($75.93 \text{ cm/s} \pm 20.3$ before treatment vs. $78.96 \text{ cm/s} \pm 24.18$, 48 hours after treatment), E/A ratio (1.03 ± 0.3 before treatment vs. 0.98 ± 0.24 , 48 hours after treatment) showed improvement after PCI. After PCI deceleration time (DT) decreased ($210.15 \text{ msec} \pm 47.43$ before treatment versus $201.64 \text{ msec} \pm 28.15$, 48 hours after treatment), and the difference was statistically significant ($p < 0.001$). It is notable that early diastolic mitral annular velocity (E') improved significantly 48 hours after PCI ($5.81 \text{ cm/s} \pm 1.65$ before treatment vs. $7.96 \text{ cm/s} \pm 1.95$, 48 hours after treatment, $p < 0.001$). E/ E2 ratio showed significant change 48 hours after PCI; it was statistically significant (10.17 ± 2.26 before treatment vs. 8.83 ± 1.7 , 48 hours after treatment $p < 0.001$).

CONCLUSIONS

Improvement in some indices of left ventricular diastolic function after PCI suggests that PCI can be an effective modality of treatment for diastolic dysfunction caused by myocardial ischemia and revascularization can restore diastolic properties of the heart after the insult in STEMI patients and thus prevent their progression to HFPEF.

KEYWORDS

STEMI, Left Ventricular Dysfunction, Percutaneous Coronary Intervention

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BACKGROUND

Heart failure is a global healthcare problem with an estimated prevalence of approximately 26 million. In Asia Pacific region it causes a major socioeconomic burden and high rates of hospitalizations.¹ Heart failure with preserved ejection fraction (HFPEF) is the major cause of morbidity and mortality all over the world and responsible for more than 50% of the heart failure cases. There was a substantial increase in prevalence HFPEF from 38% to 54% over past few decades. The prognosis of patients suffering from HFPEF is as ominous as prognosis of patients suffering from systolic heart failure (HFREF).² Left ventricular diastolic function is an important predictor of short and long term outcome in patients with Acute ST Elevation Myocardial Infarction (STEMI).^{3,4,5} Regional diastolic wall motion is impaired in ischemic myocardial segments, even when systolic contraction is preserved. Diastolic abnormalities of left ventricular (LV) relaxation and filling appear to be the earliest manifestation of myocardial ischemia.⁶ It was showed that diastolic function seemed to be more sensitive to ischemia than systolic function.⁷

The two physiologic phases of diastole, active relaxation and passive filling, are both influenced by myocardial ischemia and infarction. Active relaxation is delayed following a myocardial infarction. There is profound regional asynchrony between ischemic and normal myocardium, resulting in disturbed ventricular relaxation and increased myocardial stiffness.⁸ Left ventricular stiffness changes depending on the extent of infarction and remodelling. In addition, interstitial oedema and fibrocellular infiltration will directly cause an increase in wall stiffness. The effect on diastolic function is correlated to an increased incidence of adverse outcomes. Moreover, patients with comorbid conditions that are associated with worse diastolic function tend to have more adverse outcomes after infarction.

Doppler echocardiography has provided a rapid, feasible, and simple noninvasive method of assessing LV filling in various cardiac diseases⁹ in which diastolic abnormalities have been observed, including AMI^{10,11,12,13} and allows the assessment of regional diastolic performance and dynamics of the left ventricular myocardium. Temporal dyssynchrony between normal and ischemic myocardium may result in impaired left ventricular relaxation and filling and regional dyssynchrony would improve after normalization of myocardial perfusion in the ischemic zone. Thus, improved global left ventricular filling after PCI might result from improved regional left ventricular function.¹⁴ Improved resting LV diastolic filling after Left ventricular diastolic filling improves gradually after PCI, possibly reflecting the phenomenon of post ischemic "stunned" myocardium. In the setting of recurrent ischemia after AMI, an opportunity may exist during which early PCI can lead to improvement in LV filling characteristics.

The effects of PCI on left ventricular diastolic function have not been systematically investigated in patients Acute ST Elevation Myocardial Infarction (STEMI) in our population. The aim of this study was to investigate the

reversibility of these diastolic abnormalities and improvement in left ventricular diastolic function by using echocardiographic diastolic parameters in patients with Acute STEMI in the setting of PCI and thus determine the effects of improved myocardial perfusion on impaired left ventricular diastolic abnormalities.

