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Prognostic Value of Speckle Tracking Echocardiography on Left Ventricular Function after Percutaneous Coronary Stenting to Significant Proximal Left Anterior Descending Artery Stenosis

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ABSTRACT

Background: Left ventricular systolic dysfunction (LVSD) and diabetes mellitus (DM) usually coexist, which raises patient morbidity and mortality. It is crucial to determine how isolated cases of a significant proximal stenosis of the left anterior descending artery in diabetic patients are affected by changes in the left ventricular function assessed by echocardiography after percutaneous coronary stenting.

Objective: This study aimed to compare the early detection of changes in left ventricular systolic and diastolic performance in diabetic and non-diabetic patients using conventional and 2D speckle tracking echocardiography following successful percutaneous coronary intervention (PCI) with drug-eluting stenting of isolated stenosis of the proximal left anterior descending (LAD) artery.

Patients and methods: A prospective cross-sectional study was conducted on 82 patients presenting with chest discomfort and myocardial ischemia at Al-Ahrar Teaching Hospital and Zagazig University Hospitals. Patients were categorized into two groups based on the occurrence of major adverse cardiovascular events (MACE) following percutaneous coronary intervention (PCI): Group I (MACE occurred) and Group II (MACE did not occur), stratified by diabetes status. All participants underwent electrocardiography (ECG) and echocardiography.

Results: Pre-global longitudinal strain (GLS) and Delta GLS are significant predictor for MACE in diabetic and non-diabetic patients in univariate and multivariate logistic regression analysis.

Conclusion: Patients with ischemic heart disease who had impaired systolic function improved significantly in diastolic dysfunction and all diastolic filling pattern measures after PCI.

Keywords: Speckle tracking echocardiography, Ventricular function, PCI, Stenosis, Cross sectional study, Zagazig university.

INTRODUCTION

Patient with diabetes mellitus (DM) have more extensive atherosclerosis and more plaque than non-diabetic patient. This makes the diabetic patient more risky for acute coronary syndrome. Diabetics account for 15-20% of patients undergoing coronary revascularization **(1).** The long-term effects involving coronary bypass surgery and percutaneous coronary intervention (PCI) are less desirable in diabetic patients. This outcome is explained by a more rapid advancement a greater rate of atherosclerosis and restenosis (2).

Despite the use of stents has improved both short-term and long-term outcomes for diabetic patients, the results of PCI are still less favorable for these individuals than for those without the disease. The effectiveness of PCI in diabetic patients is predicted to be significantly improved by Drug-eluting stents are one of the new angioplasty techniques. As a result, there are more reasons to consider angioplasty in diabetics **(3).**

According to Echo Doppler studies, people mortality from all causes is higher in people with asymptomatic left ventricular (LV) diastolic dysfunction **(4).** Moderate to severe diastolic dysfunction and mild dysfunction were linked to 8.3- and 10.2-fold higher death rates, respectively. Diastolic or systolic heart failure symptoms patients' overall mortality rates are quite comparable. In the past, transmitral Doppler flow velocities have been employed to assess modifications in LV diastolic function after PCI **(5).**

It has been demonstrated that the speckle tracking echocardiograph has the potential to identify subclinical LV systolic dysfunction that is concealed by the change in longitudinal strain in asymptomatic diabetic individuals with excellent LVEF **(6). Choi *et al*.** **(7)** a good predictor of stable ischemic cardiomyopathy among asymptomatic patients without anomalies in the wall motion is a lower longitudinal strain value. Furthermore, longitudinal strain is a predictor of LV remodeling and adverse outcomes such heart failure when it is evaluated right after reperfusion therapy.

Mehrpooya *et al*. **(8)** following PCI on the left anterior descending (LAD) artery, both diabetics and non-diabetics had their systolic and diastolic echocardiographic function assessed. They demonstrated that when compared to angioplasty on other arteries, PCI on the LAD caused a greater rise in LVEF. Additionally, the presence of DM had no detrimental effects on the improvement of LVEF following LAD angioplasty. Other systolic and diastolic differences between people with

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diabetes and those without diabetes weren't particularly looked at, though better outcomes and functional abilities are related to improvements in LV systolic and diastolic function.

