

Assessment of Right and Left Ventricular Functions in Patient with Type I Diabetes Mellitus: Strain Imaging Study

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ABSTRACT

Background: Diabetes mellitus type 1 (DM1) is frequently accompanied by increasing risks of cardiovascular diseases. The prevention of cardiac complications depends on the early diagnosis of myocardial dysfunction.

Objective: Our objective was to assess both right and left ventricular (LV) functioning in DM1 patients depending on strain imaging by 2D-speckle tracking echocardiography (2D-STE).

Patients and methods: The present prospective, case-controlled study was conducted in the Cardiology Department, Menoufia Faculty of Medicine, from May 2021 to December 2022. 90 subjects were selected, divided into two groups. 60 patients with type I diabetes mellitus served as cases and 30 healthy subjects age matched served as control.

Results: E wave, E/A value, GLS endo, GLS myo and average global longitudinal strain were lower in patients than controls (70.43 ± 13.67 cm/s vs. 84.67 ± 11.97 cm/s, 1.05 ± 0.25 vs 1.4 ± 0.18 , $-20.48 \pm 2.81\%$ vs. $-24.08 \pm 1.11\%$, $-18.71 \pm 2.09\%$ vs. $-21.76 \pm 1.01\%$, and $-18.82 \pm 1.92\%$ vs $21.1 \pm .62\%$ respectively, $p < 0.001$), meanwhile there was no difference between both groups regarding ejection fraction and RV strain measurements ($68.48 \pm 5.96\%$ vs. $67.39 \pm 3.55\%$ and $-21.13 \pm 1.29\%$ vs. $-21.06 \pm 1.24\%$ respectively, $p = 0.357$ and $p = 0.807$).

Conclusion: Patients with DM1 and without known heart diseases have diastolic and subclinical systolic dysfunction with lower LV endocardial and myocardial longitudinal strain demonstrated by multi-layered STE.

Keywords: Diabetes mellitus, Longitudinal strain, Speckle tracking.

INTRODUCTION

The International Diabetes Federation anticipated that by 2040, there would be 642 million cases of DM, this now has an impact on 8.8% of the world's population. Effective hyperglycemia management has been shown to drastically lower cardiovascular events in diabetic patients, which are the main cause of mortality and morbidity in people with diabetes ^(1,2). Studies revealed that when compared to healthy persons, DM1 is linked to a 10-fold increased risk for cardiovascular disease ⁽³⁾.

The term "diabetic cardiomyopathy" (DCM) was originally used in 1972 by Rubler *et al.* to characterize a non-ischemic form of cardiac dysfunction that results in heart failure. The diagnostic standards for DCM are not yet defined. In the absence of CAD, arterial hypertension, valvular heart disease, or other myocardial dysfunction-causing conditions, it is characterised by the existence of diastolic or systolic malfunction of the heart ⁽⁴⁾.

To evaluate the structure and function of the heart, conventional echocardiographic measures are frequently applied. However, in some circumstances, routine assessments may be less sensitive for the diagnosis of moderate and subclinical heart dysfunction. The restriction of conventional echocardiogram appears to be solved by 2D-STE, which has proven to be an accurate and reliable method for the detection of mild myocardial dysfunction before changes in traditional function measures ⁽⁵⁾.

The currently established approach for assessing myocardial function is 2D-STE, which has shown to offer insightful knowledge about the workings of the heart. With STE, it is possible to examine myocardial longitudinal, circumferential, and radial deformation,

characteristics that have been demonstrated to be more effective than traditional echocardiography at detecting mild cardiac dysfunction. Furthermore, current software methods enable the measurement of endocardial, myocardial, and epicardial layers strain individually, offering an even more thorough assessment of the heart function ⁽⁶⁾.

This study aimed to assess the right and LV functions in patients with DM1 using speckle tracking imaging study.