Aims and Objectives

1. To study the proportion of patients with Acute ST-Elevation Myocardial Elevation presenting to the department of cardiology, King George Hospital, Andhra Medical college, Vishakhapatnam.
2. To study the left ventricular diastolic parameters by trans-thoracic echocardiography in patients presenting with Acute ST Elevation Myocardial Infarction and left ventricular ejection fraction >45% before undergoing percutaneous coronary intervention (PCI) and 48 hours after undergoing percutaneous coronary intervention (PCI) and study whether there is any improvement in left ventricular diastolic function in patients presenting with Acute ST Elevation Myocardial Infarction after successful PCI.

METHODS

This is a prospective non-randomized study with pre-post-test design. Total 100 consecutive patients admitted to Intensive coronary care unit (ICCU), Department of Cardiology, King George Hospital, Visakhapatnam, with Acute STEMI with EF >45% and underwent successful PCI, during the period between January 2017 and December 2017 were included in the study. Diagnosis of acute STEMI was based on chest pain lasting >30 minutes, evolving characteristic electrocardiographic abnormalities that include ST-segment elevations >1 mm in limb leads or >2 mm in chest leads in at least two consecutive leads or a new onset of LBBB and an a rise and/or fall in cardiac biomarker values (preferably cTn), with at least one value above the 99th percentile of the URL.

Before the commencement of study, permission was obtained from Ethics committee, Andhra Medical College, Vishakhapatnam. All enrolled patients were informed about the nature of the study and their rights to refuse. The informed written consent was taken before including them in the study.

Exclusion Criteria

- Patients with previous acute STEMI or prior history of percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG)
- Acute and chronic kidney disease (serum creatinine >1.7 mg/dl), or any other contraindication to CAG
- History of hypertensive heart disease, valvular heart disease, cardiomyopathies.
- ECG evidence of left ventricular hypertrophy, arrhythmias.
- Failed PCI.

Data Collection

A detailed history of coronary risk factors, prior history of MI/PCI/CABG of all the patients included in the study was taken. A thorough clinical examination of all the systems was done. Baseline blood investigations, lipid profile and cardiac biomarkers were estimated for all patients.

Electrocardiogram

ECG (BPL 50-60 Hz, 230 V or Mindray 50-60 Hz, 230 V) was taken to all patients who presented with Acute MI at the time of admission.

Echocardiogram

All patients underwent trans-thoracic echocardiogram on Day 2 of admission, 12 hours before PCI and 48 hours after PCI. Patients were evaluated by 2D-Echo, M mode, and Doppler echocardiography. Echocardiographic variables specially included RWMA (\pm), left ventricular ejection fraction (LVEF), pulsed wave Doppler assessment of mitral valve inflow patterns in apical 4-chamber view (peak early (E) and peak late (A) flow velocities, E/A ratio, A wave duration and deceleration time of early mitral flow velocity (DT), Isovolumetric relaxation time (IVRT)) Tissue Doppler Imaging (TDI) assessment at the medial mitral annulus in apical 4-chamber view (mitral annular diastolic velocity (e'), (a'), and (E/ e' ratio)) and colour M mode (propagation velocity (Vp) and E/Vp).

Coronary Angiography

Siemens cath lab system with computer-based analysis was used. Coronary angiography was performed in all patients included in the study, under local anesthesia using the Modified Seldinger technique with a radial or femoral artery approach. Culprit lesion was assessed, minimal lumen and reference diameters were determined and the degree of coronary stenosis diameter stenosis was assessed in at least two orthogonal projections. Pre-PCI angiographic variables including number of stenosed vessels (single/double/triple), and degree of stenosis (0 to 100%) were noted.

Percutaneous Coronary Intervention

After diagnostic angiography, PCI was performed in the usual manner with balloon catheters and drug eluting stents (Supralimus) in the coronary arteries with significant stenosis (>70% lumina narrowing). PCI was considered successful if the final stenosis was reduced to lower than 20%.

Statistical Analysis

The data were analysed using SPSS 11.0 for Windows (SPSS INC., Chicago, Illinois). Quantitative Variables were expressed in mean and standard deviation. Categorical variables were expressed in percentages. The means of continuous variables were compared by paired "t" test. Discrete variables were compared by Chi-square and Fisher's exact test A probability value <0.05 was statistically significant.

