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Original Article

Alterations in echocardiographic left ventricular function after percutaneous coronary stenting in diabetic patients with isolated severe proximal left anterior descending artery stenosis

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ABSTRACT

Background: There are conflicting theories regarding the use of percutaneous coronary intervention (PCI) of isolated severe proximal left anterior descending (LAD) artery stenosis in place of left internal mammary artery grafting in diabetic patients. The aim of this study was to investigate the effect of PCI on left ventricular function and determine difference between diabetics and non-diabetics.

Methods: A prospective study was conducted on 50 patients with isolated severe proximal LAD stenosis: 23 diabetic and 27 non-diabetic patients. Successful PCI with everolimus-eluting stents was performed for all of the patients. These patients underwent transthoracic echocardiography within 24 h before and 1 month after PCI, and alterations in the left ventricular parameters were compared between the two groups.

Results: There was a significant 12% increment in the mitral annular peak systolic velocity (s') ($p = 0.02$), 21% decrement in the trans mitral early filling deceleration time (DT) ($p < 0.001$), 10% decrement in the systolic left ventricular internal dimension (LVIDs) ($p = 0.002$), significant increment in the left ventricular ejection fraction (LVEF) ($p = 0.004$), and significant decrement in the left atrial diameter ($p = 0.006$) in the diabetic patients after performing PCI. Conversely, the non-diabetic patients showed a statistically significant 14% increase in the DT, 6.3% decrease in the s' velocity, 8% increase in the LVIDs, significant increment in the left atrial diameter and no change in LVEF after PCI.

Conclusion: Our study demonstrated that everolimus-eluting stents favorably improved the markers of left ventricular systolic and diastolic function in diabetic patients with isolated severe proximal LAD stenosis compared with those of non-diabetic patients with the same condition.

1. Introduction

In total, 15–20% of patients who undergo coronary revascularization are diabetic.[1] The long-term results of percutaneous coronary intervention (PCI) and coronary artery bypass graft are less desirable in diabetic patients. This outcome is most likely due to a faster progression of atherosclerosis and a higher rate of restenosis. While the use of stents has improved the short- and long-term outcomes of PCI in diabetic patients, the consequences of PCI are still less favorable in diabetics compared with non-diabetic patients. New angioplasty techniques, such as drug-eluting stents, most likely have a critical role in improving the results of PCI in diabetic patients. Thus, there are indications for angioplasty in diabetics to be extended even more in the near future.[2] Echo Doppler studies have reported that patients with asymptomatic left ventricular (LV) diastolic dysfunction have a higher incidence of all-cause mortality. Mild diastolic dysfunction and moderate to severe dysfunction were associated with 8.3-fold and 10.2-fold increased risks of mortality, respectively. The overall mortalities of symptomatic patients with diastolic or systolic heart failure are very similar.[3] Trans mitral Doppler flow velocities have previously been used for evaluating the variations in LV diastolic function after PCI.[4]

The left atrial (LA) diameter, early filling deceleration time (DT), E/e' ratio and peak atrial reversal flow velocity of the pulmonary

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veins are relatively independent of preload and are a more reliable index of LV relaxation. Additionally, the LV ejection fraction (LVEF), regional LV wall motion and peak systolic velocity (s') of the mitral annulus have been used as an index of the global LV systolic function. Few studies have compared the echocardiographic systolic and diastolic parameters between diabetics and non-diabetics after PCI on left anterior descending (LAD) artery. These studies showed that PCI on LAD induced more increase in LVEF compared with angioplasty on other vessels. Furthermore, existence of DM did not have negative effect in LVEF improvement after angioplasty on LAD. However, other systolic and diastolic variables were not separately compared between diabetic and non-diabetic patients.[5] Improvements in systolic and diastolic LV function are associated with better outcome and functional capacity.

Therefore, our study was designed to determine the changes in LV systolic and diastolic function after successful PCI and drug-eluting stenting of isolated stenosis of the proximal LAD artery in diabetic patients compared with non-diabetic patients.

2. Methods

2.1. Patients

From 2012 to 2013, a prospective clinical study was conducted in the Fatemeh Zahra teaching hospital. In total, 23 consecutive elective diabetic patients were considered for enrollment who presented with typical chest pain and documented myocardial ischemia with a considerable amount of anterior or anteroseptal ischemia on single-photon emission computerized tomography and who underwent successful angioplasty after displaying isolated severe proximal LAD coronary artery stenosis (>75% luminal diameter) in a recent angiography. Additionally, 27 non-diabetic patients were assigned to the control group. Then, alterations in the echocardiographic variables after the procedure were compared between the two groups.

