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ORIGINAL ARTICLE

Epicardial Adipose Tissue as a Predictor of Subclinical Left Ventricular Dysfunction in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background: The incidence of type 2 diabetes mellitus(T2DM) is continuously raising, posing a significant threat to public health**. Aim:** To assess the correlation between left ventricular strain and epicardial adipose tissue (EAT) in cases suffering from T2DM, two-dimensional speckle technology (2D-STE) has been utilized. **Methods:** This cross-sectional investigation was carried out on 114 cases suffering from T2DM who have been categorized into 2 groups based on their left ventricular global longitudinal strain (GLS): The first group consisted of 57 T2DM cases with a GLS of less than eighteen percent, while the second group consisted of 57 T2DM cases with a GLS of eighteen percent or higher. **Results:** Increased thickness of EAT, Hba1c (%) and waist circumference were found to be independent predictors of worse (more negative) GLS. For every 1 mm increase in EAT, there was an associated 0.884% reduction in GLS ($p<0.001$). And each 1-centimeter increase in circumference of waist was related to a 0.133% lower GLS($p<0.001$). Additionally, each 1 % increase in glycosylated hemoglobina1c (hba1c) was related to a 0.44% lower GLS($p<0.001$). **Conclusions:** Impaired left ventricular function is correlated with thickened EAT in cases with type 2 diabetes. Accumulation of EAT can result in left ventricular diastolic dysfunction as well as left ventricular systolic dysfunction. By examining the correlation among left ventricular myocardial function and EAT in cases suffer from T2DM, we can develop a more profound comprehension of the pathogenesis of impaired cardiac function in T2DM cases.

Keywords: T2DM; Epicardial adipose tissue; Left ventricular strain

INTRODUCTION

The occurrence of T2DM is progressively increasing, which has become a significant threat to public health, as a result of the continuous enhancement of people's standards of living and the rapid development of economics. The cardiovascular complications of diabetes mellitus were the primary cause of mortality in cases suffer from T2DM, as demonstrated by prior research. [1].

Postulated mechanism of cardiovascular complications in diabetes mellitus is that the following factors contribute to their development: an overload of calcium in cardiomyocytes, oxidative stress, insulin resistance, the accumulation of advanced glycation end products as a result of hyperglycemia, elevated metabolism of fatty acid, and abnormalities in the renin-angiotensin system [2,3].

Recently, there has been a growing interest in the impact of epicardial adipose tissue on the cardiovascular system, in addition to these generally accepted causes. Epicardial adipose tissue is a unique metabolically active visceral adipose tissue that is situated between the myocardium and the visceral pericardium. It is derived from the splanchno-pleuric mesoderm. A growing body of evidence indicates that epicardial adipose tissue has been suggested as a new cardiovascular risk factor. [4, 5].

Currently, a growing body of evidence suggests that left ventricular dysfunction has been related to epicardial adipose tissue in excess in a variety of illness states. Nevertheless, there is a scarcity of investigation in the literature regarding the correlation between epicardial adipose tissue and function of left ventricle in the presence of T2DM. [6,7]

An important technique is called 2D-STE. It allows for the offline calculation of deformation parameters and myocardial velocities, including strain and strain rate (SR), similar to tissue Doppler imaging (TDI). It is widely recognized that these parameters offer significant insights into the pathophysiological processes of the heart, including myocardial mechanics, ischemia, diastolic and systolic function, and many others [8].

The aim of the work was to evaluate the correlation between epicardial adipose tissue and left ventricular LV strain in cases suffer from T2DM using two-dimensional speckle technology.

METHODS

This cross-sectional investigation was performed on 114 cases suffering from T2DM who have been categorized into 2 groups based on their left ventricular global longitudinal strain: fifty-seven cases sufferings from T2DM and a global longitudinal strain of less than eighteen percent were enrolled in Group one, while fifty-seven case with a global longitudinal strain of eighteen percent or higher were enrolled in Group two. T2DM was diagnosed according to the 2010 guidelines of the American Diabetes Association (ADA) [9]. The investigation has been performed at the Cardiology Department of Zagazig University Hospitals.

Sample Size

Assuming the mean RV LSR-E was 1.93±0.32 vs 1.75±0.36 in control vs cases. At eighty percent power and ninety-five percent confident interval, the estimated sample will be 114 subjects, 57 subjects in each group.

