Original Article

Acute effect of primary PCI on diastolic dysfunction recovery in anterior wall STEMI – A non-invasive evaluation by echocardiography

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ABSTRACT

Background: It is well established fact that acute coronary occlusion leads to diastolic dysfunction, followed by systolic dysfunction when myonecrosis occur. It is also proven that primary percutaneous coronary intervention (PPCI) is an excellent therapy for ST elevation myocardial infarction (STEMI) to improve outcomes. However there is a paucity of information on efficacy of PPCI in improving diastolic function. Evaluation of the role of PPCI in improving diastolic dysfunction is required.
Methods: 61 patients with first anterior wall STEMI who underwent PPCI to left anterior descending artery were included. Echocardiographic evaluation was performed within 24 h of PPCI and then on day 15, 3 months and 6 months after PPCI. We evaluated the prevalence of diastolic dysfunction after PPCI and its recovery during 6 months along with effect of duration of chest pain on diastolic function.
Results: 54.1% of patients had diastolic dysfunction after PPCI whereas it was only 21.3% after 6 months (p value < 0.001). Diastolic function indices like deceleration time, isovolumic relaxation time, E wave, A wave, E/A ratio, left atrial volume and index improved statistically from baseline to 6 months except mitral E/e′ ratio. As time required to achieve reperfusion increases (chest pain duration and D to B time) the incidence of residual diastolic dysfunction also increases (p value < 0.001). Patients with TIMI flow < III had more diastolic dysfunction (p value < 0.001).
Conclusions: Primary PCI improves diastolic dysfunction in patients with anterior wall STEMI over a period of 6 months. Time to achieve reperfusion and effectiveness of reperfusion have significant effect on diastolic dysfunction.

1. Introduction

Acute myocardial infarction (AMI) is a leading cause of heart failure, despite significant treatment advancements in recent years.[1] Development of new onset heart failure in patients with AMI is a poor prognostic sign with higher in hospital mortality.[2] A number of studies have indicated that both mortality and morbidity rates can be reduced in ST elevation myocardial infarction (STEMI) patients who receive timely primary percutaneous coronary intervention (PPCI).[3]

Although LV (left ventricle) systolic function is a well-known prognostic factor in patients with AMI, a growing body of evidence indicates that left ventricular diastolic dysfunction (LVDD), as assessed by Doppler echocardiography, is also an important predictor of patient outcomes after AMI.[4] Two-dimensional echocar-diography (2D ECHO) is the technique of choice to assess and monitor remodelling after AMI,[5] enabling a precise definition of magnitude and timing of the process.[6] In addition, Doppler echocardiography has provided evidence that serial changes in diastolic filling pattern may parallel the evolutionary changes in LV dimensions after AMI.[7] Recently, increasing attention has been devoted to diastolic function after AMI,[8] and there is growing evidence indicating a strong association between diastolic dysfunction and adverse outcome.

Shorter reperfusion times lead to better outcomes in patients with ST-elevation myocardial infarction.[9] The incidence of diastolic dysfunction increases if longer time is required to achieve flow because of increased myocardial stiffness. The factor that is most crucial to suppressing LV remodelling and improving diastolic dysfunction is restoring flow as rapidly as possible through shortening of the door-to-balloon time and achieving substantial reperfusion.[10]

Hence, the aims of this study is to evaluate prevalence and severity of diastolic dysfunction in post primary PCI patients of anterior wall STEMI presenting for the first time & to assess

improvement in diastolic dysfunction after 6 months of anterior wall STEMI. This study also aims to evaluate the effect of duration of chest pain on diastolic dysfunction in anterior wall STEMI patients.

2. Methods

2.1. Patient information

This study was a prospective observational study study conducted in Department of Cardiology at PGIMER, Dr. Ram Manohar Lohia Hospital, New Delhi. Consecutive 61 patients of anterior wall STEMI presenting for the first time at Dr. Ram Manohar Lohia Hospital, New Delhi from November 2015 to December 2016 were included in this study.