RESULTS

Total number of patients under study was 100. The mean age of study population was 52.04 ± 9.49 years with range from 32 to 73 years. Nearly 60% of the patients were found in the age group of 40-59 years. Study population comprised of 84 male patients (84%) and 16 female patients (16%). Male to female ratio was 5: 1 in the whole study population.

Risk Factor Profile

Smoking was the most common risk factor (43%) in our study, followed by hypertension (38%), dyslipidaemia (30%) and diabetes mellitus (28%). Out of 100 patients, 56 patients presented with acute anterior wall STEMI (56%) and 44 patients presented with acute inferior wall STEMI (44%). The mean window period at presentation to hospital was 15.06 ± 17.4 hours ranging from 1 hour to 72 hours. 73 patients were thrombolysed with Streptokinase i.e., 73% and 27 patients were not thrombolysed (27%). The mean left ventricular ejection fraction (%) was $58.91 \pm 7.99\%$, ranging from 45% to 76%.

Distribution by Number of Coronary Arteries Involved

Coronary angiographic profile of the study population showed Single vessel disease in 75 patients (75%), Double vessel disease in 23 patients (23%) and Triple vessel disease in 2 patients (2%). LAD showed significant lesion in 62 of the patients (62%). PTCA to LAD was done using drug eluting stents in 59% of the patients. RCA showed significant lesion in 41 patients (41%). PTCA to RCA was done using drug eluting stents in 37% of the patients. LCX showed significant lesion in 22 patients (22%). PTCA to LCX was done using drug eluting stents in 24% of the patients.

Mitral Inflow Parameters

In our study, mean E Velocity was 63.41 ± 19.93 cm/sec before PCI and 71.51 ± 9.56 cm/sec after PCI (p value 0.700). The mean A Velocity was 75.93 ± 20.30 cm/sec before PCI and 78.96 ± 24.18 cm/sec after PCI (p value 0.124). The mean E/A ratio was 1.03 ± 0.3 before PCI and $0.98 \pm .24$ after PCI (p value 0.004). The mean A wave duration was 111.57 ± 23.07 msec before PCI and 137.11 ± 13.69 msec after PCI (p value 0.000). The mean DT was 210.15 ± 47.43 msec before PCI and 201.64 ± 28.15 sec after PCI (p value 0.000).

	Mean \pm SD Before PCI	Mean \pm SD After PCI	Correlation	p
E velocity	63.41 ± 19.93	71.51 ± 9.56	0.039	0.700
A velocity	75.93 ± 20.30	78.96 ± 24.18	0.155	0.124
E/A	1.03 ± 0.3	$0.98 \pm .24$	0.283	0.004
A - dur	111.57 ± 23.07	137.11 ± 13.69	0.421	0.000
DT	210.15 ± 47.43	201.64 ± 28.15	0.414	0.000

Table 1. Mitral Inflow Parameters before and after PCI

Tissue Doppler Parameters

In our study, mean E' Velocity was 5.81 ± 1.65 cm/sec before PCI and 7.96 ± 1.95 cm/sec after PCI (p value 0.776). The mean A' Velocity was 8.05 ± 1.60 cm/sec before PCI and 8.02 ± 1.78 cm/sec after PCI (p value 0.000). The mean

E/E' ratio was 10.17 ± 2.26 before PCI and 8.83 ± 1.70 after PCI (p value 0.000).

	Mean \pm SD Before PCI	Mean \pm SD After PCI	Correlation	p
E'	5.81 ± 1.65	7.96 ± 1.95	0.029	0.776
A'	8.05 ± 1.60	8.02 ± 1.78	0.648	0.000
E/E'	10.17 ± 2.26	8.83 ± 1.70	0.477	0.000

Table 2. Tissue Doppler Parameters before and after PCI

Isovolumetric Relaxation Time before and after PCI

In our study, mean IVRT was 85.69 ± 14.67 msec before PCI 79.18 ± 5.09 msec after PCI (p value 0.794).

	Mean \pm SD Before PCI	Mean \pm SD After PCI	Correlation	p
IVRT	85.69 ± 14.67	79.18 ± 5.09	0.026	0.794

Table 3. Isovolumetric Relaxation Time before and after PCI

Pulmonary Venous Flow Doppler Parameters

In our study pulmonary venous doppler parameters showed S<D in 6% patients before PCI and in 2% patients after PCI; S>D in 94% patients before PCI and in 98% patients after PCI. (LR ratio -11.79, p value -0.003).