The study was performed according to the guidelines of the Helsinki Declaration and was approved by the ethics committee of the hospital. Written informed consent was obtained from all of the patients. Patients with lesions in the first diagonal branch, total occlusion of the LAD or multivessel coronary artery disease were excluded from our study. None of the study patients had congenital, significant valvular heart disease (equal or more than moderate severity) or cardiomyopathy and atrial fibrillation. Additionally, patients with systemic diseases, such as cancer, collagen vascular diseases or amyloidosis, were excluded.

2.2. Echocardiography

Transthoracic echocardiography was performed at baseline within 24 h before PCI and was repeated 1 month after PCI for all patients by a Vivid S5 (GE Healthcare, Wauwatosa, WI, USA), 1–3 MHz transducer. All of the measurements represent the average of three consecutive beats between normal heart rate ranges, 60–100 beat per minute. The images were stored on a hard disk for better offline measurements, and the results were confirmed by an echocardiographer who was blind to the patient's information. Patients with a poor echo window were excluded from the study.

Estimates of the LV systolic and diastolic dimensions were derived from the LV minor-axis dimensions with the transducer in the parasternal position so that the cursor was perpendicular to the interventricular septum and posterior wall at the mid-papillary muscle level. The EF and wall motion abnormalities (WMA) were determined. The EF was defined as the end diastolic volume minus the end systolic volume divided by the end diastolic volume from biplane apical two and four chamber views using a modified Simpson's technique. To assess the reproducibility of determination of EF by a modified Simpson's technique, this index was measured in 10 randomly selected patients and was repeated 1 day later to calculate the intra-observer correlation coefficients, which was found to be 0.94.

Pulse Doppler recordings of the diastolic trans mitral flow velocity were obtained with the sample volume located at the tips of the mitral leaflets from the apical four chamber view. The peak early diastolic velocity (E wave), peak late diastolic velocity (A wave), early filling DT and E/A velocity were measured. Tissue Doppler imaging of the mitral annulus was obtained from the apical four chamber view. A 5 mm sample volume was placed at the septal and lateral mitral annuli. The following measurements were determined: the peak systolic velocity (s') and early diastolic velocity (e'). An analysis was performed for the average of each velocity at the two annular sites. Then, the E/e' ratio was calculated. The pulmonary venous flow velocity profile was obtained from an apical four chamber view. The pulse Doppler sample volume was placed 1 cm into the pulmonary vein, and the peak systolic flow velocity (S wave), peak diastolic velocity (D wave) and peak atrial reversal flow velocity (AR) were determined. The LA diameter was measured in the parasternal long-axis view from a 2D image at end systole.

Blood samples were obtained during fasting, and the levels of plasma glucose, total cholesterol (T-chol), high density lipoprotein (HDL)-chol, low density lipoprotein (LDL)-chol, and triglycerides (TG) were measured. The systolic and diastolic blood pressures were measured after 5 min of rest. The height and weight were measured, and the body mass index (BMI) was calculated as the body weight divided by the height squared. Hypertension was defined as a systolic blood pressure $\geq$140 mmHg, a diastolic blood pressure $\geq$90 mmHg[6] or the requirement for antihypertensive medication. The diabetes mellitus (DM) was defined according to the criteria of the American Diabetes Association[7] or the requirement for insulin or oral hypoglycemic drugs. A family history of coronary artery disease (CAD) was defined as having a first-degree relative (a male <55 years or female <65 years) with a history of myocardial infarction, coronary revascularization, or sudden death.[8] The history of smoking was determined by a face-to-face questionnaire.

Coronary angiography was performed for all of the patients using a cardiac angiography system (Siemens AG, Medical Solutions, Erlangen, Germany), and they all underwent PCI. PCI was performed by standard techniques, and XIENCE stents (Everolimus-Eluting Coronary Stent System, Santa Clara, CA, USA) were used.