Inclusion criteria: Patients from both genders with age ≥18 years who had T2DM with preserved left ventricular ejection fraction (LVEF).

***Exclusion criteria*:** Patients having any of the followings: patients with valvular heart illness, gestational diabetes, any degree of kidney illness, coronary heart illness, cases suffering from type 1 diabetes mellitus, cases with left ventricular ejection fraction less than fifty percent, cardiomyopathy, congenital heart illness, cerebrovascular illness and severe arrhythmia and cases suffering from acute complications of diabetes.

Methods

Each patient included in the investigation was subjected **to:** Full history taking, clinical examination, routine laboratory investigations in addition to HbA1c, first midstream morning urine was collected to assess urine microalbumin (UA) to urine creatinine ratio (UA/CR), and twelve lead electrocardiography. All patients also had detailed fundus examination, neurological assessment by neurologist and ankle brachial index to exclude peripheral vascular disease.

***Conventional Transthoracic echocardiography*:** An experienced cardiologist conducted transthoracic echocardiography examinations on all cases suffering from type 2 diabetes mellitus in the left lateral decubitus position using the GE Vivid E9 echocardiography equipment (Vivid E9, GE Healthcare, states). The following echocardiography parameters have been determined in cases suffering from type 2 diabetes: left atrial diameter (LAD), left ventricular posterior wall (LVPWD) and interventricular septum (IVSD) diameters in the end-diastolic period, , left ventricular end-systolic diameter (LVESD), LVEF, left ventricular end-diastolic diameter (LVEDD), early (E) and late (A) transmitral inflow velocity, early diastolic peak velocity of the septal of the mitral annulus (e' septal and early diastolic peak velocity of the lateral mitral annulus (e' lateral),). The septal and lateral mitral annuli were averaged to determine the early diastolic myocardial velocity (e). The ASE/EACVI recommended a diastolic dysfunction cut-off of E/e' less than 14 when evaluating left ventricular diastolic function utilizing the E/e ratio [10].

Echocardiographic measurement of EAT thickness:

In three cardiac cycles, thickness has been determined at end-systole in the parasternal long axis view perpendicular to the aortic annulus and in the parasternal short-axis view perpendicular to the ventricular septum at mid-chordal and tip of the papillary muscles level [11]. For epicardial adipose tissue estimation, the investigation utilized triplicate measurements, with the mean value being utilized for the examination. The individual has been positioned in the left lateral decubitus position, and an optimal parasternal long-axis view has been obtained by observing the left sternal two to three intercostal space. The aortic root and interventricular septum have been used as reference points for the assessment. The aortic annulus and right ventricular free wall have been positioned in the midline of the ultrasound pulses. The thickest level of the hypoechoic area, which extends from the epicardial surface to the parietal pericardium, had been vertically evaluated. In order to more accurately evaluate the thickness of EAT, the depth setting and the magnification view were improved. The mean value was determined by measuring the data in at least 3 cardiac cycles, as a single estimation was not satisfactory. Reference points have been obtained from the mid-chordal region, papillary muscle tip, and interventricular septum to acquire the parasternal short-axis view. A measurement has

been taken of the echo-lucent area between the parietal pericardium and the right ventricle.

2-D Speckle tracking echocardiography: The left ventricular global longitudinal strain has been determined using two-dimensional speckle technology [12]. Images of the apical 4-chamber, 2-chamber, and long-axis have been obtained during an end-expiratory breath-hold for 3 consecutive cardiac cycles. The Echopac software (2D-Strain, EchoPac PC v.7.x.x, GE Healthcare, Horten, Norway) was used to transmit all images. The aortic valve closure time had been subsequently verified in the view of apical long axis, and the endocardial curve has been drawn in the apical 4 chamber, apical 2-chamber, and long-axis views, respectively. Then, the software automatically generated a region of interest and adjusted it to ensure that the myocardial wall was adequately represented. The left ventricular wall has been separated into a total of eighteen parts by the software, which automatically segmented the myocardium into six parts. The average of the peak systolic longitudinal strain of all segments has been denoted as global longitudinal strain and represented as an absolute value. As previously stated, a value of longitudinal systolic strain of less than eighteen percent was suggestive of preclinical systolic dysfunction [13]. In order to assess intra-observer and inter-observer variability, the same physician (intra-observer variability) and another physician (inter-observer variability) repeated the assessments of global longitudinal strain, epicardial adipose tissue, and E/e in thirty subjects that have been randomly selected.

