Journal of Cardiovascular Disease Research

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

Effect of Percutaneous Coronary Intervention on Diastolic Function in Coronary Artery Disease

Prashank Ajmera[1], Deepak Maheshwari[2], Narendra Sharma[3], Himanshu Mahla[4], Navida Babbar[5], Sanjeev Kumar[6]

[1]Resident, Department of Cardiology, SMS Medical College Jaipur, India.
[2]Mbbs Md Dm, Department of Cardiology, SMS Medical College Jaipur, India.
[3]Resident, Department of Cardiology, SMS Medical College Jaipur, India.
[4]Mbbs Md Dm, Department of Cardiology, SMS Medical College Jaipur, India.
[5]Resident, Department of Cardiology, SMS Medical College Jaipur, India.
[6]Resident, Department of Cardiology, SMS Medical College Jaipur, India.

Received Date: 20/07/2023 Acceptance Date: 28/08/2023

Abstract

Background: Left ventricle (LV) diastolic dysfunction is often present in patients with significant coronary artery disease (CAD), even in the absence of regional or global LV systolic dysfunction. It has been suggested that abnormalities in LV diastolic function may actually precede LV systolic dysfunction, and therefore, serve as an early and sensitive marker of ischemia. This study aims to find improvement of diastolic function after percutaneous coronary intervention (PCI) in patients with stable or unstable angina. **Methods:** Prospective study, **Study setting:** Cardiology department of Tertiary care center. **Study duration:** from March 2020 to April 2022. **Study population:** patients with either stable or unstable CAD and with normal systolic function who underwent successful PCI. Two-dimensional transthoracic echocardiography was performed at baseline (before PCI) and repeated 48 h after PCI. LV diastolic parameters were compared before and after PCI using paired samples *t*-test results. **Sample size:** 309 **Results:** Mean age of study population was 56.65 ± 9.65 years. Majority of patients were male (63%). There was significant increase in mitral E-wave velocity (68.39 ± 17.52 cm/s vs. 71.64 ± 18.23 cm/s), E/A ratio (0.85 ± 0.29 vs. 0.89 ± 0.32), and early diastolic mitral annular motion (e') (7.02 ± 0.89 cm/s vs. 8.45 ± 0.86 cm/s) following PCI ($P < 0.0001$). Left atrial volume index (22.53 ± 4.43 vs. 20.81 ± 4.14), tricuspid jet velocity (0.91 ± 0.57 m/s vs. 0.76 ± 0.67 m/s), and E/e' ratio (10.03 ± 3.5 vs. 8.62 ± 2.61) decreased significantly following PCI ($P < 0.001$). **Conclusions:** This study suggests that LV diastolic filling pattern is modified significantly as early as 48 h after successful PCI. Improvement in impaired relaxation appears to be most likely explanation for these changes. PCI may be potential therapeutic target to improve diastolic function in patients with CAD.

Keywords: Coronary artery disease, diastolic dysfunction, echocardiography, percutaneous coronary intervention

Corresponding Author: Dr. Prashank Ajmera, Resident, Department of Cardiology, C-37 Blessing Gulati Flat 202 Bal Marg Tilak Nagar Jaipur -302004, India.
Email: ajmeraprashank@yahoo.in

Introduction

Coronary artery disease (CAD) and diastolic dysfunction are linked. This is because the major complications of CAD, i.e., ischemia is a major cause of diastolic dysfunction.[1]

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

In acute ischemia, diastolic dysfunction develops even before systolic dysfunction becomes apparent.

Although diastolic dysfunction is induced by myocardial ischemia, role of CAD in natural history of heart failure with preserved ejection fraction (HFpEF) has not been completely elucidated. It is plausible that treatment of CAD may be an important therapeutic target in treatment of HFpEF.

Our study aims to measure echocardiographic and Doppler parameters of left ventricular diastolic dysfunction among patients with CAD (stable and unstable angina) undergoing percutaneous coronary intervention (PCI) and its effect on left ventricular diastolic function.