The current study aimed to compare diabetic and non-diabetic patients using conventional and 2D speckle tracking echocardiography to examine the early detection of changes in left ventricular systolic and diastolic performance following successful PCI with drug-eluting stenting of isolated stenosis of the proximal LAD artery.

PATIENTS AND METHODS

A cross sectional prospective study was carried out on 82 patients with typical chest discomfort and myocardial ischemia who admitted to Al-Ahrar Teaching Hospital and Zagazig University Hospitals. Diabetic and non-diabetic patients exhibited typical chest discomfort and had myocardial ischemia confirmed by ECG or conventional Echo and 2D speckle tracking and the study included diabetic and non-diabetic individuals who had isolated significant proximal LAD coronary artery stenosis and underwent successful angioplasty with DES of at least 70% in a recent angiography.

Exclusion criteria: Congenital heart disease, significant valvular heart disease, cardiomyopathy, atrial fibrillation, cancer, collagen vascular diseases, amyloidosis, first diagonal branch lesions, complete LAD blockage, stent restenosis, or multiple vessel coronary artery disease.

All patients underwent thorough history taking that covered the duration between chest discomfort and reperfusion as well as cardiovascular risk factors like smoking. A complete clinical examination, including general and local heart examination, assessment of height, weight, and body mass index (BMI), hypertension (HTN), DM, dyslipidemia, and family history, measurement of the levels of plasma glucose, CBC, serum creatinine, CKMB, troponin and total cholesterol, ECG and echocardiography.

ECHO: Two examinations were done, the first was done with admission time and the 2nd after 3 months from PCI using the Vivid 6 system, a resting echocardiography investigation was carried out (GE Ultrasound, Horten, Norway). The following measurements were taken:

(A) Ejection fraction (EF):

- Using the modified Simpson biplane approach, the LV volumes and ejection fraction (EF) were evaluated from the apical 4-views.
- It is calculated also from the formula: **EF= [(EDV-ESV)/EDV] ×100**
- Normally it is 50- 70 % [9].

After 3 month follow up: 2D LV volumes were reassessed with the result of contractility change from the first study and three months in form of:

(B) Pulsed Doppler of mitral inflow:

- 4CV Apical Place a 1-3 mm the volume of the PW Doppler sample between the mitral leaflets' tips with the Doppler beam pointed in the direction of the inflow, and take the following measurements:
- The peak late A wave (peak late atrial filling rate) in cm/s. Maximum early E wave velocity (maximum early diastolic velocity) in cm/s.
- E/A ratio (relative contribution of early and late atrial filling [10].

(C) Global longitudinal strain (GLS) using the speckle tracking technique:

It was performed only at admission time using the same echo machine Vivid 9 system (GE Ultrasound, Horten, Norway).

- By measuring the ratio of the change in shape to the original length of an object, STE is a non-Doppler echocardiographic method for assessing the longitudinal strain (LS) of LV segments. Standard apical views are used for STE, and strain is automatically measured at frame rates between 60 and 90 frames per second [11].

Using the apical three strain gauges, the longitudinal strain of each LV segment was measured with 4, and 2 chamber views. The best frame for endocardial identification was used to trace the LV endocardial border in each of the three apical views, then manually adjusting the automatically generated region of interest to the thickness of the myocardium. Segments were dropped if tracking quality remained persistently bad even when the region of interest was changed. The deformation parameters were then automatically produced into quantitative and graphical bulls eye representations for each LV segment. Then, end-systole was determined by using the space of time between each R wave and this time point, which was automatically timed. The apical long-axis picture was used to determine the aortic valve closure [12].

The average value of global longitudinal strain "***GLS"*** obtained from averaging the strain values of whole LV segments at rest was calculated automatically, where values less than (-20%) is considered abnormal [13].

Angiographic finding: Invasive coronary angiography was performed on all patients by experienced interventional cardiologists using a standard transfemoral approach. Sheaths were inserted into the right femoral artery, and guiding catheters were carefully advanced into the left and right coronary ostia. Contrast dye was injected into each catheter to visualize the coronary arteries. Quantitative analysis of the angiograms was performed using an automated edge detection system (GE Medical Systems/Siemens), which provided detailed measurements of vessel diameters and lesion dimensions. Despite the use of this automated system, the final

determination of lesion severity was expressed as percentage luminal diameter stenosis, was based on independent visual assessment by two experienced interventional cardiologists, ensuring a comprehensive evaluation.