Ethical approval: Approval from the Faculty of Medicine's ethical committee (Institutional Research Board IRB). Number of IRB approval: 10882-14-6-2023. Written informed assent has been obtained from each case.

STATISTICAL ANALYSIS

All data was collected, tabulated and statistically analyzed using SPSS 20.0 for windows (SPSS Inc., Chicago, IL, USA). Quantitative data were expressed as the mean ± SD & median (range), and qualitative data were expressed as absolute frequencies (number) & relative frequencies

RESULTS

This cross-sectional investigation has been conducted in Cardiology Department, Zagazig University Hospitals on 114 T2DM to examine the correlation between thickness of epicardial adipose tissue and function of left ventricular in cases suffer from T2DM utilizing (2D-STE). The study included 114 patients separated into two groups: Group I: T2DM cases with GLS of eighteen percent or less (57patients) and Group II: T2DM patients with global longitudinal strain of eighteen percent or high (57patients).

Table 1 compares baseline characteristic and anthropometric data between both categories. Group I with GLS <18% was significantly younger with a mean age of 53.54±2.78 years compared to 56.40±3.28 years in group II with GLS ≥eighteen percent (p=value less than 0.001). There were insignificant variances in gender distribution or T2DM duration between both groups. In terms of co-morbidities, while there were numerically more smokers (40.4% vs 31.6%, p=0.329) and more cases suffering hypertension (50.9% vs 45.6%, p-value equal 0.452) and dyslipidemia (45.6% vs 52.6%, p=0.454) in group I, these differences did not reach statistical significance. Also, Table 1 shows comparison of anthropometric data between T2DM patients categorized by GLS. The group with reduced GLS (<18%) had significantly higher mean body weight (89.44±8.93 kg vs 79.93±8.45 kg, p-value less than 0.001), body mass index (BMI) (32.39±4.99 kilogram/meter^2 vs 28.65±4.29 kg/m^2, p<0.001) and circumference of waist (90.02±3.15 centimeters vs 79.79±2.52 cm, p-value less than 0.001) compared to those with preserved GLS (≥18%). There was insignificant variance in height among both groups.

Patients with depressed GLS (<18%) had significantly greater mean systolic blood pressure (139.44±5.05 mmHg) compared to the normal GLS (≥18%) group (129.18±5.62 mmHg) (p-value less than 0.001). However, there was insignificant variance in diastolic BP. Among laboratory data, Group I had markedly higher LDL cholesterol (113.79±17.86 vs 102.88±11.16, p=0.003), HbA1c (10.00±1.41% vs 9.00±0.85%, p<0.001) and urine microalbumin (38.89±12.88 vs 30.86±12.08, p-value less than 0.001) when compared with group II. Total cholesterol, serum creatinine and triglycerides levels did not substantially differ among both groups (Table 2).

Regarding the prevalence of microvascular/macrovascular complications and medication use in T2DM patients categorized by GLS values, there were insignificant differences in the rates of diabetic retinopathy, neuropathy, peripheral vascular disease or nephropathy between the GLS <18% and GLS ≥18% groups. Moreover, the usage of various anti-diabetic medications (including insulin, metformin, sulfonylureasDPP4 inhibitors, GLP-1 receptor agonists, SGLT2 inhibitors, and thiazolidinediones), antihypertensive agents (ACEi/ARB, CCB, diuretics, beta-blockers) and statins did not markedly vary based on GLS value (Table 3).

Table 4 shows comparison of standard echocardiographic parameters between the reduced GLS (<18%) and preserved GLS (≥18%) groups. As expected based on group definitions, patients with GLS <18% had markedly lower mean GLS (15.93±0.90% vs 19.63±1.19%, p<0.001) compared to those with GLS ≥18%.

Additionally, the GLS <18% group demonstrated worse diastolic dysfunction as evidenced by an elevated E/e ratio (9.83±1.12 vs 8.28±0.62, p<0.001). There was increased thickness of epicardial adipose tissue (EAT) among GLS <18% patients (7.42±0.49mm vs 5.55±0.53mm, p-value less than 0.001). However, there were insignificant inter-group variances in traditional measures of systolic function (LVEF, LV dimensions) or parameters reflective of cardiac remodeling (left atrial size, LV wall thickness).