PATIENTS AND METHODS

The present prospective, case-controlled study was conducted in the Cardiology Department, Menoufia Faculty of Medicine, from May 2021 to December 2022. 90 subjects were selected, divided into two groups. 60 patients with type I diabetes mellitus served as cases and 30 healthy subjects age matched served as control.

The included patients' ECGs and echocardiograms were both normal, and the recording of routine echocardiographic and speckle tracking measures was possible due to the satisfactory picture quality of the echocardiograms. But patients who had LV ejection fraction $< 55\%$ or any regional wall motion abnormality, ischemic heart disease, arrhythmia, valvular, pericardial, congenital heart diseases, endocrinal or other system diseases were excluded from the study.

Both groups underwent:

A complete history was obtained from the patient, with specific focus placed on a history of diabetes mellitus duration, medication, hypertension, arrhythmia, heart failure, valvular heart diseases,

ischemic heart disease and endocrine or other system disease.

Complete clinical examination was done including weight and height and calculating body mass index (BMI) according to the formula ($BMI = \text{weight (kg)} / \text{height (m}^2\text{)}$). Glycated hemoglobin (HBA1C) and fasting blood sugar (FBS) were done as a laboratory workup. Also resting ECG was done.

Conventional Echocardiography:

Image acquisition with transthoracic echocardiography was done using Vivid 9 machine (General Electric Healthcare, equipped with harmonic M5S variable frequency (1.7–4 MHz) phased-array transducer, Norway), by parasternal, apical (two, three and four) and subcostal views through 2D, M mode, pulsed wave Doppler and continuous wave Doppler for all included patients those were positioned in there left lateral decubitus position. The following parameters were measured in accordance with the norms and guidelines of the European Association of Cardiovascular Imaging and the American Society of Echocardiography. Left atrium diameter (LAD (mm)), aortic diameter (mm), interventricular septum in diastole(mm), left ventricular end diastolic dimension (LVEDD (mm)), left ventricular end systolic dimension (LVESD (mm)), left ventricular posterior wall diameter in diastole (mm), end diastolic volume(ml), end systolic volume (ml), stroke volume(ml), fractional shortening (%), E-velocity, left ventricular ejection fraction (LV EF %), mitral annular plane systolic excursion (MAPSE), tricuspid annular plane systolic excursion (TAPSE), E wave, E/A ratio. Using tissue Doppler imaging, early (E') and late (A') diastolic velocities were calculated in septal and lateral LV wall and right ventricular free wall and E/ E' ratio in septal and lateral LV wall knowing that E/ E' ratio normal value is less than 8⁽⁷⁾.

Two-dimensional grey scale dedicated software for Echo Pac version BT13 was used to analyze speckle tracking pictures offline while employing an 18 segment LV model. End diastole was determined by the peak of the R wave on the QRS echocardiogram trace, whereas end systole was established by the aortic valve closing on the pulsed wave Doppler recordings acquired by sampling the LV outflow tract. Through manual

delineation of the endocardium contour, the area of interest was identified. After specifying the range of interest, the values of the global longitudinal deformation (GLS) for the endocardium (GLS endo), myocardium (GLS myo), and epicardium (GLS epi) were automatically calculated. Based on the index echocardiogram, normal LV GLS absolute value is < -18%.

Using a modified 4-chamber image and a 6-segment model, RV GLS was calculated by tracing the RV free wall and the endocardium of the interventricular septum. The longitudinal strain of the RV free wall could only be measured by tracing the endocardium border of the RV free wall (3 segments only). According to the index echocardiography, the typical RV GLS absolute value is < -20%.

Ethical approval:

Menoufia Medical Ethics Committee of the Menoufia Faculty of Medicine gave its approval to this study (IRB No.: 4/2020CARD10). All participants gave written consent after receiving all information. The Helsinki Declaration was followed throughout the study's conduct.