Ar-A Duration

In our study, mean Ar-A dur 6.04 ± 32.93 msec before PCI 14.49 ± 31.83 msec after PCI (p value 0.000).

	Mean \pm SD Before PCI	Mean \pm SD After PCI	Correlation	p
Ar-A dur	6.04 ± 32.934	14.49 ± 31.83	0.634	0.000

Table 4. Ar-A Duration before and after PCI

Colour M Mode Parameters- Flow Propagation Velocity

In our study, mean Vp was 32.36 ± 7.59 cm/sec before PCI and 47.05 ± 12.71 cm/sec after PCI (p value 0.179) and mean E/Vp was 2.01 ± 0.89 before PCI and 1.55 ± 0.68 after PCI (p value 0.000).

	Mean \pm SD Before PCI	Mean \pm SD After PCI	Correlation	p
Vp	32.36 ± 7.59	47.05 ± 12.71	0.135	0.179
E/Vp	2.01 ± 0.89	1.55 ± 0.68	0.353	0.000

Table 5. Colour M Mode Parameters - Flow Propagation Velocity before and after PCI

Grades of LV Diastolic Dysfunction

In our study 57% patients had normal diastolic function before PCI and 57% after PCI. Impaired diastolic dysfunction was seen in 41% patients before PCI and only in 17% after PCI. Pseudonormal pattern of diastolic function was seen in 43% patients before PCI but only 24% showed similar pattern even after PCI. Restrictive pattern of diastolic dysfunction was seen only in 6% patients before and 2% patients after PCI (LR ratio 83.95, p value 0.000).

		DD after PCI				Total
		Normal	I	II	III	
DD Before PCI	Normal	8	2	0	0	10
	I	39	2	0	0	41
	II	10	13	20	0	43
	III	0	0	4	2	6
Total		57	17	24	2	100

Table 6. Grades of LV Diastolic Dysfunction before and after PCI

DISCUSSION

The mean age of the patients was 52.04 ± 9.49 years ranging from 32 to 73 years. The majority (84%) of study population were males. Similar pattern of age distribution was reported by the studies done by R Sharma et al¹⁵ (mean Age 54.71 ± 19.90) and showed 79.5% patients were males. This study showed that smoking (43%) is the most frequent risk factor, followed by hypertension (38%), dyslipidaemia (30%), and diabetes mellitus (28%). Study by Sharma R et al showed a similar risk profile -smoking (49.3%), hypertension (40.2%), diabetes (37%).

Acute anterior wall STEMI was seen in 56% and acute inferior wall STEMI in 44%. The present study population had a mean Left ventricular ejection fraction of $58.91 \pm 7.99\%$, ranging from 45% to 76%. Coronary angiographic profile of the study population showed Single vessel disease in 75%, double vessel disease in 23% and Triple vessel disease in 2%.

In our study the change in E velocity and A velocity 48 hours after PCI was not found to be significant but the change in E/A, DT and Ar duration was found to be significant. Similar pattern of change in Mitral inflow parameters was seen in the study by Mandal et al.¹⁶

	Mean \pm SD Before PCI	Mean \pm SD After PCI	Significance (p value)	Mean \pm SD Before PCI	Mean \pm SD After PCI	Significance (p value)
E velocity	63.41 ± 19.93	71.51 ± 9.56	0.700	58.9 ± 11.8	78.1 ± 13.9	0.008
A velocity	75.93 ± 20.30	78.96 ± 24.18	0.124	76.6 ± 13.5	67.7 ± 15.2	0.001
E/A	1.03 ± 0.3	$0.98 \pm .24$	0.004	0.80 ± 0.24	1.2 ± 0.31	0.001
dur	111.57 ± 23.07	137.11 ± 13.69	0.000			
DT	210.15 ± 47.43	201.64 ± 28.15	0.000	245.6 ± 41.6	175.5 ± 31.5	0.001

Table 7. Mitral Inflow Parameters before and after PCI

Tissue Doppler Parameters

In our study, mean E' Velocity was 5.81 ± 1.65 cm/sec before PCI and 7.96 ± 1.95 cm/sec after PCI (p value 0.776). The mean A' Velocity was 8.05 ± 1.60 cm/sec before PCI and 8.02 ± 1.78 cm/sec after PCI (p value 0.000). The mean E/E' ratio was 10.17 ± 2.26 before PCI and 8.83 ± 1.70 after PCI (p value 0.000). The change in mitral annular tissue doppler parameters E' and A' with PCI is of no significance but the E/E' ratio showed a change with significance.