If a patient's left main coronary artery narrowed by 50%, their circumflex artery by 70%, or their LAD by 70%, they were classified as having significant angiographic coronary artery disease [(14)]. A combination of mortality, stroke, reinfarction, heart failure, arrhythmia, and revacularization is known as MACE. All of our patients were divided into two groups based on the frequency of major adverse cardiovascular events (MACE): Group I consisted of patients who experienced MACE in both diabetic and non-diabetic patients following PCI, and group II consisted of patients in which MACE did not occur in either group following PCI.

Ethics Approval: The Institutional Review Board of Zagazig University's Faculty of Medicine granted ethical approval for this study. All participants provided written informed consent. The Declaration of Helsinki, the World Medical Association's guideline of ethics for research involving humans, was followed in the conduct of this study.

Statistical analysis

Version 22.0 of the Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, USA) was used to introduce and statistically analyze the acquired data. Numbers and percentages were used to define qualitative data. When necessary, the Chi-squared test and Fisher's exact test were employed to compare categorical variables. The Kolmogorov-Smirnov test was used to check the normality of quantitative data. The mean and standard deviation (SD) of a normal distribution of variables were used to compare groups using the Wilcoxon signed ranks, Mann-Whitney U test, independent sample t-test, and paired t-test. A P value $\leq$ 0.05 was deemed statistically significant.

RESULTS

The patients had MACE divided to 8 diabetic and 6 non-diabetic after PCI to Proximal LAD (Table 1).

Table (1): MACE among diabetic and non-diabetic patients in three months

Variable	Diabetic Patients (n=41)	Non Diabetic Patients (n=41)
	MACE (n=8)	MACE (n=6)
	Positive (No)	Positive (No)
Stroke	1	1
HF	3	2
Re-infarction	1	1
Arrhythmia	2	3
REVAS	2	0
Death	0	0

There was no significant difference between MACE and no MACE in diabetic patients as regards age and sex in diabetic and non-diabetic groups (Table 2).

Table (2): Demographic characteristics in relation to MACE among diabetic and non-diabetic patients

Variable	Diabetic patients (n=41)				P-value	Test
	No MACE (n=33)		MACE (n=8)			
Sex	**No**	**%**	**No**	**%**	0.713 (NS)	**X^2** 0.150
Male	19	57.6	4	50		
Female	14	42.4	4	50		
Age (Years)						
Mean ± SD	47.03 ± 4.66		50.6 ± 6.18		0.158 (NS)	**U** 65.500
Median (Range)	47 (36-58)		51 (43-58)			
Variable	**Non diabetic patients (n=41)**				**P-value**	**Test**
	No MACE (n=35)		**MACE (n=6)**			
Sex	**No**	**%**	**No**	**%**	1.000 (NS)	**X^2** 0.004
Male	17	48.6	3	50.0		
Female	18	51.4	3	50.0		
Age (Years)						
Mean ± SD	50 ± 4.43		51.33 ± 7.03		0.669 (NS)	**U** 61.000
Median (Range)	51 (40-59)		49.5 (42-61)			

There was no significant difference between occurred in diabetic patients as regards clinical data: BMI, laboratory readings as HB, TLC, PLT, RBS, CKMB, troponin, creatinine and total cholesterol in diabetic and non-diabetic patients (Table 3).