In order to identify factors that are independently correlated with left ventricular (GLS) function in cases suffering from (T2DM), we performed a multiple linear regression model. After adjustment for other covariates, elevated epicardial adipose tissue thickness, Hba1c and circumference of waist have been detected to be significant independent predictors of worse (more negative) GLS. For every 1 mm increase in EAT, there was an associated 0.884% reduction in GLS (p-value less than 0.001). Additionally, each one-centimeter rise in circumference of waist was related to a 0.133% lower GLS (p<0.001) (Table 5).

We then performed multiple linear regression analysis to detect predictors of the echocardiographic parameter E/e, which represents an estimate of left ventricular filling pressure (Table 6). After adjustment for confounding variables, EAT and Hba1c (%) were the sole independent predictor of elevated E/e ratio. For every 1 mm thicker EAT, there was a corresponding 0.416 unit rise in E/e (p=0.004). For every 1 % increase hba1c (%), there was a corresponding 0.051 unit rise in E/e (p=0.003). Other clinical factors like age, blood pressure, waist circumference and anthropometric measurements did not show significant associations with E/e.

We performed ROC curve analysis for EAT to predict LV dysfunction by GLS. At cut-off point 6.4650, EAT had 96% Sensitivity and 94% specificity with high significance in detection of LV dysfunction by GLS (AUC = 0.970, P -value less than 0.001, CI 0.941-0.999) (Figure 1)

Table 1: Demographic and Anthropometric data of studied groups: patients with GLS <18% and ≥ 18%.

Variables		Group				*p value*
		Group I T2DM patients with GLS <18% (N=57)		**Group II T2DM patients with GLS ≥18% (N=57)**		
Demographic						
Age (years)		56.40 ± 3.28		53.54 ± 2.78		**<0.001***
Gender	male	32	56.1%	34	59.6%	0.704
	female	25	43.9%	23	40.4%	
T2DM duration (years)		8.19 ± 2.14		7.96 ± 2.23		0.578
Co-morbidity						
Smoking		18	31.6%	23	40.4%	0.329
Hypertension		33	57.9%	29	50.9%	0.452
Dyslipidemia		30	52.6%	26	45.6%	0.454
Anthropometric Measurement						
Height (m)		1.67 ± .09		1.68 ± .08		0.644
Weight (kg)		89.44 ± 8.93		79.93 ± 8.45		**<0.001***
BMI (kg/m2)		32.39 ± 4.99		28.65 ± 4.29		**<0.001***
waist circumference (cm)		90.02 ± 3.15		79.79 ± 2.52		**<0.001***

Abbreviations: T2DM: type 2 diabetes mellitus; GLS: global longitudinal strain; BMI: body mass index; Data represented as mean ±SD

SD: **Standard deviation**,

p: p value for comparing between the two studied groups.

*: Statistically significant at p ≤ 0.05

Table 2: Clinical and laboratory data between both groups

Variables	Group 1 T2DM patients with GLS <18% (N=57)	Group 2 T2DM patients with GLS ≥18% (N=57)	*p value*
Clinical data			
SBP (mm/Hg)	139.44 ± 5.05	129.18 ± 5.62	<0.001*
DBP (mm/Hg)	82.11 ± 8.13	79.98 ± 7.22	0.065
Laboratory data			
Total cholesterol	207.28 ± 20.08	207.67 ± 16.87	0.832
Triglycerides	174.58 ± 21.31	171.84 ± 19.42	0.511
LDL-C	113.79 ± 17.86	102.88 ± 11.16	0.003*
HbA1c (%)	10.00 ± 1.41	9.00 ± 0.85	<0.001*
Serum creatinine (mg/dL)	1.06 ± 0.38	1.07 ± 0.36	0.911
Urine microalbumin (mg/L)	38.89 ± 12.88	30.86 ± 12.08	<0.001*

Abbreviations: T2DM: type 2 diabetes mellitus; GLS: global longitudinal strain; SBP: systolic blood pressure; DBP: diastolic blood pressure; LDL-C: low-density lipoprotein cholesterol; HbA1c: glycosylated hemoglobinA1c. Data represented as mean ±SD, SD: **Standard deviation**, p: p value for comparing between the two studied groups. *: Statistically significant at $p \leq 0.05$

Table (3): T2DM complications and medications between patients with GLS <18% and ≥ 18%.

