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Short Term Effect of Primary Percutaneous Coronary Intervention on Left Ventricular Diastolic Function in Diabetic Patients

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ABSTRACT

Background: Diabetes mellitus (DM) is associated with microvascular dysfunction and myocardial stiffness, which may impair post-reperfusion recovery following ST-segment elevation myocardial infarction (STEMI). While primary percutaneous coronary intervention (PCI) improves systolic outcomes, its short-term impact on left ventricular (LV) diastolic function in diabetic patients remains uncertain.

Objective: To evaluate the short-term effect of primary PCI on LV diastolic function in diabetic versus non-diabetic patients presenting with acute STEMI.

Patients and Methods: A prospective cohort study included 100 STEMI patients undergoing primary PCI at Tanta University and Nasser Institute Hospitals between July 2023 and July 2024. Patients were divided into two equal groups: diabetics and non-diabetics. Comprehensive clinical, laboratory, and echocardiographic assessments were performed before PCI and at three-month follow-up, including E/A ratio, E/e′ ratio, isovolumic relaxation time (IVRT), deceleration time (DT), and diastolic dysfunction (DD) grade.

Results: Diabetic patients were older (61.9 ± 6.2 vs. 54.6 ± 5.7 years, $p<0.001$) and had higher BMI, heart rate, and blood pressure. After three months, both groups showed significant improvements in LV systolic function (EF: from 49.9 ± 7.3 to 55.2 ± 7.2% in diabetics, and from 51.3 ± 4.0 to 57.0 ± 3.5% in non-diabetics; $p<0.001$ each). Diastolic indices changed significantly within groups (E/A decreased, E/e′, IVRT, and DT increased; $p<0.001$), but intergroup differences were non-significant. DD Grade III emerged in 22% of diabetics and 12% of non-diabetics ($p=0.39$).

Conclusion: Primary PCI improved LV systolic function in both diabetic and non-diabetic STEMI patients; however, early diastolic recovery remained limited, with comparable short-term outcomes between groups.

Keywords: Diabetes mellitus; ST-segment elevation myocardial infarction; Percutaneous coronary intervention; Diastolic dysfunction; Echocardiography.

INTRODUCTION

Coronary artery disease (CAD) remains the leading cause of morbidity and mortality worldwide, with an increasing burden among patients with diabetes mellitus (DM). Diabetes confers a two- to four-fold higher risk of developing CAD and is associated with more extensive and diffuse atherosclerosis, endothelial dysfunction, and microvascular disease [1]. These pathophysiological alterations accelerate myocardial ischemia and impair both systolic and diastolic performance, predisposing diabetic patients to adverse cardiovascular events and higher post-infarction mortality. Despite advances in reperfusion therapy, diabetes continues to adversely affect the short- and long-term outcomes of acute myocardial infarction (AMI) [2]. Primary percutaneous coronary intervention (PCI) is currently the gold standard for the management of ST-segment elevation myocardial infarction (STEMI), providing superior myocardial salvage and survival benefits compared to thrombolytic therapy. However, the beneficial effects of primary PCI may be attenuated in diabetic patients due to microvascular obstruction, impaired collateral circulation, and abnormal myocardial metabolism [3,4]. These factors hinder complete myocardial reperfusion and limit left ventricular (LV) functional recovery even after successful revascularization. While numerous studies have addressed systolic function recovery post-PCI, the effect on LV diastolic function—an early and sensitive marker of myocardial performance—remains less well explored, particularly in diabetic populations [5,6].

Diastolic dysfunction is one of the earliest manifestations of diabetic cardiomyopathy, occurring even in the absence of overt systolic impairment. It results from increased myocardial stiffness, interstitial fibrosis, and altered calcium handling, all of which impair LV relaxation and filling. Following acute ischemic injury, these abnormalities may be exacerbated, leading to elevated LV filling pressures and progression to heart failure with preserved ejection fraction (HFpEF) [7,8]. Evaluating diastolic function in the early post-PCI period is therefore clinically relevant, as it provides insights into myocardial recovery, risk stratification, and potential therapeutic targets for optimizing outcomes in diabetic patients [9].

Given these considerations, the present study was designed to evaluate the short-term effect of primary percutaneous coronary intervention on left ventricular diastolic function in diabetic patients presenting with ST-segment elevation myocardial infarction, comparing their echocardiographic changes with those of non-diabetic counterparts.