All patients with age > 18 years who presented in the emergency department within 12 h (hrs) of the onset of ischemic chest pain & fulfill the diagnostic criteria of acute Anterior wall STEMI as defined by the universal definition of Myocardial Infarction and eligible for primary PCI were included in this study. All patients with prior history of myocardial infarction (MI)/ coronary artery bypass graft (CABG) or PCI, significant stenosis (>50%) of any other coronary arteries other than left anterior descending artery (LAD), ST elevation on electrocardiogram (ECG) without obvious coronary artery diseases such as acute myocarditis, early repolarization, or Takotsubo Cardiomyopathy, Pericardial disease, Patients with AF, atrial flutter, or complete heartblock, Cardiogenic shock, Hypertension, Diabetes and valvular heart disease were excluded.

Detailed history, demographic characteristics & physical examination findings were recorded with special attention to the coronary risk factors like smoking, dyslipidemia, hypertension, diabetes, family history of Ischemic heart disease, previous myocardial infarctions etc. Routine biochemical investigations were done & pharmacotherapy started as per department's protocol. Primary PCI with stenting was done by standard technique on all eligible consenting patients as per department's protocol. 2D Echocardiography with colour Doppler assessment was done within 24 h after PPCI on Philips SONOS 5500 Echo machine. Door to balloon (D to B) time recorded from arrival at emergency department to first balloon inflation and duration of chest pain recorded from symptom onset to arrival to the emergency department.

2.2. *Assessment of diastolic dysfunction done as per American society of echocardiography (ASE) guidelines*[11]

Pulsed wave (PW) Doppler was performed in the apical 4chamber view within 3 mm sample volume at the tip of the mitral leaflets to obtain mitral inflow velocities to assess LV filling.

E (early diastolic)/**A** (late diastolic) - Using PW Doppler the peak E and A velocities were recorded, then ratio of E/A were calculated.

IVRT (isovolumic relaxation time) - derived by placing the cursor of Continuous wave (CW) Doppler in the LV outflow tract to simultaneously display the end of aortic ejection and the onset of mitral inflow.

Deceleration time (DT) - from peak of E wave to baseline (Fig. 1).

E/e'- PW tissue Doppler imaging (TDI) was performed in the apical views to acquire mitral annular velocities. The sample volume was positioned at or 1 cm within the septal and lateral insertion sites of the mitral leaflet.

Left atrial volume and left atrial volume index (LAVI) - The maximal left atrial (LA) volume was measured from the apical

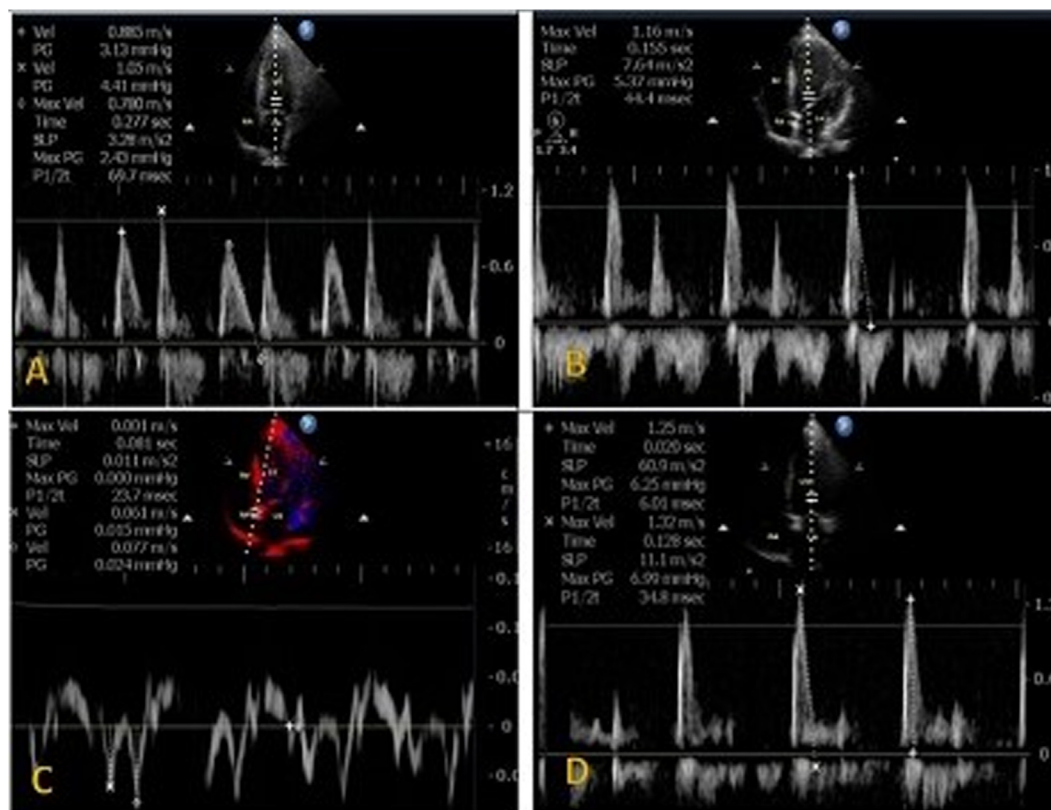**Fig. 1.** Echocardiogram of diastolic dysfunctions. A. Pulse Wave (PW) Doppler at mitral valve level showing grade I LVDD with DT-277 msec. B and C showing grade II LVDD – normal E/A ratio on PW Doppler at the mitral valve level, but tissue doppler at medial mitral annulus shows a' > e' with E/e'- 12. D. grade III LVDD with DT- 128 msec. LA- left atrium, LV-left ventricle, RA-right atrium, and RV-right ventricle.