Variables	Group 1 T2DM patients with GLS <18% (N=57)		Group 2 T2DM patients with GLS ≥18% (N=57)		*p value*
Retinopathy	23	40.4%	22	38.6%	0.848
Neuropathy	31	54.4%	27	47.4%	0.454
Peripheral vascular disease	26	45.6%	19	33.3%	0.250
Nephropathy	15	26.3%	9	15.8%	0.168
Medication					
ACEi/ARB	26	45.6%	25	43.9%	0.851
CCB	18	31.6%	17	29.8%	0.839
Diuretics	8	14.0%	7	12.3%	0.782
β-Blocker	9	15.8%	13	22.8%	0.342
Statins	22	38.6%	27	47.4%	0.344
Insulin	42	73.7%	37	64.9%	0.310
SGLT-2I	33	57.9%	33	57.9%	1.000
DPP-4I	23	40.4%	19	33.3%	0.437
GLP-1RA	17	29.8%	20	35.1%	0.548
Metformin	38	66.7%	38	66.7%	1.000
Sulfonylureas	9	15.8%	11	19.3%	0.622
Thiazolidine	13	22.8%	12	21.1%	0.821

Abbreviations: T2DM: type 2 diabetes mellitus; GLS: global longitudinal strain; ACEi/ARB: angiotensin converting enzyme inhibitor/angiotensin II receptor blockers; CCB: calcium channel blockers; SGLT-2I: sodium–glucose co-transporter-2 inhibitors; DPP-4I: dipeptidyl peptidase-4 inhibitor; GLP-1RA: glucagon like peptide-1receptor agonist. Data represented as number (percentage)

SD: **Standard deviation**,

p: p value for comparing between the two studied groups. *: Statistically significant at $p \leq 0.05$

Table 4: Comparison of conventional echocardiographic characteristics between patients with GLS <18% and ≥ 18%.

Variables	Group		*p value*
	T2DM patients with GLS <18% (N=57)	T2DM patients with GLS ≥18% (N=57)	
Conventional echocardiographic characteristics			
LAD (mm)	36.35 ± 1.97	35.81 ± 1.99	0.151
IVSD (mm)	9.16 ± 0.64	9.27 ± 0.60	0.306
LVPWD (mm)	8.15 ± 0.69	8.21 ± 0.61	0.677
LVEDD (mm)	47.32 ± 1.22	47.51 ± 1.39	0.378
LVESD (mm)	32.64 ± 1.40	32.37 ± 1.01	0.209
LVEF (%)	60.72 ± 3.80	59.84 ± 3.25	0.201
E/A	0.96 ± 0.01	0.97 ± 0.12	0.052
GLS (%)	15.93 ± 0.90	19.63 ± 1.19	<0.001*
E/e	9.83 ± 1.12	8.28 ± 0.62	<0.001*
EAT (mm)	7.42 ± 0.49	5.55 ± 0.53	<0.001*

Abbreviations: LAD: left atrial diameter; IVSD: interventricular septal thickness in end-diastolic period; LVPWD: left ventricular posterior wall thickness in enddiastolic period; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; LVEF: left ventricular ejection fraction; E: early diastolic mitral inflow velocity; A: late diastolic mitral inflow velocity; e:average mitral annular velocity; EAT: epicardial adipose tissue; GLS: global longitudinal strain.. Data represented as mean ±SD SD: **Standard deviation**, p: p value for comparing between the two studied groups. *: Statistically significant at p ≤ 0.05

Table 5: Independent predictors of left ventricle GLS in multiple linear regression analysis.

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval for B	
	B	Std. Error	Beta	T	Sig.	Lower Bound	Upper Bound
Age (years)	.026	.034	.046	.783	0.435	-.040	.093
BMI (kg/m2)	-.020	.021	-.053	-.956	0.341	-.063	.022
waist circumference (cm)	-.133	.030	-.408	-4.385	<0.001*	-.193	-.073
SBP (mm/Hg)	.015	.020	.057	.750	0.455	-.024	.054
LDL-C	-.008	.007	-.069	-1.228	0.222	-.022	.005
HbA1c (%)	-.044	.085	-.029	-.518	<0.05*	-.213	.125
EAT (mm)	-.884	.152	-.494	-5.810	<0.001*	-1.186	-.582

Table 6: Independent predictors of E/e in multiple linear regression analysis.