Procedural success was defined as the successful deployment of the stent and residual stenosis of less than 30%.[9] Procedural anticoagulation was achieved with unfractionated heparin; glycoprotein IIb/IIIa inhibitors were used whenever needed. Patients received 300 mg of aspirin before the intervention. A 300 mg oral dose of clopidogrel was recommended before the procedure. Thereafter, 80 mg of aspirin and 75 mg of clopidogrel were prescribed daily. Other standard drugs (angiotensin converting enzyme inhibitors, beta blockers, statins and oral hypoglycemic agents) remained unchanged during the study in order to minimize the effects of alterations on the echocardiographic variables.

2.3. Statistical analysis

Continuous variables are expressed as the mean ± SD. Percentage changes in all of echocardiographic variables following PCI were determined. These variables were considered as new variables, separately. Then, two groups were compared for these new variables by an independent t-test. Paired-samples t were used for comparing

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Table 1
Demographics and measurements of common cardiovascular risk factors of subjects categorized as having or not having diabetes mellitus.

	Non-diabetics	Diabetics	*p* value
Gender			
Male	19 (70.4%)	7 (30.4%)	0.01
Female	8 (29.6%)	16 (69.6%)	
Hypertension	16 (59.3%)	18 (78.3%)	0.225
Cigarette smoking	6 (22.2%)	2 (8.7%)	0.261
Family history	5 (18.5%)	10 (43.5%)	0.07

the means of variables before and after PCI for each group, separately. For determining normality, we evaluated each variable individually in both groups, separately by Shapiro–Wilk test. For variables that were non-normally distributed, the Mann–Whitney *U* test was used. Categorical variables were compared with a Chi-squared test. A $p < 0.05$ was considered to be statistically significant. Well known CAD risk factors were included in a logistic regression model of CAD (for those who demonstrated statistically significant difference between two groups).

All of the statistical calculations were performed using SPSS/PASW (Predictive Analytics SoftWare) Statistics 18 (SPSS Inc., Chicago, IL, USA). The sample numbers were determined by previous studies with the following statistical formula (according to standard deviation of time variable in Table 2 of Ref. 4):

$$n_1 = n_2 = \frac{2\times(Z_{1-(\alpha/2)} + Z_{1-\beta})^2\sigma^2}{\epsilon^2} = 21$$
$$\alpha = 0.05$$
$$\beta = 0.2$$
$$\sigma^2 = 49$$
$$\epsilon = 6$$

3. Results

A total of 24 women and 26 men were enrolled in the study. The mean age was 57.68 ± 8.8 years. Within the study population, 23 patients (46%) were diabetic; 8 patients (16%) were smokers; 15 patients (30%) had a positive family history of CAD; and 34 patients (68%) were hypertensive. The demographics and measures of common cardiovascular risk factors of the study population and the study group categorized by having or not having DM are presented in Tables 1 and 2. An independent *t*-test did not show any statistically significant difference for age between two groups. Therefore, we used this test for determining differences between two groups.

We assessed the association between the DM and the variables related to cardiovascular risk factors by a Chi-squared test. No significant correlation was found for smoking ($p = 0.261$), hypertension ($p = 0.225$), or a family history of CAD ($p = 0.07$). The correlations between the DM and fasting blood sugar (FBS), TG, T-chol, HDL-chol, LDL-chol and BMI levels (evaluated by an independent *t*-test, Levene's test for equality of variances) were analyzed. There was significant correlation between the DM and FBS level ($p < 0.001$). However, there was not a significant correlation between the DM and TG levels ($p = 0.754$), T-chol levels ($p = 0.95$), HDL-chol levels ($p = 0.43$), LDL-chol levels ($p = 0.43$), or BMI levels ($p = 0.22$). In diabetics, glycated hemoglobin level (HbA1c) was $7.83 \pm 2.61\%$ and $7.37 \pm 2.87\%$ before and after PCI, respectively. This was not statistically significant ($p = 0.5$). There were statistically insignificant differences ($p = 0.36$) in the percentage diameter stenosis of LAD before the PCI between the non-diabetics ($91 \pm 9.91\%$) and diabetics ($87.5 \pm 12.3\%$).