Table (3): Clinical Data in relation to MACE among diabetic and non-diabetic patients

Variable	Diabetic patients (n=41)		P-value	Test
	No MACE (n=33)	MACE (n=8)		
	Mean ± SD Median (Range)	Mean ± SD Median (Range)		
BMI	28.48 ± 3.03 29 (23-34)	27.75 ± 1.8 27.5 (25-30)	**0.517 (NS)**	**U 121.00**
HB	10.18 ± 2.2 10 (6-14)	11.25 ± 2.37 11.5 (7-14)	**0.232 (NS)**	**U 178.00**
TLC	8.54 ± 2.35 9 (3-12)	7.62 ± 2.61 7.5 (3-11)	**0.338 (NS)**	**U 139.500**
PLT	326.27 ± 65.10 331 (150-443)	318.6 ± 45.9 315 (260-388)	**0.756 (NS)**	**U 119.00**
RBS	140.75 ± 16.7 142 (94-173)	143.25 ±21.41 149.5 (111-166)	**0.722 (NS)**	**U 97.500**
CKMB	133.87 ± 87.9 121 (16-315)	129.25 ± 66.13 116 (45-240)	**0.980 (NS)**	**U 174.500**
Troponin T	4.69 ± 2.24 4 (1-10)	3.12 ± 2.16 3 (0-6)	**0.081 (NS)**	**U 151.00**
creatinine	1.21 ± 0.69 1 (0-2)	1.25 ± 0.46 1 (1-2)	**0.885 (NS)**	**U 116.00**
Total cholesterol	221.45 ± 38.5 217 (139-304)	208.37 ±38.65 211.5 (132-266)	**0.394 (NS)**	**U 89.500**
Variable	**Non diabetic patients (n=41)**		**P-value**	**Test**
	No MACE (n=35)	**MACE (n=6)**		
	Mean ± SD Median (Range)	**Mean ± SD Median (Range)**		
BMI	24.43 ± 3.2 24 (18-32)	23.33 ± 3.9 23 (19-30)	**0.542 (NS)**	**U 82.500**
HB	11.8 ± 2.04 12 (8-16)	12.66 ± 2.33 13 (10-15)	**0.385 (NS)**	**U 60.400**
TLC	6.97 ± 1.7 7 (4-10)	6.83 ± 0.75 7 (6-8)	**0.849 (NS)**	**U 69.00**
PLT	333.85 ± 57.8 330 (204-464)	366.33 ± 54.58 362 (299-428)	**0.208 (NS)**	**U 63.00**
RBS	111.97 ± 13.58 112 (87-139)	108.8 ± 12.84 113 (84-119)	**0.602 (NS)**	**U 53.00**
CKMB	127.97 ± 84.5 124 (21-310)	114.0 ± 123.5 70 (15-342)	**0.729 (NS)**	**U 65.500**
Troponin T	2.77 ± 4.8 3 (-9 - 12)	-0.166 ± 4.66 0 (-5 - 5)	**0.173 (NS)**	**U 50.500**
creatinine	1.2 ± 0.41 1 (1-2)	1.16 ± 0.41 1 (1-2)	**0.859 (NS)**	**U 50.00**
Total cholesterol	140.85 ± 29.37 137(68-225)	145 ± 34.26 152 (99-183)	**0.789 (NS)**	**U 70.00**

There was statistically significant increase in EF before and after intervention among MACE and no MACE patients by 18.91% and 13.82%, respectively, with P-value <0.001 (Table 4).

Table (4): Change in EF in non-diabetic patients in both groups, pre and post PCI in three months

Item	Ejection Fraction		% of change	P-value	Test
	Pre EF	Post EF			
No MACE (n=41)					
Mean ± SD	42.46 ± 3.34	50.49 ± 3.46	**↑18.91 %**	0.000* (HS)	-341.0
(Range)	(41-57)	(44-56)			
MACE (n=41)					
Mean ± SD	46.31 ± 3.6	52.46 ± 3.4	**↑13.82 %**	0.000* (HS)	45.00
(Range)	(40-65)	(42-60)			

Wilcoxon Signed Ranks Test. *P < 0.05 is significant. HS: Highly Significant

There was statistically significant increase in GLS before and after intervention among non-diabetic and diabetic patients by -12.03% and -12.73% respectively, with P-value <0.001 (Table 5).