Material and Methods

Study designs: Prospective study,

Study setting: Cardiology department of Tertiary care center.

Study duration: from March 2020 to April 2022

Study population: All patients with either stable or unstable CAD and with normal systolic function who underwent successful PCI. Two-dimensional transthoracic echocardiography was performed at baseline (before PCI) and repeated 48 h after PCI. LV diastolic parameters were compared before and after PCI using paired samples *t*-test results.

Sample size: 309

Inclusion criteria

1. All patients with either stable or unstable CAD and with normal systolic function who underwent successful PCI.

Exclusion criteria

1. Not willing to participate
2. Incomplete proforma

Sampling techniques:

All the participants were selected using random consecutive sampling method.

Study procedure

In this single-center hospital-based study, we enrolled 309 patients with either stable or unstable CAD and ≥1 diastolic dysfunction but with normal systolic function who underwent successful PCI over a period of 12 months (August 2018 to July 2019). Written informed consent was obtained from all patients. Complete revascularization was attempted in all patients when possible.

Patients with positive cardiac markers or systolic dysfunction (ejection fraction <50%), significant valvular heart disease (≥ moderate) on echocardiogram, bundle branch block, and atrial fibrillation were excluded from the study.

Detail clinical evaluation including presenting complaint, past and present medical records, and clinical examinations was performed of all patients. Transthoracic two-dimensional (2D) and Doppler echocardiographic studies were done at baseline (before PCI) and 48 h following successful PCI which was defined as residual diameter stenosis ≤10% following stent placement.[2]

Assessment of diastolic function and chamber quantification was made as per guidelines led down by the American Society of Echocardiography.[3] Significant valvular heart disease was excluded by 2D echocardiography and Doppler interrogation using PWD and CWD. Stages of diastolic dysfunction were characterized as Grade 1 – (mitral E/A <0.9, average E/E' = 10–14, peak tricuspid jet (TR) vel <2.8 m/s, and left atrial volume index [LAVi] <34 ml/m^2), Grade 2 – (mitral E/A = 0.9–2, average E/E' <10, peak TR vel <2.8 m/s, and LAVi-

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

normal or increased), and Grade 3 – (mitral E/A >2, average E/E' >14, peak TR vel >2.8 m/s, and LAVi >34 ml/m^2 normal).

Left atrium volume was calculated using standard biplane area-length method, at the end of systole avoiding inclusion of pulmonary veins and left atrial appendage.[3] Body surface area (BSA) was measured using Mosteller formula,[4] and LAVi was obtained by dividing LA volume with BSA.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation. Student's paired *t*-test was used to compare data before and after PCI. $P \leq 0.05$ was considered statistically significant.

Results and Observations

The study population comprised 309 patients with mean age of 56.65 ± 9.65 years. Majority of patients were male (*n* = 195; 63.1%). The baseline characteristics of the patients are shown in Table no 1

Table no. 1: Baseline characteristics of study population (*n*=309)

Variable	Male, *n* (%)	Female, *n* (%)	Total, *n* (%)
Study population	195 (63.1)	114 (36.9)	309
Mean age	55.66±10.16	58.34±8.5	56.65±9.65
Diastolic dysfunction	195	114	309 (100)
Grade 1	164	102	266 (86.1)
Grade 2	10	6	16 (5.1)
Grade 3	21	6	27 (8.8)
Vessel involvement	195	114	309 (100)
SVD	125	69	194 (62.8)
DVD	67	43	110 (35.6)
TVD	3	2	5 (1.6)

Echocardiographic parameters of diastolic function were recorded before and after PCI, as shown in Table no 2. There was no significant change in left ventricular end-diastolic and end-systolic dimension and left atrial dimension before and after PCI. There was a significant decrease in TR jet velocity along with LAVi in the post-PCI group compared to the pre-PCI group ($P \leq 0.001$).