PATIENTS AND METHODS

Study Design and Participants

This prospective cohort study was conducted on one hundred patients presenting with acute STEMI who underwent primary PCI. The study was performed

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at the Cardiology Departments of Tanta University Hospitals and the Nasser Institute Hospital between July 2023 and July 2024.

Study Population and Grouping

Eligible patients were adults aged between 18 and 65 years of both sexes who presented with acute chest pain associated with electrocardiographic ST-segment elevation and/or elevated cardiac biomarkers consistent with MI. Only patients eligible for urgent percutaneous coronary angiography were included.

Exclusion criteria comprised valvular or pericardial diseases, AF or flutter, pericardial effusion, or any condition that could interfere with accurate assessment of LV function. Patients refusing to participate or failing to provide written consent were also excluded. Based on diabetic status, the enrolled subjects were categorized into two equal groups: fifty diabetic patients representing the study group and fifty non-diabetic patients serving as the control group.

Clinical Assessment

All participants underwent detailed clinical evaluation and comprehensive data collection at baseline. Demographic characteristics including age, sex, weight, and height were recorded, and body mass index (BMI) was calculated. Medical history focused on cardiovascular risk factors such as hypertension, diabetes mellitus, dyslipidemia, and smoking status, along with the duration and control of diabetes when applicable. A thorough general and cardiovascular examination was conducted for each patient. Vital signs including heart rate, blood pressure, respiratory rate, and temperature were measured, and systemic examination of the chest and abdomen was performed to detect signs of heart failure or pulmonary congestion.

Laboratory Investigations and Electrocardiography

Upon admission, all patients underwent laboratory testing including complete blood count, liver enzymes, renal function tests, and cardiac biomarkers such as troponin I and CK-MB to confirm myocardial injury. Glycated hemoglobin (HbA1c) was measured to assess glycemic control and confirm diabetic status. Standard twelve-lead electrocardiography (ECG) was obtained to verify ST-segment elevation, localize the infarct-related artery, and identify rhythm abnormalities.

Echocardiographic Assessment

Transthoracic echocardiography (TTE) was performed for all patients before and after PCI using a high-resolution ultrasound system equipped with a phased-array transducer. All echocardiographic examinations adhered to the recommendations of the American Society of Echocardiography (ASE)[10]. Patients were examined in the left lateral decubitus position, and standard imaging views—including parasternal long- and short-axis, apical four- and two-chamber, and subcostal views—were obtained. Pulsed-wave Doppler at the mitral leaflet tips was used to measure early (E) and late (A) diastolic filling velocities, from which the E/A ratio was derived.

Tissue Doppler imaging (TDI) was performed at the septal and lateral mitral annulus to record myocardial velocities (e′ and a′), and the average E/e′ ratio was calculated as an estimate of LV filling pressures. Additional parameters such as isovolumic relaxation time (IVRT), deceleration time (DT), left atrial volume index (LAVI), and left ventricular ejection fraction (LVEF) were also recorded. Echocardiographic evaluations were repeated 48–72 hours after PCI to assess early changes and again at three-month follow-up to evaluate short-term recovery.

Definition and Grading of Diastolic Dysfunction

Diastolic function was graded according to the criteria proposed by **Nagueh *et al.*** [10], integrating mitral inflow pattern, tissue Doppler indices, tricuspid regurgitant (TR) velocity, and left atrial volume index. Grade I (impaired relaxation), Grade II (pseudonormal pattern), and Grade III (restrictive filling) were defined based on standard thresholds for E/A ratio, E/e′ ratio, TR velocity, and LAVI. All echocardiographic measurements were performed by a single experienced cardiologist blinded to the patients' diabetic status and clinical data, and the average of three consecutive cardiac cycles was used for analysis to minimize variability.

Follow-Up Protocol

All patients were followed for three months after the index procedure. Follow-up assessments included repeat clinical evaluation, ECG, and echocardiographic examination to monitor LV systolic and diastolic function. Data were collected prospectively and stored in a dedicated database for analysis. Patient confidentiality was maintained through coded identifiers to ensure anonymity.

Ethical considerations

The study was done after being accepted by the Research Ethics Committee, Tanta University. All patients provided written informed consents prior to their enrolment. The consent form explicitly outlined their agreement to participate in the study and for the publication of data, ensuring protection of their confidentiality and privacy. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Data Management

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 29 (IBM, Chicago, IL, USA). Categorical variables were expressed as frequencies and percentages, whereas continuous variables were presented as mean ± standard deviation. Comparisons between diabetic and non-diabetic groups were conducted using the chi-square test for categorical variables and the independent Student's t-test or Mann–Whitney U test for continuous variables, as appropriate. Paired t-tests were used to compare pre- and post-PCI values within each group, and the McNemar test was applied for categorical paired comparisons. A two-tailed p-value of less than 0.05 was considered statistically significant.