four-chamber view by using the modified Simpson method in end-systole before mitral valve opening. The LAVI was obtained for all patients by dividing the LA volume by the body surface area.

In this study, LVDD was classified as

(A) Normal pattern: E/A ≥ 0.8, DT = 160–240 msec, IVRT- 70–90 msec and (E/e′) < 10.
(B) Grade I: E/A ≤ 0.8, deceleration time DT > 240 msec, IVRT- > 100 msec and (E/e′) < 10.
(C) grade II: E/A = 0.8–2, DT = 160–240 msec, IVRT – 70–100 msec, E/e′ ≥ 10–14,
(D) Grade III - E/A > 2, DT < 160 msec, IVRT < 70 msec and E/e′ > 14.

LV Volumes & LV ejection fraction (EF) measured by modified Simpson's method.

2.3. Follow up

Information on death and other major adverse cardiac events (MACEs) were obtained by clinical follow up visit and echocardiography was performed at 15 days, 3 months and 6 months of follow-up. Patient who did not come for clinical visit, they were contacted by phone for information on death and other MACE. Final follow up visit was performed at 6 months for primary endpoint registration. At each follow up visit we looked for clinical outcomes including recurrence of angina, acute coronary syndrome, and death.

3. Results

3.1. Patient characteristics

Out of 61 patients, 47 were males (77%) and 14 (33%) were females, mean age of the study group was 48.56 ± 11.64 years. The majority of the patients were > 50 years (range 25–72 yrs.). Most of the patients were smoker 37 (60.7%), Family history of CAD was present in 16 (26.2%) patients (Table 1).

Table 1
Baseline characteristics of patients.[a]

Mean age	48.56 ± 11.64
Male	47 (77%)
Smoking	37 (60.7%)
Family history	16 (26.2%)
D to B time < 90 min	32 (52.5%)
D to B time ≥ 90 min	29 (47.5%)
Mean time in D to B < 90 group (min)	54.25 ± 17.39
Mean time in D to B ≥ 90 group (min)	109.5 ± 12.55
Duration of chest pain	
<3 h	10 (16.3%)
3–6 h	14 (22.9%)
>=6 h	37 (60.7%)
TIMI III flow after PCI	48 (78.7%)

[a] Data are presented as mean ± SD or n (%), D to B time -Door to balloon time, TIMI- Thrombolysis in myocardial infarction.

Table 2
Grading of diastolic dysfunction at baseline.

Grade	Number of subjects (n)	Percentage (%)
Grade 0 – No dysfunction	28	45.9
Grade I	21	34.4
Grade II	7	11.5
Grade III	5	8.2
Total	61	100.0

3.2. Diastolic function at baseline

Initial ECHO was done at the time range of 2–20 h (mean-11.9 h). The prevalence of diastolic dysfunction at baseline, after Primary PCI was 54.1%. Twenty one (34.4%) patients had grade I, seven (11.5%) patients had grade II and five (8.2%) patients had grade III LV diastolic dysfunction (Table 2).