Table (5): Relation between GLS and diabetic patients pre- and post-PCI in three months

Item	GLS		% of change	P-value	Test
	Pre GLS	Post GLS			
No DM (N=41)					
Mean ± SD	-12.97 ± 1.31	-14.53 ± 2.14	**↑- 12.03 %**	0.001* (HS)	570
(Range)	(-16.0 - -8.0)	(-19.0 - -10.0)			
DM (N=41)					
Mean ± SD	-13.43 ± 2.89	-15.14 ± 0.85	**↑-12.73 %**	0.001* (HS)	447
(Range)	(-20.0 - -8.0)	(-18.0 - -14.0)			

Wilcoxon Signed Ranks Test. *P <0.05 is significant. HS: Highly Significant

There was statistically significant decrease in GLS after intervention among non-MACE in diabetic patients by -14.2% and there was no significant change in GLS among MACE in diabetic patients. Also, there was MACE occurrence in correlation to increased GLS in non-diabetic patients but there was no significant change in GLS among MACE patients (Table 6).

Table (6): GLS in relation to MACE among diabetic and non-diabetic patients

Item	Diabetic patients (n=41)		% of change	P-value	Test
	Pre GLS	Post GLS			
No MACE (n=33)					
Mean ± SD	-13.24 ± 3.13	-15.12 ± 0.89	**↑- 14.2 %**	0.002* (HS)	-301
(Range)	(-20 - -8)	(-18 - -14)			
MACE (n=8)					
Mean ± SD	-14.25 ± 1.48	-15.25 ± 0.71	**↑-7.02 %**	0.131 (NS)	180
(Range)	(-17 - -12)	(-16 - -14)			
Item	**Non-diabetic patients (n=41)**		**% of change**	**P-value**	**Test**
	Pre GLS	**Post GLS**			
No MACE (n=35)					
Mean ± SD	-12.94 ± 1.32	-14.74 ± 2.17	**↑- 13.91 %**	0.000* (HS)	644
(Range)	(-16 - -8)	(-19 - -10)			
MACE (n=6)					
Mean ± SD	-13.16 ± 1.32	-13.33 ± 1.63	**↑-1.29 %**	0.915 (NS)	-156
(Range)	(-15 - -12)	(-15 - -11)			

Pre-GLS and Delta GLS are significant predictor for MACE in diabetic patients in univariate and multivariate Logistic regression analysis. In univariate analysis, P in pre-GLS 0.014 and in Delta GLS was 0.019. In multivariate analysis, P in pre-GLS was 0.018 and in Delta GLS was 0.015. Also, pre-GLS and Delta GLS were significant predictor for MACE in non-diabetic patients in univariate and multivariate Logistic regression analysis. In univariate analysis, P in pre-GLS was 0.031 and in Delta GLS was 0.024. In multivariate analysis, P in pre-GLS was 0.015 and in Delta GLS was 0.024 (Table 7).

Table (7): Univariate and multivariate Logistic regression analysis for MACE prediction (n=41) in diabetic patients

Items	Univariate		#Multivariate	
	P-value	**OR (95%CI)**	**P-value**	**OR (95%CI)**
GLS in diabetic				
Pre	0.014*	1.383 (1.068 – 1.792)	0.018*	1.272 (1.056 – 1.652)
Post	0.185	2.507 (0.644 – 9.760)		
Delta GLS	0.019*	0.744 (0.580 – 0.953)	0.015*	0.620 (0.422 – 0.912)
Item	**Univariate**		**#Multivariate**	
	P-value	**OR (95%CI)**	**P-value**	**OR (95%CI)**
GLS in non-diabetic				
Pre	0.031*	3.127 (1.11 – 8.811)	0.015*	3.025 (1.11 – 8.811)
Post	0.103	0.633 (0.366 – 1.097)		
Delta GLS	0.024*	0.532 (0.308 – 0.921)	0.024*	0.532 (0.308 – 0.921)

DISCUSSION

In this study we found that there was statistically significant increase in ejection fraction before and after intervention among MACE and no MACE patients by 18.91% and 13.82% respectively (P-value <0.001).

Ahmed [15] found that EF before PCI was 47.8±4.1% by M-mode method and 43.5% (SD 3.9) by modified Simpson's method, and after PCI, the mean EF increased to 57.4% (SD 2) by M-mode method and to 52.8% (SD 2.2) by modified Simpson's method. According to M-mode and modified Simpson's techniques, there was a highly significant statistical difference between the mean EF before and after PCI (P<0.001). **Mahgoub *et al.*** [16] reported a highly significant increase in left ventricular ejection fraction (LVEF), measured by 2D echocardiography using Simpson's method, following the procedure (p < 0.001).