RESULTS

Diabetic patients were significantly older, had higher body weight, and body mass index. Heart rate, systolic and diastolic blood pressures were significantly elevated among diabetics compared with non-diabetics. Hemoglobin level and platelet count were significantly lower in diabetics. Liver enzymes, ALT and AST, as well as troponin were significantly higher in diabetic patients compared with non-diabetics **(Table 1)**.

In contrast, no significant differences were observed between the two groups regarding sex, hypertension, chronic liver disease, height, respiratory rate, total leucocytic count, serum creatinine, serum urea, or CK-MB **(Table 1)**.

Table 1: Baseline clinical, anthropometric, and laboratory characteristics of the study population

		Total (n = 100)	Diabetics (n = 50)	Non- diabetics (n = 50)	P-value
Age (years)		58.2 ± 6.97	61.9 ± 6.22	54.6 ± 5.66	**<0.001***
Sex	Male	81 (81.0)	39 (78.0)	42 (84.0)	0.41
	Female	19 (19.0)	11 (22.0)	8 (16.0)	
Hypertension		17 (17.0)	10 (20.0)	7 (14.0)	0.42
Chronic liver disease		15 (15)	10 (20.0)	5 (10.0)	0.16
Weight (kg)		84.8 ± 9.42	89.0 ± 8.57	80.6 ± 8.33	**< 0.001***
Height (m)		1.73 ± 0.08	171.2 ± 8.32	174.9 ± 7.77	**0.024***
BMI (Kg/m²)		28.6 ± 4.51	30.5 ± 3.91	26.6 ± 4.24	**< 0.001***
HR (beat/minute)		78.7 ± 7.48	83.9 ± 2.73	73.6 ± 7.19	**< 0.001***
SBP (mmHg)		134 ± 7.75	137.2 ± 2.51	131.1 ± 9.80	**< 0.001***
DBP (mmHg)		77.0 ± 8.13	79.8 ± 7.28	74.2 ± 8.04	**< 0.001***
RR (cycles/minute)		19.4 ± 1.55	18.6 ± 1.07	18.3 ± 1.08	0.4
Hemoglobin (g/dL)		12.8 ± 1.62	12.02 ± 1.55	13.66 ± 1.24	**< 0.001***
TLC (×10³/mm³)		10.5 ± 2.40	10.29 ± 2.87	10.78 ± 2.13	0.57
PLT (×10³/mm³)		261 ± 61.4	237.92 ± 55.42	284.76 ± 58.45	**< 0.001***
S. Cr (mg/dL)		0.89 ± 0.19	0.94 ± 0.24	0.80 ± 0.09	0.11
S. urea (mg/dL)		41.6 ± 6.4	44.14 ± 5.63	39.00 ± 6.93	0.12
ALT (IU/L)		41.7 ± 1.7	44.08 ± 8.94	39.28 ± 3.67	**0.04***
AST (IU/L)		42.8 ± 2.1	54.40 ± 4.17	41.24 ± 5.17	**0.03***
Troponin I (ng/mL)		0.63 ± 0.01	0.78 ± 0.07	0.48 ± 0.07	**< 0.001***
CK-MB (IU/L)		78.2 ± 5.3	82.52 ± 6.31	73.96 ± 5.06	0.42

Values are presented as mean ± standard deviation or number (percentage), n: number, BMI: Body mass index, HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, RR: Respiratory rate, TLC: Total leucocytic count, PLT: Platelet count, S. Cr: Serum creatinine, S. urea: Serum urea, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, CK-MB: Creatine kinase–myocardial band, *: Significant P-value.

Both groups showed significant improvements in several echocardiographic parameters from baseline to three-month follow-up. Left ventricular ejection fraction significantly increased in diabetics and non-diabetics, while end-diastolic and end-systolic volumes significantly decreased. Significant improvements were also observed in wall motion abnormalities. Diastolic parameters demonstrated notable changes, with the E/A ratio significantly decreasing after PCI and IVRT, DT, and E/e′ ratio all significantly increasing at follow-up. Left atrial and right atrial volumes and their indices significantly increased after PCI across both groups. Interventricular septal thickness also showed a significant rise at follow-up **(Table 2)**.