Statistical analysis

The data were coded and entered using SPSS version 28. Quantitative variables were given as mean and standard deviation to summarise the data, while categorical variables were given as frequencies (number of occurrences) and relative frequencies (percentages) to summarise the data. When performing comparisons, unpaired t tests were employed to compare quantitative data. To compare categorical data, the chi square (X^2) test was utilised. A P-value of less than 0.05 was used to indicate statistical significance.

RESULTS

Our study detected that diabetic patients were significantly than control group while gender, height, weight and BMI differences between the two groups were statistically insignificant. HBA1C and FBS were significantly higher in patients than control group (**Table 1**).

Table (1): Demographic data and Laboratory distribution of the studied groups

		Patients (n=60)		Controls (n=30)		P value
		Count	%	Count	%	
Gender	Male	31	51.7%	21	70.0%	0.097
	female	29	48.3%	9	30.0%	
		Mean	StandardDeviation	Mean	StandardDeviation	P value
Age		25.73	3.37	23.67	2.48	0.004
Height		166.77	6.91	168.27	6.17	0.318
Weight		62.67	8.32	64.40	8.00	0.348
BMI		22.39	2.21	22.52	2.15	0.784
HBA1C		7.32	1.31	4.77	0.21	< 0.001
Fasting blood sugar		112.47	12.46	82.17	7.63	< 0.001

Conventional Echocardiographic Measurements: Patients had substantially lower LVEDD, LVESD, end diastolic volume, end systolic volume, and stroke volume than the control group. On the other hand, patients had considerably larger diastolic LV septal diameter and diastolic LV posterior wall diameter than the control group. Other conventional echocardiographic parameters showed no significant difference between both groups (Table 2).

Table (2): Conventional echocardiographic data among the studied groups

	Patients (A)		Controls (B)		P value
	Mean	Standard Deviation	Mean	Standard Deviation	
AO Diam	28.07	3.91	28.93	3.73	0.317
LA Diam	33.13	4.27	33.23	4.05	0.915
LVSD	9.18	1.47	8.03	0.93	< 0.001
LVEDD	44.37	4.90	48.40	3.33	< 0.001
LVESD	27.43	4.13	30.17	2.04	< 0.001
LVPWD	8.77	1.54	8.14	1.07	0.048
EDV	90.98	24.23	109.96	17.37	< 0.001
ESV	29.43	11.28	35.71	5.88	0.001
SV	61.93	15.39	74.55	13.75	< 0.001
FS	34.90	2.76	33.77	2.13	0.052
EF	68.48	5.96	67.39	3.55	0.357
MAPSE	1.54	0.15	1.57	0.13	0.462

LV echocardiographic parameters relieved that E wave, E/A, E' septal and E' lateral were statistically significantly lower in patients than control group while, E/ E' septal and E/ E' lateral were statistically significantly higher in patients than control group. But RV echocardiographic parameters relieved that TAPSE was statistically not significantly different between both groups. While E' and A' wave velocity of free wall (fw) of RV were significantly lower in patients than control group (Table 3).

Table (3): Tissue Doppler imaging data of LV and RV among the studied groups

	Patients (A)		Controls (B)		P value
	Mean	Standard Deviation	Mean	Standard Deviation	
E WAVE (MV)	70.43	13.67	84.67	11.97	< 0.001
E/A (MV)	1.05	0.25	1.40	0.18	< 0.001
E' septal (LV)	9.81	2.90	13.48	1.76	< 0.001
E' lateral (LV)	11.36	3.75	15.80	2.11	< 0.001
E/ E' septal (LV)	7.41	0.98	6.25	0.83	< 0.001
E/ E' lateral (LV)	6.48	0.99	5.28	0.70	< 0.001
TAPSE (RV)	2.36	0.34	2.32	0.31	0.573
E' fw (RV)	12.48	2.33	14.73	2.07	< 0.001
A' fw (RV)	9.33	1.72	11.30	1.88	< 0.001

Speckle Tracking Measurements in the studied groups: Endocardial, myocardial and average global longitudinal strain of LV in apical four, three, two views data were lower in patients than control group, while pericardial global longitudinal strain was lower only in apical three view and statically insignificantly different in apical four and two views. But global right ventricular strain data of the studied groups showed no significant difference between both groups (Table 4).