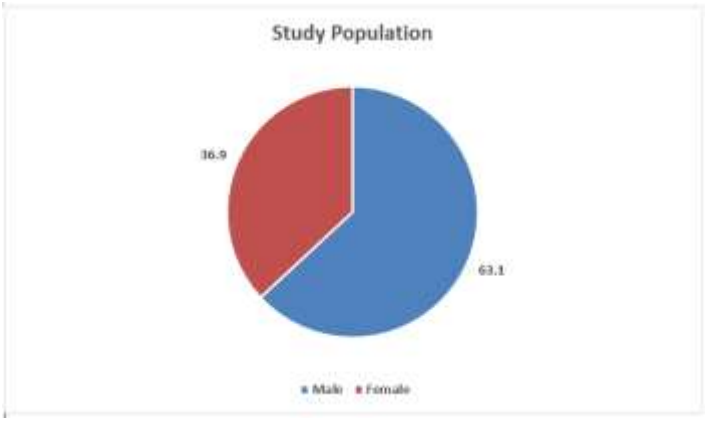

Figure 1

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

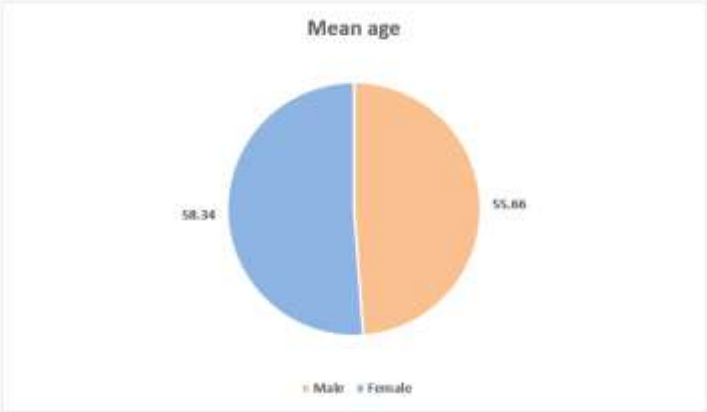

Figure 2

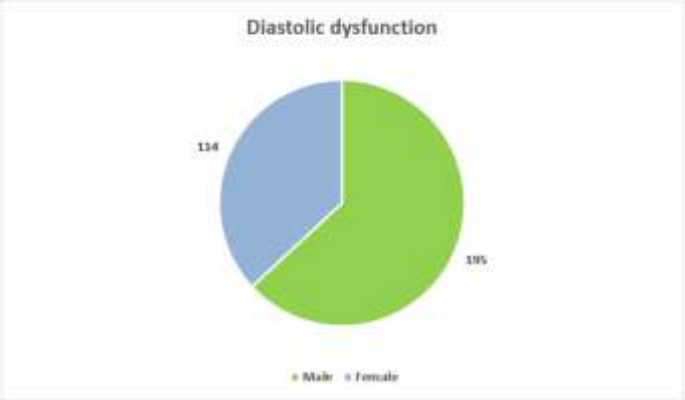

Figure 3

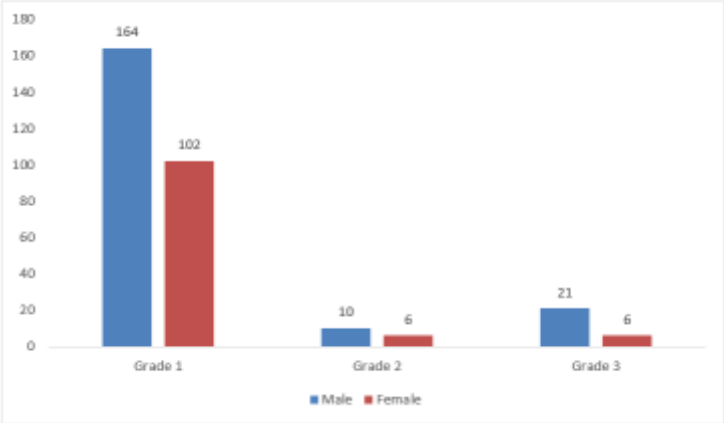

Figure 4

Table no 2: Pre-and post-percutaneous coronary intervention echocardiography finding of study population (*n*=309)