The door to balloon time was < 90 min (54.25 ± 17.39 min) in 52.5% of patients and ≥90 min (112.5 ± 12.55) in 47.5% of patients. The diastolic dysfunction was present in 31.2% of patients who had door to balloon < 90 min, whereas it was 79.3% in patients with door to balloon time ≥ 90 min (p value < 0.001). The diastolic dysfunction in patients who had chest pain < 3, 3–6 and ≥ 6 h was 10%, 14.3% and 81.3% (Table 3) respectively (p value < 0.001). The patients with LVEF < 40% had more diastolic dysfunction than patients with LVEF ≥ 40% (p value < 0.001). Thrombolysis in myocardial infarction (TIMI) III flow was achieved in 78.7% of patients after Primary PCI. All patients with TIMI flow less than grade III had diastolic dysfunction (100%) when compared to only 41.7% in patients who had TIMI III flow which is statistically significant (Table 4).

3.3. Diastolic function after 6 months

Diastolic dysfunction at baseline was 54.1%, and after 6 months only 21.3% of patient had diastolic dysfunction (Table 5), which is statistically significant (p value < 0.001).

The different grading of diastolic dysfunction also improved after 6 months (p value < 0.001). The various diastolic indices like E wave, A wave, E/A ratio, DT, IVRT, left atrial volume and LAVI improved statistically from baseline to 6 months except mitral E/e′ ratio (Table 6).

Table 3
Association between duration of chest pain and diastolic dysfunction at baseline.

Duration of chest pain	Diastolic dysfunction		Total
	Present	Absent	
< 3 h	1	9	10
	10.0%	90.0%	100.0%
3–6 h	2	12	14
	14.3%	85.7%	100.0%
>=6 h	30	7	37
	81.1%	18.9%	100.0%
Total	33	28	61
	54.1%	45.9%	100.0%

Chi- square value = 27.62, p value < 0.001 significant.

Table 4
Association of diastolic dysfunction with other variables at baseline.

Variables		Dysfunction present	P value
Age group (years)	<40	7 (58.3%)	0.194
	40–50	10 (41.2%)	
	>=50	16 (64%)	
Sex	Male	21 (44.7%)	**0.007**
	Female	12 (85.7%)	
Smoking	Present	23 (62.2%)	0.117
	Absent	10 (41.7%)	
D to B time	<90 min	10 (31.2%)	**<0.001**
	≥90 min	23 (79.3%)	
TIMI III flow	Present	20 (41.7%)	**<0.001**
	Absent	13 (100%)	
LVEF	<40	23 (62.2%)	**<0.001**
	≥40	10 (41.7)	

D to B time -Door to balloon time, LVEF- left ventricular ejection fraction, TIMI- Thrombolysis in myocardial infarction.

Table 5
Improvement in Diastolic dysfunction from baseline to sixth month.

Diastolic dysfunction base	Diastolic dysfunction at six months		Total
	Present	Absent	
Present	13	20	33
	39.4%	60.6%	100%
Absent	0	28	28
	0	100%	100%
Total	13	48	61
	21.3%	78.7%	100%

Chi-square = 14.02, p < 0.001 significant.

The mean LVEF improved to 46.19 ± 5.52 at 6 months from 41.92 ± 4.82 (p value < 0.001). Most of the patients were in New York Heart Association (NYHA) class I at six months and class II symptoms were more in patients with diastolic dysfunction (Table 7). The relationship between diastolic function and drug intake like dual antiplatelet, high dose statin, angiotensin converting enzyme inhibitor (ACEI)/ angiotensin receptor blockers (ARBs), β blocker and diuretics also had a significant effect on diastolic function at 6 months (Table 8).

During follow up stress evaluation (by tread meal test and Dobutamine stress ECHO) was required in 4 patients and all were negative. Indication for evaluation were chest pain in three patients and in one patient pre op evaluation for hysterectomy for fibroid uterus causing severe menorrhagia. None of the patient required angiography, because all the patients had single vessel disease and most of Co morbid conditions were excluded.