Table (4): Endocardial, myocardial and epicardial global longitudinal strain data in apical four, three, two views among the studied groups

	Patients (A)		Controls (B)		P value
	Mean	Standard Deviation	Mean	Standard Deviation	
Apical 4 (LV endocardium)	-21.45	2.24	-24.16	1.31	< 0.001
Apical 2 (LV endocardium)	-20.67	3.36	-24.47	2.40	< 0.001
Apical 3 (LV endocardium)	-19.42	4.13	-24.09	1.88	< 0.001
Average (LV endocardium)	-20.48	2.81	-24.08	1.11	< 0.001
Apical 4 (LV myocardium)	-19.70	1.49	-21.47	1.40	< 0.001
Apical 2 (LV myocardium)	-19.05	2.87	-22.61	2.07	< 0.001
Apical 3 (LV myocardium)	-17.52	3.12	-21.30	1.47	< 0.001
Average (LV myocardium)	-18.71	2.09	-21.76	1.01	< 0.001
Apical 4 (LV epicardium)	-18.22	1.39	-17.73	1.41	0.118
Apical 2 (LV epicardium)	-17.12	1.73	-17.45	1.47	0.374
Apical 3 (LV epicardium)	-15.77	2.33	-17.00	1.20	0.008
Average (LV epicardium)	-17.03	1.20	-17.37	0.86	0.168
GLPS-Average	-18.82	1.92	-21.10	0.62	< 0.001
GS-endo (RV strain)	-22.57	1.43	-22.93	1.30	0.248
GS-myo (RV strain)	-21.08	1.51	-21.39	1.56	0.355
GS-epi (RV strain)	-19.78	1.92	-19.89	1.86	0.440
GS-average (RV strain)	-21.13	1.29	-21.06	1.24	0.807

Correlation between LV strain in separate layers and right ventricular strain parameters with other conventional echocardiographic parameters:

The correlation between LV strains in the separate layers (endo, myo and epicardial layers in different views apical two, three and four) plus right ventricular strain data and (tissue Doppler imaging of echocardiographic data) showed statistically highly significant negative correlation with E/ E' septal wall value of LV and E/ E' lateral wall value of LV, while LV strain showed highly significant positive correlation with E wave of LV, E' lateral wall of L), E' septal wall of LV, E wave of RV fw, GS-epicardium of RV strain and GS-average of RV strain. Negative correlation between both left and right ventricular strain and duration of diabetes mellitus was found (Table 5).

Table (5): Correlation between LV strains in the separate layers in different views in relation to tissue Doppler image echocardiography and rightventricular strain data