Parameters	Before PCI	After PCI	*P*	95% CI
LVIDd (cm)	4.46±0.28	4.45±0.27	0.102	−0.007
LVIDs (cm)	2.27±0.13	2.25±0.12	0.32	0.0002-0.039
LA (cm)	2.93±0.59	2.92±0.58	0.83	−0.184
LAVi (ml/m^2)	22.53±4.43	20.81±4.14	<0.0001	1.598-2.016
TR jet velocity (m/sec)	0.91±0.57	0.76±0.67	<0.0001	0.091-0.202
EF %	55.4±1.54	55.5±1.49	0.41	−0.478

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

E velocity (cm/s)	68.39±17.52	71.64±18.23	<0.0001	−3.46-−3.04
A velocity (cm/s)	81.97±9.11	81.18±9.06	0.28	0.70-0.88
E/A	0.85±0.29	0.89±0.32	<0.0001	−0.093
E’ (cm/s)	7.02±0.89	8.45±0.86	<0.0001	−0.19
A’ (cm/s)	9.51±1.3	9.49±1.3	0.88	−0.43
E/E’	10.03±3.5	8.62±2.61	<0.0001	0.922-1.898
IVRT (ms)	83.15±18.36	74.6±11.95	<0.0001	6.10-10.99
DT (ms)	202.48±30.3	195.38±27.34	0.002	2.53-11.66
Diastolic dysfunction, n (%)				

DT=Deceleration time, EF=Ejection fraction, IVRT=Isovolumic relaxation time, LA=Left atrium, LAVi=Left atrial volume index, LVIDd=Left ventricle internal diameter-diastolic, LVIDs=Left ventricle internal diameter-systolic, TR=Tricuspid regurgitation, CI=Confidence interval, PCI=Percutaneous coronary intervention

On the measurement of physiological indices, there was a significant increase in mitral E velocity and the ratio of early and late filling velocities before and after PCI but no significant difference in mitral A velocity before and after PCI ($P = 0.28$).

Among the parameters measured by pulse tissue Doppler, there was a significant increase in mean mitral E' septal velocity ($P < 0.0001$) and a decrease in mean E/E' after PCI) ($P < 0.0001$) in the post-PCI group. However, there was no significant difference in mitral A' septal velocity before and after PCI ($P = 0.28$). Mean isovolumetric relaxation time (IVRT) and deceleration time were significantly decreased after PCI ($P < 0.0001$ and $P = 0.002$, respectively).

Before PCI, all patients had diastolic dysfunction which was labeled as Grade 1 in 266 (86.1%), Grade 2 in 16 (5.1%), and Grade 3 in 27 (8.8%) patients. After PCI, significant improvement was noted as they reverted to normal in 107 (34.7%), Grade 1 in 178 (58.6%), Grade 2 in 14 (13.4%), and Grade 4 in 10 (3.3%) patients [Table 2]. Significant improvement was noted among patients with Grade 1 and Grade 3 dysfunction though Grade 2 dysfunction was insignificantly improved with PCI.

Discussion

Left ventricular diastolic dysfunction is often present in patients with significant CAD, even in the absence of regional or global left ventricular systolic dysfunction and actually preceeds left ventricular systolic dysfunction. Therefore, it may serve as an early and sensitive marker of ischemia.[2] Previous studies have shown severe LV diastolic dysfunction in 10% of patients with stable coronary heart disease with normal ejection fraction and no history of HF,[5] which was similar to our study, which showed Grade 2 or Grade 3 diastolic dysfunction in 13.9% of the patients. This population may be a potential target for the treatment of diastolic dysfunction by PCI.

There was no significant change in left ventricular dimension indices (end-diastolic and end-systolic) and left atrial dimensions before and after PCI. This was possibly because the reduction in dimensions and volume are time-related events. These chronic changes succeed improvement in diastolic function and may not be apparent in the first 48 h.