Regarding diastolic dysfunction grading, the overall prevalence of dysfunction changed significantly after PCI, with an increase in Grade III restrictive pattern from 0% at baseline to 22% in diabetics and 12% in non-diabetics **(Table 2)**. No significant intergroup differences were observed for RAV, RAVI, LAV, LAVI, RVD, LVD, EF, IVSd, EDV, ESV, E/A ratio, IVRT, DT, E/e′ ratio, TRV, and DD grade **(Table 2)**.

Table 2: Echocardiographic parameters before and three months after primary PCI in diabetic and non-diabetic patients

	Total (n = 100)	Diabetics (n = 50)	Non-diabetics n = 50)	P-value 1
RAV (mL)				
Baseline	30.7 ± 3.54	28.66 ± 2.75	29.01 ± 2.91	0.54
After 3 months	31.7 ± 3.04	30.58 ± 2.67	31.10 ± 3.01	0.08
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
RAVI (mL/m²)				
Baseline	25.6 ± 2.95	23.87 ± 2.30	24.33 ± 2.48	0.34
After 3 months	30.4 ± 3.86	31.36 ± 3.58	30.76 ± 3.90	0.09
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
LAV (mL)				
Baseline	46.9 ± 3.53	48.00 ± 4.35	47.03 ± 1.94	0.33
After 3 months	48.4 ± 3.79	51.04 ± 3.36	52.60 ± 1.94	0.11
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
LAVI (mL/m²)				
Baseline	34.0 ± 3.54	35.89 ± 2.90	34.04 ± 3.07	0.21
After 3 months	35.4 ± 6.11	39.70 ± 5.90	38.91 ± 1.82	0.34
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
RVD (mm)				
Baseline	37.6 ± 1.94	38.88 ± 1.44	38.40 ± 1.54	0.06
After 3 months	35.6 ± 1.74	37.18 ± 1.44	36.90 ± 1.54	0.23
P-value 2	0.32	0.54	0.11	
LVD (mm)				
Baseline	50.1 ± 5.28	50.88 ± 6.14	49.32 ± 4.16	0.14
After 3 months	49. ± 4.18	49.88 ± 5.14	49.32 ± 4.16	0.14
P-value 2	0.13	0.11	0.34	
EF (%)				
Baseline	50.6 ± 5.90	49.96 ± 7.33	51.32 ± 3.96	0.25
After 3 months	56.1 ± 5.73	55.24 ± 7.22	57.04 ± 3.54	0.12
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
WMA				
Baseline	59 (59)	27 (54)	32 (64)	0.31
After 3 months	27 (27)	15 (30)	12 (24)	0.49
P-value 2	**< 0.001***	**< 0.015**	**< 0.001***	
IVSd (cm)				
Baseline	1.01 ± 0.11	0.82 ± 0.12	0.85 ± 0.11	0.07
After 3 months	1.21 ± 0.16	1.44 ± 2.21	0.99 ± 0.10	0.14
P-value 2	**0.04***	**< 0.001***	**< 0.001***	
EDV (mL)				
Baseline	53.3 ± 4.59	54.70 ± 4.33	53.90 ± 4.44	0.11
After 3 months	43.8 ± 16.9	45.42 ± 5.81	42.19 ± 19.77	0.06
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
ESV (mL)				
Baseline	38.5 ± 4.08	40.14 ± 4.45	39.88 ± 2.89	0.06
After 3 months	31.1 ± 12.6	33.32 ± 5.14	32.96 ± 13.72	0.33
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
E/A ratio				
Baseline	1.33 ± 0.39	1.37 ± 0.41	1.34 ± 0.37	0.66
After 3 months	0.69 ± 0.20	0.60 ± 0.26	0.65 ± 0.05	0.06
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
IVRT (ms)				
Baseline	85.0 ± 16.5	86.14 ± 18.08	81.48 ± 14.98	0.16
After 3 months	94.8 ± 20.8	91.1 ± 22.8	92.12 ± 2.1	0.09
P-value 2	**0.004***	**< 0.001***	**< 0.001***	
DT (ms)				