Again, after performing PCI, these differences were not significant (residual stenosis of $6 \pm 6.4\%$ in non-diabetics and $4.1 \pm 5.9\%$ in diabetics; $p = 0.37$). Additionally, the stent diameter was 2.8 ± 0.26 mm in non-diabetics and 2.7 ± 0.29 mm in diabetics ($p = 0.33$); the stent length was 23.9 ± 6.8 mm in non-diabetics and 25 ± 9.25 mm in diabetics ($p = 0.72$), and the stent number was 1.05 ± 0.22 in non-diabetics and 1.06 ± 0.25 in diabetics ($p = 0.9$). For determining normality, we evaluated each variable individually in both groups, separately by Shapiro–Wilk test. LVEF, LA diameter and *E*/*A* ratio were not normally distributed. For these variables that were non-normally distributed, the Mann–Whitney *U* test was used.

When comparing differences between the two groups after the procedure by an independent samples *t*-test (Table 3), diabetic patients showed that the systolic left ventricular internal dimension (LVIDs) decreased by 10% and *s*′ velocity increased by 12%. Additionally, there was a 21% decrement in the DT in these patients. All of these data were statistically significant. Furthermore, they showed statistically significant increases in the LVEF (median 5%, 95% confidence interval [2.5–7.5%], $p = 0.004$) and significant decreases in LA diameter (median −0.1 cm, 95% confidence interval [−0.25 to 0.1 cm], $p = 0.006$). Conversely, the non-diabetic patients showed a statistically significant 8% increase in the LVIDs, a 14% increase in the DT, and a 6.3% decrease in the *s*′ velocity. Also, there was significant increase in LA diameter (median 0.11 cm, 95% confidence interval [0.085–0.3 cm], $p = 0.006$). LVEF did not change in non-diabetics (median 0%, 95% confidence interval [−2.5 to 2.5%], $p = 0.004$). More females (69.6%) were included in the diabetic group. Conversely, more men (70.4%) were present in the non-diabetic group.

We evaluated the potential confounding effect of gender by multiple regression analysis, separately. It showed that confounding effect of gender was not statistically significant (EF: $p = 0.59$ for sex, $p = 0.012$, $B = 6.76$, 95% confidence interval [1.55–11.96] for DM, DT: $p = 0.35$ for sex, $p = 0.001$, $B = -89.24$, 95% confidence interval [−139.18 to −39.30] for DM, LA diameter: $p = 0.54$ for sex, $p = 0.014$, $B = -0.275$, 95% confidence interval [−0.49 to −0.059] for DM, LVIDs: $p = 0.74$ for sex, $p = 0.007$, $B = -0.53$, 95% confidence interval [−0.92 to −0.15] for DM). Regarding the *e*′ velocity alteration, the paired samples *t*-test showed a significant increase in the *e*′ velocity after PCI in

Table 2
Demographics and measurements of common cardiovascular risk factors of study population and subjects by study group.

	Whole study population		Non-diabetics ($n = 27$)		Diabetics ($n = 23$)		*p* value
	Mean	SD	Mean	SD	Mean	SD	
Age (y)	57.68	8.82	59.6296	9.38963	55.3913	7.69734	0.091
FBS (mg/dl)	144.22	79.25	100.7692	11.94088	195.5909	93.73248	<0.001
TG (mg/dl)	212.69	128.79	207.1923	148.63351	218.9130	104.93674	0.754
CHOL (mg/dl)	184.83	50.21	184.4231	49.92248	185.3043	51.65835	0.95
HDL (mg/dl)	43.89	18.05	41.9615	9.20209	46.0870	24.60767	0.43
LDL (mg/dl)	102	21.05	104.2308	20.36724	99.3636	22.00767	0.43
BMI (kg/m2)	26.76	3.93	26.1259	4.09320	27.5253	3.69471	0.22

SD: standard deviation, FBS: fasting blood sugar, TG: triglycerides, CHOL: cholesterol, HDL: high density lipoprotein, LDL: low density lipoprotein, BMI: body mass index.

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Table 3
Echocardiographic indices of left ventricular systolic and diastolic function before and after PCI categorized as having or not having DM.