Tissue Doppler image data		Apical 4 (LV Endo-cardium)	Apical 2 (LV Endo-cardium)	Apical 3 (LV endo-cardium)	Average (LV endocardium)	Apical 4 (LV Myocardium)	Apical 2 (LV myocardium)	Apical 3 (LV myocardium)	Average (LV Myocardium)	Apical 4 (LV epicardium)	Apical 2 (LV epicardium)	Apical 3 (LV epicardium)	Average (LV epicardium)	GLPS-Average
E wave (MV)	r	0.566	0.431	0.617	0.63	0.467	0.413	0.511	0.559	0.037	0.064	0.559	0.401	0.529
	P value	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	0.777	0.625	<0.001	0.001	<0.001
E/A (MV)	r	0.572	0.534	0.695	0.708	0.496	0.547	0.583	0.657	0.037	0.161	0.591	0.463	0.603
	P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.779	0.219	<0.001	<0.001	<0.001
E' septal (LV)	r	0.577	0.523	0.678	0.699	0.501	0.507	0.584	0.65	0.066	0.238	0.589	0.517	0.608
	P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.614	0.067	<0.001	<0.001	<0.001
E' lateral (LV)	r	0.638	0.522	0.697	0.723	0.557	0.51	0.6	0.672	0.089	0.19	0.608	0.513	0.624
	P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.498	0.147	<0.001	<0.001	<0.001
E/E' septal (LV)	r	-0.428-	-0.496-	-0.563-	-0.593-	-0.382-	-0.480-	-0.526-	-0.580-	-0.046-	-0.313-	-0.460-	-0.464-	-0.537-
	P value	<0.001	<0.001	<0.001	<0.001	0.003	<0.001	<0.001	<0.001	0.725	0.015	<0.001	<0.001	<0.001
E/E' lateral (LV)	r	-0.542-	-0.515-	-0.642-	-0.667-	-0.481-	-0.509-	-0.583-	-0.643-	-0.052-	-0.240-	-0.515-	-0.461-	-0.575-
	P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.694	0.064	<0.001	<0.001	<0.001
Right ventricular strain data														
TAPSE (RV)	r	0.071	0.166	0.061	0.057	0.102	0.127	0.059	0.033	0.045	-0.012-	0.129	0.105	0.027
	P value	0.59	0.204	0.644	0.665	0.439	0.333	0.653	0.8	0.733	0.925	0.327	0.425	0.839
E' fw (RV)	r	0.359	0.314	0.533	0.483	0.359	0.332	0.45	0.464	0.046	0.042	0.439	0.317	0.452
	P value	0.005	0.014	<0.001	<0.001	0.005	0.01	<0.001	<0.001	0.727	0.753	<0.001	0.013	<0.001
A' fw (RV)	r	0.159	0.067	0.268	0.199	0.179	0.118	0.199	0.198	0.125	0.11	0.187	0.113	0.195
	P value	0.224	0.61	0.039	0.127	0.171	0.367	0.128	0.129	0.343	0.402	0.152	0.392	0.136
GS-endo (RV strain)	r	0.093	0.067	0.232	0.064	0.133	0.102	0.185	0.016	0.206	0.089	0.179	0.001	0.005
	P value	0.479	0.61	0.074	0.628	0.313	0.438	0.158	0.901	0.114	0.497	0.172	0.992	0.972
GS-myo (RV strain)	r	0.491	0.262	0.06	0.265	0.402	0.264	0.066	0.25	0.173	0.1	0.123	0.194	0.286
	P value	<0.001	0.043	0.65	0.041	0.001	0.042	0.615	0.055	0.188	0.447	0.351	0.136	0.027
GS-epi (RV strain)	r	0.589	0.5	0.46	0.584	0.488	0.508	0.423	0.559	0.05	0.17	0.275	0.277	0.49
	P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.706	0.195	0.033	0.032	<0.001
GS-Aver (RV strain)	r	0.508	0.359	0.156	0.356	0.452	0.38	0.156	0.359	0.191	0.149	0.111	0.214	0.345
	P value	<0.001	0.005	0.233	0.005	<0.001	0.003	0.233	0.005	0.144	0.257	0.399	0.101	0.007

DISCUSSION

Using the novel echocardiographic method known as 2D-STE, myocardial function may be precisely assessed. The entire evaluation of regional and global function in three dimensions is made possible by this approach, which is accurate, reproducible, and angle independent^(8,9).

The groups selected for our study were comparable because they shared common traits that affect myocardial strain values, such as male and female gender proportion and BMI, even though diabetic patients' ages were statistically significantly older than those of the control group.

In the same line, a study by **Berceanu et al.** revealed that patients in the diabetes group were marginally but considerably older than those in the control group, but no other significant differences in BSA, BMI, weight, or height were observed⁽¹⁰⁾.

According to our conventional echocardiogram results, the mean Echo readings for both groups had normal ranges. However, there were a few minor but statistically significant differences found between diabetics and the healthy control group, including decreased LVEDD, LVESD, end diastolic volume, end systolic volume, and stroke volume in the patient group. As opposed to that, patients had larger LV septal and posterior walls during diastole than did the control group. The fact that E wave, E' septal wall, E' lateral wall, and E' RV fw were statistically lower in patients than in the control group, while E/ E' septal and E/ E' lateral were statistically higher in diabetic patients than in the control group, shows that in this relatively young T1DM cohort, both ventricles are functioning poorer during diastole.