3.4. Statistics

The statistical evaluation was performed by computer analysis with SPSS Software (Statistical Package for the Social Sciences, version 16.0, SPSS Inc, Chicago, III, USA). Continuous variables were expressed as mean ± standard deviation (Mean ± SD). The comparisons between the baseline and follow-up diastolic indices were made using paired samples t-test. Chi-squared test (χ^2) was used to calculate the effect of chest pain on diastolic dysfunction and improvement in diastolic dysfunction after 6 months. Statistical significances were defined as the p value $\leq$ 0.05.

4. Discussion

4.1. Diastolic dysfunction recovery

Coronary occlusion causes initially diastolic dysfunction followed by systolic dysfunction in proportion to amount of myocar-

Table 6
Changes in diastolic indices from baseline to six month.

Variables	Time	Mean	t value	p value
DT	Baseline	195.67 ± 38.92	5.442	**<0.001**
	Sixth month	172.97 ± 23.85		
IVRT	Baseline	92.18 ± 14.74	3.953	**<0.001**
	Sixth month	83.84 ± 14.16		
E/A ratio	Baseline	1.226 ± 0.463	−6.601	**<0.001**
	Sixth month	1.588 ± 0.38		
E/e'	Baseline	7.5 ± 3.602	1.802	0.077
	Sixth month	6.91 ± 2.95		
EF	Baseline	35.92 ± 4.82	−17.21	**<0.001**
	Sixth month	39.19 ± 5.52		
LA volume	Baseline	55.6 ± 16	1.983	**0.026**
	Sixth month	52.3 ± 13		
LAVI	Baseline	31.2 ± 11	1.692	**0.047**
	Sixth month	29.3 ± 06		

A- peak transmitral late diastolic flow velocity, DT-deceleration time, IVRT- isovolumic relaxation time, E- peak transmitral early diastolic flow velocity, e′- mitral annular velocity, EF- Ejection fraction, LAVI- left atrial volume index.

Table 7
showing functional class of patients at six months.

NYHA class	Diastolic dysfunction present (13)	Diastolic dysfunction absent (48)	P value	Odds ratio
class I	9 (69.2%)	47 (97.9%)	**<0.001**	0.004 to 0.4
class II	4 (30.8%)	1 (2.08%)		
class III/IV	-/-	-/-		

NYHA- New York Heart Association.

Table 8
Shows diastolic function in relation to medication at baseline and six months.

Drugs		Diastolic dysfunction present	Diastolic dysfunction absent	P value	Odds ratio
DAPT	Baseline	33	28	**0.001**	1.8–9.2
	6 months	13	46		
Statin	Baseline	33	28	**<0.001**	1.9–9.6
	6 months	13	48		
ACEI/ARBs	Baseline	29	26	**<0.001**	2.0–11.0
	6 months	11	47		
βblocker	Baseline	25	24	**<0.001**	2.0–12.6
	6 months	9	44		
Diuretics	Baseline	30	14	**0.016**	1.2–8.4
	6 months	12	18		

ACEI/ARBs-angiotensin converting enzyme inhibitor/angiotensin receptor blockers, DAPT-dual antiplatelet.

dial necrosis.[12] Peri infarct area, are of chronic ischemia and scar also contributes to diastolic dysfunction in MI. If ischemia is resolved early and effectively these changes can be reverted.[13] Doppler echocardiography is a non-invasive technique that has been used to evaluate left ventricular relaxation and filling pattern.[14] Our prospective study shows that more than half of patients (54.1%) had diastolic dysfunction within 24hr of primary PCI in patients with first anterior STEMI who had single vessel disease on angiography. This diastolic dysfunction improved after 6 months of primary PCI from 54.1% to 21.3% (p value < 0.001). Most of the patients had impaired relaxation and all grades of diastolic dysfunction improved from baseline to 6 months. Diastolic function parameters such as deceleration time, isovolumic relaxation time, peak transmitral early diastolic flow velocity (E wave), peak transmitral late diastolic flow velocity (A wave), left atrial volume and LAVI improved significantly from baseline to 6 months except mitral E/e' ratio. Patients with LVEF < 40% and TIMI flow < III had more diastolic dysfunction than in patients with EF ≥ 40% and TIMI flow III respectively.