	Total (n = 100)	Diabetics (n = 50)	Non-diabetics n = 50)	P-value 1
Baseline	194.3 ± 36.8	201.98 ± 26.11	196.58 ± 43.88	0.09
After 3 months	224.2 ± 45.4	220.0 ± 52.6	227.3 ± 30.0	0.11
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
E/e′ ratio				
Baseline	9.83 ± 2.50	9.56 ± 2.66	10.10 ± 2.32	0.28
After 3 months	14.18 ± 0.88	14.4 ± 1.0	14.2 ± 0.7	0.12
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
TRV (m/s)				
Baseline	2.41 ± 0.37	2.49 ± 0.34	2.39 ± 0.39	0.06
After 3 months	2.54 ± 0.45	2.69 ± 0.38	2.59 ± 0.46	0.08
P-value 2	**< 0.001***	**< 0.001***	**< 0.001***	
DD grade				
Baseline				
No	31 (31)	14 (28)	17 (34)	0.79
Grade 1	49 (49)	26 (52)	23 (46)	
Grade 2	20 (20)	10 (20)	10 (20)	
Grade 3	0 (0)	0 (0)	0 (0)	
After 3 months				
No	37 (37)	18 (36)	19 (38)	0.44
Grade 1	46 (46)	21 (42)	25 (50)	
Grade 2	0 (0)	0 (0)	0 (0)	
Grade 3	17 (17)	11 (22)	6 (12)	
P-value 2	**0.01***	**0.02***	**0.09***	

Data are expressed as mean ± standard deviation or number (percentage). P-value 1 denotes inter-group comparison between diabetics and non-diabetics, while P-value 2 represents intra-group comparison between baseline and three-month follow-up values, n: number, RAV: Right atrial volume, RAVI: Right atrial volume index, LAV: Left atrial volume, LAVI: Left atrial volume index, RVD: Right ventricular diameter, LVD: Left ventricular diameter, EF: Ejection fraction, WMA: Wall motion abnormalities, IVSd: Interventricular septal thickness in diastole, EDV: End-diastolic volume, ESV: End-systolic volume, IVRT: Isovolumic relaxation time, DT: Deceleration time, E/e′ ratio: Ratio of early transmitral flow velocity to early diastolic mitral annular velocity, TRV: Tricuspid regurgitant velocity, DD grade: Diastolic dysfunction grade, PCI: Percutaneous coronary intervention, *: Significant P-value.

After three months of follow-up, the pattern of change in diastolic dysfunction did not significantly differ between diabetic and non-diabetic patients (P = 0.39). Among diabetics, 50% showed no change, 28% exhibited improvement, and 22% experienced worsening of diastolic function, whereas in non-diabetics, 54% had no change, 34% improved, and 12% worsened.

DISCUSSION

DM is tightly linked to adverse coronary outcomes through diffuse atherosclerosis, microvascular dysfunction, and myocardial remodeling that together blunt post-reperfusion recovery [11]. Because diastolic dysfunction often precedes systolic decline in DM and predicts HFpEF, contrasting our short-term post-PCI results with contemporary literature clarifies whether diabetes meaningfully modifies early reverse remodeling and diastolic trajectories after reperfusion.

We studied 100 STEMI patients (50 diabetics; 50 non-diabetics). Diabetics were older with higher weight/BMI, higher HR/SBP/DBP, lower Hb, lower PLT, higher ALT/AST, and higher troponin I. Across three months, both groups showed improved EF and reduced EDV/ESV; diastolic indices shifted (lower E/A; higher IVRT, DT, E/e′), atrial volumes rose, and Grade III diastolic dysfunction emerged, yet intergroup differences remained largely non-significant, including the distribution of change categories in diastolic dysfunction.

Regarding age and sex distribution, supporting our findings, **Lee *et al.*** [12] reported diabetics with CAD were significantly older than non-diabetics and that males exceeded 50% in both groups. **Wang *et al.*** [13] likewise found higher age among diabetics. **Aronson *et al.*** [14] quantified this gap (63 ± 11 vs. 59 ± 12 years, P = 0.008) with male predominance in both cohorts. In contrast, the ISCHEMIA analysis by **Newman *et al.*** [15] and **Andersson *et al.*** [16] observed no age or sex differences between groups.

Regarding hypertension prevalence, conversely, several large series showed higher hypertension in diabetics: **Lee *et al.*** [12] reported 64% vs. 49.4% (P < 0.001); **Aronson *et al.*** [14] 65% vs. 43% (P < 0.001), **Newman *et al.*** [15] 82.6% vs. 70.8% (P <

0.001); and **Andersson *et al.*** [16] 71% vs. 45% (P = 0.03).