In our study, we found that there was statistically significant increase in GLS before and after intervention among non-diabetic and diabetic patients by -12.03% and -12.73% respectively (P-value <0.001).

Ahmed [15] found that mean GLS by STE before PCI was −7.0% (SD 2.1), and after PCI, the mean GLS increased to −13.9% (SD 1.7). Regarding the mean GLS by STE, there was a significantly significant statistical difference between before and after PCI (P<0.001). **Mahgoub *et al.*** [16] reported significant improvements in 2D global longitudinal strain (GLS) in both diabetic and non-diabetic patients following the procedure (p<0.001). In diabetic patients, mean GLS improved from -14.1% (SD 2.7) pre-procedure to -15.4% (SD 2.8) post-procedure, with a mean difference of 1.4% (SD 0.7). In non-diabetic patients, mean GLS improved from -16.5% (SD 1.8) to -18.6% (SD 2.2), with a mean difference of 2.1% (SD 0.8)

This study demonstrated that PCI preserved left ventricular ejection fraction (LVEF), significantly improved left ventricular (LV) function in coronary artery disease (CAD) patients with or without diabetes mellitus (DM), and prevented heart failure. While, conventional echocardiography showed no overt LV systolic dysfunction, GLS, S', and Tei index assessments, which revealed subclinical LV impairment in stable CAD patients and documented LV functional improvement post-PCI.

Choi *et al.* [7] revealed that despite the localized wall motion or normal resting, repeated ischemia events of the LV myocardium caused by substantial coronary stenosis might decrease longitudinal performance. This may help to explain why GLS and S', the two longitudinal

measurements, are sensitive indicators of ischemia and diminished LV function. **Biering-Sorensen *et al.*** [17] reported that patients with stable CAD and an LVEF and those without greater than 50% were found to have different GLS scores. Patients having at least one coronary artery stenosis of at least 70% had GLS values that were considerably lower than those of patients without myocardial stenosis. How useful tissue Doppler echocardiography indices are in the diagnosis of coronary artery disease (CAD) was evaluated by **Agarwal *et al.*** [18] who discovered that LV S' was much lower in people with CAD than in people without CAD.

In the current investigation, the global LV function was evaluated using the LVEF, the systolic longitudinal function was evaluated using the GLS and S', and the myocardial performance measured as a sum of systoles and diastoles was evaluated using the Tei index. This made it possible to sensitively and completely evaluate the LV function both before and after PCI.

GLS readings were significantly greater after PCI than before it in all of the study's subjects. Data on GLS alterations in patients with stable CAD and intact LVEF following elective PCI are scarce. **Ryo *et al.*** [19] demonstrated improvements in LV function in 35 patients 1 month after PCI as measured by GLS. After an acute MI, **Antoni *et al.*** [20] used GLS to evaluate LV function over the course of a year of follow-up. Patients are classified as improving when their GLS increases by less than 10%. Despite not having experienced a MI and having less LV dysfunction, all patient groups in the current trial experienced a 17% increase in GLS following PCI.

The Tei index was drastically lowered by PCI. Compared to individuals with CAD alone, diabetic patients with CAD benefited more from a modification in the Tei index. There were few and only patients with MI included in the Tei index data prior to and following optional PCI. According to the currently known research, in stable CAD, the Tei index has not been used to measure changes in how well the LV works after revascularization. In patients with stable CAD and intact LVEF, TLS improvement confirms PCI's beneficial effects on LV function.

Patients with CAD and diabetes had worse Tei index, LVEF, and GLS values before PCI than people with CAD only. This was true even though SYNTAX and EXTENT scores that didn't show any statistically significant changes in the amount of atherosclerosis in the coronary arteries. This conclusion is consistent with the hypothesis that diabetics have worse systolic and diastolic LV function, stiffer myocardium, higher resting myocyte tension, and an accumulation of advanced glycated end products in their hearts [21].