Zairi et al. study agreed with our results as diabetes patients had considerably smaller LV end-diastolic diameters than non-diabetics (40.87 5 vs. 42.94 1.43, respectively); $P < 0.01$)⁽¹¹⁾. Additionally, there were no discernible variations in TAPSE, RV free-wall thickness, 2D fractional area change, or RV end-diastolic diameter. According to **Kapeliou et al.** study, which supported our findings, LV diastolic dysfunction, LV concentric hypertrophy, and increased cardiac mass are common symptoms in type 1 diabetic individuals⁽¹²⁾. Similar to the findings of the **Suran et al.** research on LV echocardiographic measures, they found that T1DM patients had substantially higher IVS and LVPW thickness and LVEF than healthy controls. But **Suran et al.** disagree with us as they found that end diastolic LV dimension (LVIDd) is equal in both groups⁽¹³⁾.

In a study conducted in collaboration with us, **Weber et al.** found that the T1DM group had a longer deceleration time for E velocity as well as lower lateral and mean early diastolic velocities (lateral and mean E'), as measured by tissue Doppler, when assessing LV diastolic function⁽¹⁴⁾. Additionally, **Yoldas et al.** study demonstrated that the E/E' ratio was commonly utilized as a measure of diastolic dysfunction⁽¹⁵⁾, and **Bradley**

et al. found that diabetes patients' E/E' ratios were higher than those of non-diabetic participants⁽¹⁶⁾.

In their study, **Zairi et al.** agreed with us that the diabetes group's E' wave velocity at the septal mitral annulus showed considerably lower late diastolic myocardial velocity than the non-diabetic group's, but, they did so despite the fact that trans mitral flow did not significantly alter between the two research groups, contrary to our findings. However, in trans tricuspid flow, the diabetic group's E' wave velocity was considerably larger, although the E/E' ratio did not change between the two researches groups⁽¹¹⁾.

In addition, a recent study by **Kaushik et al.** showed that the population with T1DM had echocardiographic abnormalities consistent with preclinical ventricular dysfunction⁽¹⁷⁾. In particular, despite the lack of apparent HF and adequate EF, they discovered lower LV strain indices, mid-lateral LV, basal septum, and mid septum in children and adolescents with T1DM compared to non-diabetic controls. These results support the conception that the metabolic milieu of diabetes may have an early impact on the function of the myocardium. Recently, it was shown that diabetics had a higher-than-expected percentage of LV diastolic dysfunction (30.8%), compared to the age-matched non-diabetic group. In large observational echocardiographic research by **Redfield et al.** with 1091 T1DM patients (mean age 49.6 years, 53% men), there was a similar incidence of LV diastolic dysfunction in the general population in those over the age of 13⁽¹⁸⁾. Additionally, a study by **Fagan et al.** that included patients with T1DM who had it for longer than 50 years, surprisingly found that the E/E' septal ratio was only marginally higher in these patients than in controls⁽¹⁹⁾.

In our study right ventricular results showed that E' wave of the RV and A' wave of the RV fw were significantly lower in diabetics than controls. While TAPSE was statistically not significantly different between both groups.

Indicators of RV diastolic function, such as the tricuspid E and E/A ratio, were lower in the T1DM group than in the control group, as confirmed with us by **Weber et al.** study⁽¹⁴⁾. Additionally, **Suran et al.** concurred that TAPSE did not significantly differ between T1DM patients and healthy controls⁽¹³⁾. Additionally, **Karamitsos et al.**'s confirmation of decreased tricuspid E/A ratio and E- value in T1DM patients as compared to controls supported our findings⁽²⁰⁾.