This finding is similar to Hashemi et al. who have shown that not all the diastolic parameters will improve after PCI.[15] They evaluated diastolic echocardiographic findings before PCI, 48 h and 3 months after PCI in 30 patients with EF more than 40%. All left ventricular diastolic parameters showed significant improvement after 48 h except mitral E/A ratio. Our results also did not show significant improvement in E/e', but it is close to significance (p- 0.077). One of the recent study in ACS patients who had diastolic dysfunction at baseline shown that in 82% diastolic dysfunction become normal after PCI.[16]

The mean LVEF at the baseline were 41.92 ± 4.82 which was improved to 46.19 ± 5.52 (p value < 0.001) in this study. This finding is similar to study by Remmelink et al.[17] They have shown that LVEF improved from 40 ± 17% to 54 ± 15% in patients presenting with first anterior wall STEMI within 6hrs of chest pain. Another prospective study has shown that LVEF improved from 48.8 ± 11.6% to 52.5 ± 11.5% at 6 months stenting to LAD.[18]

4.2. Effect of duration of chest pain and D to B time on diastolic dysfunction

Time to reperfusion is a known powerful prognostic marker in AMI patients undergoing reperfusion[19] and major consideration is given to minimizing the ischemic duration in order to improve survival following AMI.[20] Time to reperfusion is known to determine infarct size[21] and systolic function[22] but there are only a few studies regarding its effect on diastolic function and filling pressure.[23] In the present study duration of chest pain and door to balloon time on the baseline and its improvement were evaluated. The diastolic dysfunction in patients who had chest pain duration < 3, 3–6 and ≥ 6 h was 10%, 14.3% and 81.3% respectively (p value < 0.001). The diastolic dysfunction was present in 31.2% of patients who had door to balloon < 90 min, whereas it was 79.3% in patients with door to balloon time ≥ 90 min (p value < 0.001).

Our results are comparable to Shacham, et al which was a large cohort study done to evaluate whether delayed time to reperfusion would be associated with worse diastolic function.[24] Median time to reperfusion, defined as the time from symptom onset to reperfusion at the end of primary PCI, was 185 min (min). Early reperfusion group (≤185 min) had 57% diastolic dysfunction when compared to 80% in late (>185 min) reperfusion group (p < 0.001).

Shacham et al.[24] and Prasad et al.[23] study included patients with diabetes, hypertension, multivessel coronary artery disease and initial evaluation by ECHO done up to 3 days of MI which all may influence the diastolic function. Whereas our study excluded diabetes, hypertensive patients and included only single vessel LAD

disease. Early evaluation by ECHO done within 24 h of primary PCI and follow up was done by ECHO up to 6 months. All these factors potentially reduce the confounding factor causing diastolic dysfunction. We also studied the relationship between diastolic function and various cardiac drugs at baseline and six months, which most of the studies have not done.

The limitations of this studies are (1) this study included relatively small number of patients (n = 61). However, we still considered our study is significant because we have included only patients with chest pain up to 12 h who had the single vessel disease and excluded patients with diabetes and hypertension. (2) We have not measured left ventricular end diastolic pressure (LVEDP) because, we don't want to delay the reperfusion time. (3) Our study hasn't measured the tricuspid flow velocity, but we have measured LAVI which most the studies were lacking and also measured IVRT, DT and especially E/A ratio constitute the most significant parameters for an analysis of diastolic dysfunction.

5. Conclusion

This study concluded that left ventricular diastolic dysfunction occurs more commonly in patients with anterior wall STEMI. Doppler echocardiography is a simple and useful non-invasive method for assessing left ventricular diastolic dysfunction and recovery of diastolic dysfunction in anterior wall STEMI after primary PCI. Our finding strengthens a dedicated approach to shorten the Door to balloon time and achieving TIMI III flow is essential to preserve and improve diastolic dysfunction over a period of time in anterior wall STEMI patients with improved outcomes and thereby reducing future diastolic heart failure.

Declarations of interest

None.

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