In study in our hands, we found that there was statistically significant increase in GLS before and after intervention among non-MACE patients by -13.91% and significant increase in GLS among MACE patients by 1.29%. Also, we found that pre- and delta-GLS were significant predictor for MACE with P value 0.014% and 0.019% respectively in univariate regression and significant predictor for MACE with P values 0.018 and 0.015 in multivariate regression respectively among non-diabetic patients.

The pre- and delta-GLS also were significant predictor for MACE with P values 0.014 and 0.019 respectively in univariate regression and significant predictor for MACE with P values 0.018 and 0.015, respectively in multivariate regression among diabetic patients.

The term "MACE" is typically used to refer to a clinical outcome composed of death, myocardial infarction, repeat intervention due to restenosis, and stent thrombosis. In a research by **Aniyathodiyil *et al.*** [22] involving 50 patients, 29 individuals experienced a total of 51 incidents during the course of a 6-month follow-up period. He discovered that the GLS, MPI, WMS, WMSI, and E/e' values among the total of 29 patients with events were compared to those patients without having any of the listed events at the time of discharge. When compared to the no event group, the values of GLS were lower in the event group. The GLS values between the two groups did not differ appreciably. Studies have indicated that, like an EF value of 40%, MPI value of > 0.47, and WMSI of 1.4, a GLS value of less than 12% at discharge was linked to a greater event rate following MI. A GLS value of <12% was able to identify incidents in 58% of the 29 individuals who had them. Similar to this, patients having events could be identified in 72% of cases where the MPI value was > 0.47. Only 20% of the patients may, however, have events detected by an LVEF value of less than 40%. **Cong *et al.*** [23] found that Patients in the MACE group had lower GLS compared with the non-MACE group (8.7 ± 2.0 vs. 14.0 ± 2.7, P <0.01). Furthermore, GLS was quantitatively altered in patients with three vascular diseases (−15% ± 2.3%) significantly more than in cases with one vessel disease (−17.3% ± 3.7%) or two vessel diseases (−16.6% ± 2.8%), according to **Caspar *et al.*** [24]; however, none of these differences were statistically significant.

Compared to non-diabetic patients, those with DM had CAD that is more advanced and are more likely to produce subpar clinical results. In a recent observational study on PCI in diabetics, **Safley *et al.*** [25] found that these patients did not appear to benefit from successful PCI in terms of survival as much as those without diabetes.

Also, significant increase in GLS pre- and post-intervention among non-MACE diabetic patients by -14.2% with Mean: -13.24 (SD 3.13) and -15.12 (SD 0.89), respectively and P-value 0.002 and there was no

significant change in GLS among MACE in diabetic patients. We illustrated that there was no significant difference between non-MACE and MACE diabetic patients and non-MACE and MACE non-diabetic patients as regards clinical data as BMI, laboratory readings as HB, TLC, PLT, RBS, CKMB, troponin, creatinine and total cholesterol. **Aniyathodiyil *et al.* [22]** discovered that there were no discernible differences between non-MACE and MACE diabetic patients and non-MACE and MACE non-diabetic patients as regard clinical data (P >0.05).

In this study we demonstrated that there was statistically significant difference in Univariate linear regression as regard Post-LVidS. **Nabati *et al.* [26]** found that the LVIDs in the diabetic group decreased by 10% (P=0.002) between baseline and one month after PCI, with baseline values of 316.3 and 285.8 respectively. While, in the non-diabetic group, the LVIDs at baseline and one month after PCI were 284.1 and 30.96.6 respectively, and an 8% increase in LVIDs occurred. **Chowdhury *et al.* [27]** also discovered that LVIDs had higher internal dimensions of the left ventricle during systole in the patients who develop events than those in the non-MACE (P <0.001).

In the current trial, people with and without diabetes showed an improvement in LV function after PCI. Nevertheless, following PCI, individuals with CAD and diabetes exhibited lower LVEF and GLS values than patients with CAD but no DM. This may imply that PCI increased LV function uniformly in all research participants and that differences were caused by lower baseline LV function in DM patients.

CONCLUSION

Speckle tracking echocardiography was a viable, non-invasive, and promising modality to assess subclinical left ventricular systolic dysfunction and predict MACE. Following PCI, diastolic dysfunction and all diastolic filling pattern parameters significantly improved in patients with ischemic heart disease who had impaired systolic function.

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