The two physiologic phases of diastole, active relaxation, and passive filling are both influenced by myocardial ischemia.[6] The diastolic process is a complex phenomenon that depends on a large number of interrelated factors.[6] Chamber stiffness and relaxation have contrasting influences on the Doppler-derived pattern of diastolic filling. Abnormal relaxation in the presence of normal chamber stiffness produces a decrease in both peak early velocity and early to atrial peak velocity ratio. Conversely, increasing chamber stiffness is

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

associated with an enhanced peak early filling velocity and decreased atrial contribution to filling.[7]

In this study, there was a significant increase in mitral E velocity and ratio of early and late filling velocities following PCI. Improvement in impaired relaxation after PCI appears to be the most likely mechanism to explain the changes in this group of patients. The increase in the ratio of early and late filling velocities post-PCI has been observed previously.[8] Castello *et al*. in a study, also noted significant changes in Doppler indexes after coronary angioplasty. Both early to late peak velocities and time velocity integral ratios increased significantly.[7]

The early rapid filling phase of diastole is associated with a brisk motion of mitral annulus as the chamber expands to accommodate the inflow of blood. This process is recorded and quantified using tissue Doppler E'. The ratio of mitral E wave to E' velocity (E/E') has been shown to accurately reflect the LV filling pressures.[9] In this study, a significant decrease in E/E' ratio after PCI reflects a decrease in filling pressures after revascularization. An increase in septal E' velocity after revascularization further supports the improvement in impaired relaxation of the left ventricle. A similar decrease in E/E' ratio after PCI has been observed in the previous studies.[10]

It has been established previously that a significant correlation exists between systolic pulmonary artery (PA) pressure and left atrial pressure, whether derived invasively or noninvasively. In the absence of pulmonary pathology, an increased systolic PA pressure is suggestive of increased leukocyte alkaline phosphatase (LAP) and is a marker of diastolic dysfunction.[3] We observed a significant decrease in TR jet velocity in the post-PCI group, reflecting the decrease in LAP.

Similarly, LAVi which an established parameter for assessment of diastolic dysfunction assessment[3] and a strong predictor of mortality[11,12] was also significantly reduced in the post-PCI group, which was in concordance with the previous studies showing the benefit of PCI for improvement in diastolic dysfunction in patients with acute MI.[9] IVRT measurement provides insight into the rate of early diastolic left ventricular relaxation. When relaxation is prolonged, the mitral valve opening is delayed, and IVRT is increased.

Conversely, when left atrial pressure is elevated mitral valve opening will occur earlier, and IVRT will be shortened. Thus, impaired relaxation lengthens IVRT.[3] Prolonged deceleration time in mild diastolic dysfunction reflects impaired relaxation of the ventricle in early phase of the diastole as chamber stiffness increases deceleration time shortens.[3] In this study, the decrease in IVRT and deceleration time after PCI was statistically significant, which reflects an improvement in impaired relaxation following revascularization.

Our study demonstrated that improvement in diastolic dysfunction following successful PCI was noted within 48 h. This finding is concordant with study by Diller *et al*.,[13] who reported such improvement within 24 h of successful revascularization. Our study had patients with stable as well as unstable ischemic disease, and improvement was noted across both groups of patients.

Ischemia is cornerstone behind such diastolic dysfunction whose mechanism is different in both subgroups. In patients with stable angina, it is supply demand mismatch (normal supply with increased demand) over a long period which leads to hibernation with diastolic dysfunction. If the element of ischemia is eliminated by PCI, diastolic dysfunction improves. Diastole is an active phenomenon which is impaired because of ischemia, and PCI improves this by increasing the blood flow. In patients with acute coronary syndrome, ischemia is precipitated by decreased perfusion in the background of normal demand. In these subset of patients, PCI by ameliorating element of impaired supply improves the diastolic dysfunction.[14] It has been demonstrated that balloon occlusion of coronary artery for 50 s

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

produces diastolic dysfunction apparent which lasts at least 30 min after balloon deflation. Revascularization by PCI improved diastolic dysfunction in patients with ischemic cardiomyopathies.[15]

Conclusion

PCI leads to significant improvement in physiological parameters of diastolic function in patients with stable and unstable angina. Therefore, this population may be a potential target for the treatment of diastolic dysfunction.