	LVIDd (cm)	LVIDs (cm)	DT (ms)	*S*/*D*	AR (m/s)	*e*′ average (cm/s)	*E*/*e*′	*s*′ average (cm/s)
No DM								
Pre-PCI	4.69 ± 0.36	2.8 ± 0.41	280 ± 70	1.5 ± 0.3	0.347 ± 0.09	8 ± 2.2	7.76 ± 2.44	7.2 ± 1.34
Post-PCI	4.76 ± 0.55	3.09 ± 0.66	321 ± 78	1.47 ± 0.36	0.36 ± 0.07	8.6 ± 2.1	7.78 ± 2	6.7 ± 1.34
DM								
Pre-PCI	4.77 ± 0.45	3.1 ± 0.63	266 ± 59	1.47 ± 0.37	0.36 ± 0.068	7.98 ± 2.3	8.49 ± 2.98	6.56 ± 1.3
Post-PCI	4.6 ± 0.47	2.8 ± 0.58	210 ± 63	1.5 ± 0.44	0.33 ± 0.053	9.16 ± 2.66	7.98 ± 2.6	7.28 ± 1.29
Difference								
No DM	0.075 ± 0.53 (1.6%)	0.22 ± 0.63 (8%)	41 ± 83 (14%)	−0.03 ± 0.35 (−2%)	0.007 ± 0.095 (2%)	0.57 ± 1.68 (7%)	0.0168 ± 2.2 (0.2%)	−0.46 ± 1.66 (−6.3%)
DM	−0.15 ± 0.48 (−3%)	−0.33 ± 0.56 (−10%)	−57 ± 74 (−21%)	0.03 ± 0.41 (2%)	−0.03 ± 0.091 (−0.8%)	1.18 ± 2.4 (14%)	−0.5 ± 2.41 (−5%)	0.8 ± 1.76 (12%)
p value	0.135	0.002	<0.001	0.56	0.183	0.3	0.42	0.02

PCI: percutaneous coronary intervention, DM: diabetes mellitus, LVIDd: diastolic left ventricular internal dimension, LVIDs: systolic left ventricular internal dimension, DT: deceleration time, AR: atrial reversal.

diabetics ($p = 0.03$) and a trend toward significance in non-diabetics ($p = 0.09$) separately. However, when the two groups were compared by an independent samples *t*-test, there was no significant difference in the e' velocity improvement between diabetics and non-diabetics ($p = 0.3$). Furthermore, the diastolic left ventricular internal dimension (LVIDd), AR, E/i (diabetics: median 0.068, 95% confidence interval [−0.1 to 0.2], non-diabetics: median 0.116, 95% confidence interval [0.07–0.15], $p = 0.4$), S/D and E/e' did not change significantly after performing PCI between the diabetic and non-diabetic groups.

Before PCI, the WMA was observed in 2.2 ± 2.91 segments that significantly decreased to 1.5 ± 2.58 segments ($p = 0.04$) after the procedure. However, there was no significant difference in WMA improvement between the diabetics and non-diabetics, (before PCI: 2.69 ± 2.96 and 1.77 ± 2.86 and after PCI: 1.69 ± 2.9 and 1.33 ± 2.24 segments, respectively, $p = 0.4$).

4. Discussion

Stents control two of the three mechanisms of restenosis: the initial elastic recoil and late remodeling. However, they cannot reduce intimal hyperplasia; indeed, stents may increase it.[2] The introduction of stents that release drugs with inhibition effects on intimal hyperplasia may reduce or eliminate the main limitation of angioplasty.[10] Polymer-based paclitaxel-eluting stents and sirolimus-eluting stents have been shown to significantly reduce angiographic restenosis in comparison with bare metal stents. However, the rates of primary stent thrombosis are increased. With the goal of further augmenting the security and effect of DES, an everolimus-eluting stent (EES) has been designed.[11] Diabetic patients with CAD experience worse outcomes compared with those without diabetes mellitus. In the absence of multivessel or left main CAD, PCI is a simple and efficient technique for revascularizing involved coronary arteries in these patients. Lower probability of repeat revascularization and stent thrombosis with DES have influenced selected options of revascularization for diabetic patients. This is particularly useful with the second generation of DES using everolimus.[12]

In a 14-day rabbit iliac model, the endothelialization over the stent struts was more rapid with the EES than with the stents eluting sirolimus, zotarolimus, or paclitaxel.[13] In 2000, Bayata et al.[4] evaluated the early effect of PCI on LV diastolic dysfunction in 30 patients with isolated severe LAD stenosis. All of the measurements were performed within 4 h before PCI and repeated within 24 h after angioplasty. After angioplasty, none of the parameters (DT, isovolumic relaxation time, E/A and E wave transit time), except for the A wave transit time, were changed significantly. However, the tissue Doppler study and the calculation of the EF, LV and LA diameter were not included in this study. Additionally, the effects of common cardiovascular risk factors, such as DM, were not determined. Furthermore, stenting was not performed for all of the patients.