Regarding obesity, our findings of higher body weight and BMI among diabetics were consistent with those of **Lee *et al.*** [12], who reported significantly higher BMI and weight in diabetics (P = 0.002), and with **Wang *et al.*** [13], who similarly demonstrated greater obesity indices. **Andersson *et al.*** [16] further quantified this difference, showing a BMI of 29 ± 5 versus 25 ± 3 (P < 0.001), supporting our observation of increased obesity in diabetics. In terms of hemodynamic parameters, **Wang *et al.*** [13] found higher heart rate and blood pressures among diabetics, and **Aronson *et al.*** [14] also documented elevated admission HR, SBP, and DBP in diabetics, reinforcing the hemodynamic burden of diabetes. Regarding hematologic parameters, **Lee *et al.*** [12] confirmed lower hemoglobin levels in diabetics (P = 0.005) with no differences in TLC or PLT, partially aligning with our results except for our finding of lower platelet counts among diabetics.

Concerning renal function, our neutral between-group results agree with **Lee *et al.*** [12] and **Aronson *et al.*** [14] (sCr 1.1 ± 0.4 vs. 1.0 ± 0.4 mg/dL, both within normal ranges), whereas **Newman *et al.*** [15] demonstrated a lower eGFR in diabetics (78 vs. 81 mL/min/1.73 m²; P < 0.001) and **Wang *et al.*** reported similarly impaired renal indices in diabetic patients. For liver enzymes, they found no significant ALT or AST differences between diabetics and non-diabetics, in contrast to our observed higher transaminase levels among diabetics. Finally, supporting our findings of higher troponin levels, they also demonstrated greater elevations in troponin, CK-MB, and NT-proBNP in diabetic compared with non-diabetic patients during acute coronary syndromes [13].

Regarding conventional echocardiographic remodeling (EF, EDV, ESV) after PCI**, i**n line with our early improvements in both groups, **Melika** [17] showed EF increased while ESV/EDV decreased after PCI irrespective of diabetes status. Conversely, **Allam *et al.*** [18] and **Celik *et al.*** [19] noted attenuated reverse remodeling in diabetics, with smaller mean changes in EDV, ESV, and EF versus non-diabetics.

Regarding diastolic indices (E/A, IVRT, DT, E/e′, atrial volumes), consistent with elements of our trajectory, **Subramaniyan *et al.*** [20] observed significant improvements from baseline to six months in E, A, E/A, DT, IVRT, LAV, and LAVI after PCI. **Melika** [17] and **Bayat *et al.*** [21] also reported favorable shifts in IVRT, DT, E/e′, and E/A over follow-up. In contrast, **Celik *et al.*** [19] found diabetes dampened changes in DT and IVRT compared with non-diabetics. **Allam *et al.*** [18] reported no intergroup differences in E/A or DT over three months, aligning with our lack of between-group separation despite within-group change.

Regarding diastolic dysfunction grading and its evolution, **Newman *et al.*** [15] supported our neutral intergroup comparisons after revascularization, reporting no significant differences in systolic or diastolic outcomes by diabetes status. **Andersson *et al.*** [16] found broadly comparable grades between groups but a higher E/e′ in diabetics (9.9 ± 5.8 vs. 7.0 ± 1.6; P = 0.01), suggesting higher filling pressures despite similar categorical grading. In contrast to our three-month pattern, **Subramaniyan *et al.*** [20] documented a decline in overall diastolic dysfunction prevalence from 54.1% at baseline to 21.3% at six months (P < 0.001). Further contrary evidence comes from **Wang *et al.*** [13], who, using CMR, reported worse diastolic grades in diabetics, and from **Aronson *et al.*** [14], who observed higher rates of diastolic dysfunction and more frequent moderate–severe EF reduction in diabetics after PCI (P < 0.001).

This study has several limitations. First, the relatively small sample size and single-center design may limit the generalizability of the findings. Second, the follow-up period of three months was relatively short and may not fully capture the long-term course of diastolic recovery after reperfusion. Third, despite using comprehensive echocardiographic assessment, more advanced techniques such as strain imaging or cardiac MRI were not utilized to quantify subtle diastolic changes. Finally, potential confounders such as medication use, glycemic variability, and infarct size were not extensively analyzed, which could have influenced the observed outcomes.

CONCLUSIONS

In patients with acute STEMI undergoing primary PCI, both diabetic and non-diabetic groups demonstrated significant short-term improvement in systolic function, whereas diastolic function showed limited recovery with no significant difference between groups. These findings suggest that diabetes does not independently hinder early diastolic improvement following successful reperfusion. Larger, multicenter studies with longer follow-up and advanced imaging modalities are warranted to better delineate the trajectory of diastolic remodeling in diabetic patients post-PCI.

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