Isovolumetric Relaxation Time

In our study, mean IVRT was 85.69 ± 14.67 msec before PCI 79.18 ± 5.09 msec after PCI (p value 0.794). This implies that the change was of no significance.

Pulmonary Venous Flow Doppler Parameters

In our study pulmonary venous doppler parameters showed S<D in 6% patients before PCI and in 2% patients after PCI; S>D in 94% patients before PCI and in 98% patients after PCI. (LR ratio -11.79, p value -0.003). In our study, mean Ar-A dur 6.04 ± 32.93 msec before PCI 14.49 ± 31.83 msec

after PCI (p value 0.000). Both the parameters showed improvement within 48 hours of PCI, which is of significance.

Colour M Mode Parameters- Flow Propagation Velocity

In our study, mean Vp was 32.36 ± 7.59 cm/sec before PCI and 47.05 ± 12.71 cm/sec after PCI (p value 0.179) and mean E/Vp was 2.01 ± 0.89 before PCI and 1.55 ± 0.68 after PCI (p value 0.000). The Change in Vp within 48 hours of PCI was not significant but the change in E/Vp with PCI was significant.

Grades of LV Diastolic Dysfunction

In our study 57% patients had normal diastolic function before PCI and 57% after PCI. Impaired diastolic dysfunction was seen in 41% patients before PCI and only in 17% after PCI. Pseudo normal pattern of diastolic function was seen in 43% patients before PCI but only 24% showed similar pattern even after PCI. Restrictive pattern of diastolic dysfunction was seen only in 6% patients before and 2% patients after PCI (LR ratio 83.95, p value 0.000).

	Our Study		Mandal et al	
	Before PCI	After PCI	After PCI	After PCI
Normal	10%	57%	2%	79%
I	41%	17%	45%	1%
II	43%	24%	51%	19%
III	6%	2%	2%	1%

Table 8. Grades of LV Diastolic Dysfunction before and after PCI

On a whole, our study showed that the changes in E/A ratio, A wave duration, Deceleration time, A' velocity, E/E' ratio, Ar-A duration, and E/Vp showed significance but the changes in E velocity, A velocity, E' velocity, IVRT, and Vp did not showed no significance. Study by Bayat et al¹⁷ demonstrated improvement in regional LV early diastolic function after PCI and except pulmonary vein flow all of the left ventricular diastolic parameters such as Mitral E velocity, Mitral A velocity, mitral E' septal, E/E' ratio, IVRT and E/A ratio showed significant improvement after elective PCI. In a similar study Hashemi et al¹⁸ evaluated diastolic echocardiographic findings before PCI, 48 hours and 3 months after PCI in 30 patients with EF of more than 40% scheduled for elective PCI. Except mitral E/A ratio and mitral E' septal velocity all of the left ventricular diastolic parameters showed no significant improvement after 48 hours. Hashemi et al. provided significant improvement in Mitral A velocity moreover two previous parameters after 3 month. Tanaka et al.¹⁹ investigated the effects of PCI on global and regional left ventricular diastolic function, using (SR) imaging in 27 patients and finally concluded that Mitral E velocity, Mitral A velocity, DT and E/A did not reveal significant improvement after PCI.

Limitations

A limitation to the current study could have been the relatively small number of patients evaluated. The results in our study should be confirmed in larger prospective studies. The present study is a single center study only. Moreover, early assessment of LV diastolic function after PCI without

any follow up could be another limitation of this study. There were no sequential observations of the dynamic changes of LV functions, for the progression of coronary artery disease, morbidity and mortality in study population, as it was difficult for follow up. In the present study, we did not correlate with nT pro-BNP, strain and strain rate imaging due to non-availability of these diagnostic tests and equipment in our hospital. Another shortcoming may have been the fact that we did not consider clinical improvement along with echocardiographic parameters.

CONCLUSIONS

PCI can be an effective modality of treatment for diastolic dysfunction caused by myocardial ischemia and revascularization can restore diastolic properties of the heart after the insult in STEMI patients and thus prevent their progression to HFPEF.

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