In 2013, Mehrpooya et al.[5] assessed the effects of PCI on LVEF and WMA in 40 patients who presented with ischemic cardiac chest pain, an ejection fraction less than 40%, and significant coronary occlusion (70%). Echocardiography was performed at baseline and one month after the procedure. The mean EF increased significantly after angioplasty ($p < 0.000$). All of the patients (100%) had wall motion abnormality at baseline, but the frequency decreased to 65% of them after the procedure. The improvement of EF in patients with significant stenosis of the LAD was higher than in patients without LAD lesions ($p = 0.008$).

In 2005, Agirbasli et al.[14] evaluated LV contractility and myocardial perfusion after PCI in 60 patients who underwent successful LAD stenting. Myocardial perfusion and LVEF improved at 6 ± 3 months after the procedure ($p = 0.05$). Patient-related factors,

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such as DM, presentation with acute coronary syndrome and age, did not affect the LVEF change after the procedure.

Impaired LV relaxation is the earliest manifestation of myocardial ischemia. The primary abnormality of myocardial relaxation is characterized by a decrease in the *E* wave velocity, an increase in the *A* wave velocity and *E*/*A* ratio, and an increase in the DT and AR velocity. Later, it is followed by an increase in the LA diameter. The LA acts as a conduit between the pulmonary vascular bed and the LV. In addition, the atrium acts as an efficient volume sensor, releasing natriuretic peptides and other neurohormones into the circulation as a consequence of increased atrial wall stress. Thus, when the LA empties against a noncompliant LV and/or there is an increase in the LV end-diastolic pressure, the LA pressure will rise. This pressure is poorly tolerated by the thin wall of the LA, and subsequent dilation will occur. Chronic LA pressure overload will eventually cause LA wall fibrosis. Indeed, LA size can be considered a biomarker of sustained elevations in LV filling pressures.[15] Additionally, a decrease in the early diastolic mitral annular motion (e') is related to a decreased LV elastic recoil, which is noted as an early sign of ischemia and impaired relaxation. In contrast, LV systolic function is an important predictor of prognosis, and parameters that represent systolic function, such as the LVEF, WMAs and LVIDs and LVIDd, can be used for predicting outcomes.[16] Recently, mitral annular systolic motion (s') was introduced as a method for measuring LV systolic function. Cardiac disorders can disturb both longitudinal and radial contractile function. However, longitudinal function is impaired initially. Therefore, the longitudinal contractile function of the annulus (s') can be useful in earlier diagnosis of ischemia.[17]

In our study, diabetics showed significant improvement in the systolic LVID, LA diameter, DT, EF and s' velocity compared with non-diabetics. At baseline, most of these parameters were worse in diabetics compared with non-diabetics. This outcome may be due to a worse effect of ischemia in diabetics. However, our study also showed excellent reversibility of these adverse effects after PCI in this subgroup. In non-diabetics, there was not any change in LVEF after PCI. WMA improvement was observed in both diabetics and non-diabetics. Additionally, in spite of increased DT after performing PCI in non-diabetics, it remained in a category of impaired relaxation (Grade I of diastolic dysfunction) with normal filling pressure. The decreased s' velocity in non-diabetics may be due to hibernated myocardium, micro embolization or microvascular dysfunction. Regarding the explanation for such a possible difference in the LV function improvement with PCI in LAD between diabetic and non-diabetic patients, baseline echocardiographic variables were significantly worse in diabetic patients. Another possible explanation may be "chance" in view of the small sample data.

A limitation of our study was the small sample size. Additionally, there were more females in the diabetic group compared with the non-diabetic group. However, we tried to modulate the confounding effect of gender with statistical analyses. Another limitation was the use of the LV and LA diameters instead of the volume. Our follow-up period was one month after PCI.

It was in concordance with previous similar studies such as Bayata and Mehrpoya that their follow up periods were 24 h and one month after angioplasty, respectively. It seems that shorter follow up period may minimize confounding effect of time on echocardiographic variables. In conclusion, our study showed that EES favorably improved markers of LV systolic function, such as the LVIDs, s' velocity and EF, in diabetics compared with non-diabetics. Additionally, there were significant improvements in the LA diameter and DT, which are markers for LV diastolic function.

Conflicts of interest

The authors have none to declare.

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