Bibliography

1. Ohara T, Little WC. Evolving focus on diastolic dysfunction in patients with coronary artery disease. *Curr Opin Cardiol.* 2010;25:613–21.
2. Bristow JD, Van Zee BE, Judkins MP. Systolic and diastolic abnormalities of the left ventricle in coronary artery disease.Studies in patients with little or no enlargement of ventricular volume. *Circulation.* 1970;42:219–28.
3. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF, 3rd, Dokainish H, Edvardsen T, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: An update from the American Society of Echocardiography and the European Association of Cardiovascular imaging. *J Am Soc Echocardiogr.* 2016;29:277–314.
4. Mosteller RD. Simplified calculation of body-surface area. *N Engl J Med.* 1987;317:1098.
5. Ren X, Ristow B, Na B, Ali S, Schiller NB, Whooley MA. Prevalence and prognosis of asymptomatic left ventricular diastolic dysfunction in ambulatory patients with coronary heart disease. *Am J Cardiol.* 2007;99:1643–7.
6. Nishimura RA, Housmans PR, Hatle LK, Tajik AJ. Assessment of diastolic function of the heart: Background and current applications of Doppler echocardiography. Part I. Physiologic and pathophysiologic features. *Mayo Clin Proc.* 1989;64:71–81.
7. Castello R, Pearson AC, Kern MJ, Labovitz AJ. Diastolic function in patients undergoing coronary angioplasty: Influence of degree of revascularization. *J Am Coll Cardiol.* 1990;15:1564–9.
8. Hashemi SR, Motamedi M, Khani M, Hekmat M, Gachkar L, Rezaeefar A. Evaluation of the effect of elective percutaneous coronary intervention as a treatment method on the left ventricular diastolic dysfunction in patients with coronary artery disease. *J Tehran Heart Cent.* 2010;5:194–8.
9. Appleton CP, Hatle LK, Popp RL. Demonstration of restrictive ventricular physiology by Doppler echocardiography. *J Am Coll Cardiol.* 1988;11:757–68.
10. Salehi N, Saidi M, Rai A, Najafi F, Javeedannejad S, Babanejad M, et al. Effect of percutaneous coronary intervention on left ventricular diastolic function in patients with coronary artery disease. *Glob J Health Sci.* 2015;8:270–6.
11. Patel DA, Lavie CJ, Milani RV, Ventura HO. Left atrial volume index predictive of mortality independent of left ventricular geometry in a large clinical cohort with preserved ejection fraction. *Mayo Clin Proc.* 2011;86:730–7.
12. Khan MA, Yang EY, Zhan Y, Judd RM, Chan W, Nabi F, et al. Association of left atrial volume index and all-cause mortality in patients referred for routine cardiovascular magnetic resonance: A multicenter study. *J Cardiovasc Magn Reson.* 2019;21:4.
13. Diller GP, Wasan BS, Thom SA, Foale RA, Hughes AD, Francis DP, et al. Evidence of improved regional myocardial function in patients with chronic stable angina and apparent normal ventricular function-A tissue Doppler study before and after

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 08, 2023

percutaneous coronary intervention. *J Am Soc Echocardiogr*. 2009;22:177–82.
14. Wang J, Abraham TP, Korinek J, Urheim S, McMahon EM, Belohlavek M. Delayed onset of subendocardial diastolic thinning at rest identifies hypoperfused myocardium. *Circulation.* 2005;111:2943–50.
15. Carluccio E, Biagioli P, Alunni G, Murrone A, Leonelli V, Pantano P, et al. Effect of revascularizing viable myocardium on left ventricular diastolic function in patients with ischaemic cardiomyopathy. *Eur Heart J.* 2009;30:1501–9.