In this study, we used 2D STE to evaluate all cardiac segments, including both ventricles. Furthermore, we examined endocardial (GLS endo), myocardial (GLS myo), and epicardial (GLS epi) global longitudinal strain in three perspectives: apical four, three and two. We detected that in diabetic group, endocardial, myocardial and average global longitudinal strain of left ventricle in apical four, three,

two views data were lower than control group while pericardial global longitudinal strain was lower only in apical three view and statically insignificantly different in apical four and two views.

Our results corroborate those of the **Berceanu et al.** study, which found that people with diabetes had considerably lower endocardial strain and mid wall strain than participants in the control group ⁽¹⁰⁾. According to **Zairi et al.**, who concurred with us, LV GLS was considerably lower in the group with diabetes than in the group without diabetes ⁽¹¹⁾. Furthermore, our findings agree with those of a pediatric investigation by **Labombarda et al.**, which assessed GLS in 100 T1DM children in comparison to 79 controls. In the T1DM group, longitudinal deformation was noticeably less ⁽²¹⁾.

In concordance with **Ghoreyshi-Hefzabad et al.** their met analysis showed that all 3D LV strain values were lower in diabetic patients compared to healthy participants ⁽²²⁾. It was similar to **Ringle et al.** they showed that, after six years of follow-up, only longitudinal strain by 2D STE has significantly decreased in persons with type 1 DM compared to healthy controls ⁽²³⁾.

Regarding right ventricular speckle tracking data our study showed that global right ventricular strain (endocardium, myocardium, and epicardium) data of the studied groups were statically insignificantly different between diabetic and non-diabetic.

Our data are concordant to **Berceanu et al.** who revealed that RV GLS derived from 6-segments analysis and RV free wall strain did not demonstrate any significant differences between diabetics and control group ⁽¹⁰⁾. Also, **Weber et al.** agreed with us and detected that RV GLS strains are similar in both groups ⁽¹⁴⁾. In concordant to our data, **Ahmed et al.** did not identify a significant difference in RVD or TAPSE between young T1DM patients and healthy controls ⁽⁸⁾.

But **Ahmed et al.** study was discordant with us and revealed that when the same patients underwent 2D-STE, it was discovered that diabetic individuals had RV systolic dysfunction (found by a decrease in RV global and segmental "basal, mid, and apical" longitudinal strain) ⁽⁸⁾. In disagreement with our findings **Zairi et al.** study ⁽¹¹⁾ showed that despite being within the normal range, RV speckle tracking values were lower in the diabetes group. Contrary to our research, **Kosmala et al.** and **Tadic et al.** studies ^(24,25) found that diabetic patients' RV global strain reduced as compared to the control group. In contrast with **Al-Biltagi et al.** study, in which they discovered statistically significant variations in the mean values of TAPSE, pulmonary artery pressure, RV GLS, MPI, and RV EF between the diabetic and non-diabetic groups, which supported the presence of subclinical RV systolic and diastolic dysfunction in 30 children with T1DM ⁽²⁶⁾.

In the current study, we discovered a negative association between LV strain and duration of diabetes mellitus with no significant correlation with other data. Similarly, **Zairi et al.** found negative correlation

between LV GLS and diabetes duration but against our data he found positive correlation with HBA1C⁽¹¹⁾.

In our study we did not find any correlation between ages and LV strain while **van Grootel et al.** found that LV strain is strongly associated with age ⁽²⁷⁾.

The study limitations are due to the study's very small sample size of participants. The possible layer-specific deformation problems in DM1 patients must be clarified in future investigations with bigger sample sizes and various times after DM1 diagnosis. Last but not least, despite prior research showing a high link between albuminuria and GLS, we lacked information on the presence and severity of albuminuria.

CONCLUSIONS

Layered-specific strain study of asymptomatic DM1 patients indicated that, despite unchanged ejection fraction, they exhibit a proportionate decrease in LV longitudinal systolic strain across the endocardium and myocardium.

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