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval for B	
	B	Std. Error	Beta	T	Sig.	Lower Bound	Upper Bound
Age (years)	-.025	.031	-.070	-.800	0.425	-.087	.037
BMI (kg/m2)	.020	.020	.085	1.008	0.316	-.019	.060
waist circumference (cm)	.025	.028	.125	.897	0.372	-.031	.082
SBP (mm/Hg)	.023	.018	.143	1.247	0.215	-.014	.060
LDL-C	-.003	.006	-.038	-.456	0.650	-.015	.010
HbA1c (%)	.051	.080	.054	.646	0.003*	-.106	.209
EAT (mm)	.416	.142	.374	2.925	0.004*	.134	.698

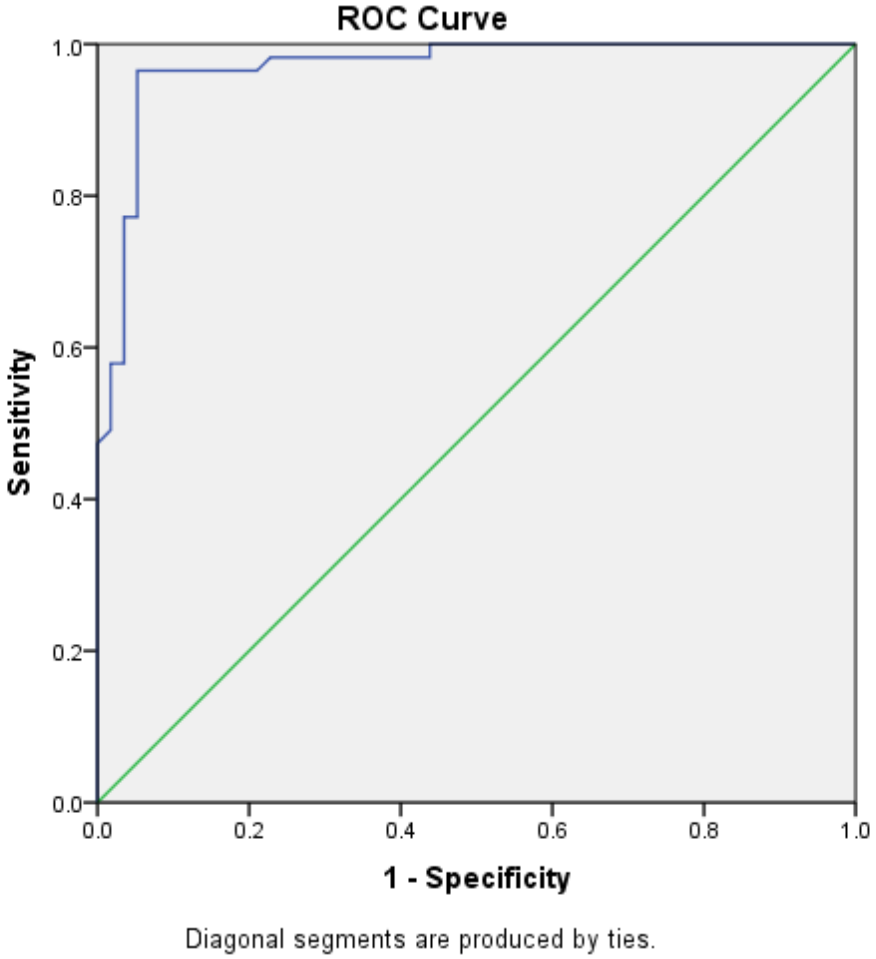

Fig 1: ROC curve analysis of epicardial adipose tissue to detect LV dysfunction by GLS.

DISCUSSION

The current investigation reported that group II with GLS ≥18% was significantly younger with a mean age of 53.54±2.78 years compared to 56.40±3.28 years in group I with GLS <eighteen percent (p-value less than 0.001). While there were insignificant variances in distribution of sex or T2DM duration between both groups.

The present investigation were in agreement with Song et al.**,** who investigated the correlation n among left ventricular function and epicardial adipose tissue in cases suffering from type 2 diabetes mellitus utilizing 2D-STE. They revealed that patients with GLS <18% was significantly older with a mean age of 58.08 ± 4.55 years compared to years 53.27 ± 7.03 in patients with GLS ≥18% (p-value less than 0.001). While there were statistically insignificant variances among both groups under investigation regarding gender and T2DM duration [14].

As regards co-morbidities, our findings revealed that there were numerically more smokers (40.4% vs 31.6%, p-value equal 0.329) in group II while more cases with hypertension (57.9percent vs 50.9%, p-value equal 0.452) and dyslipidemia (52.6% vs 45.6%, p=0.454) in group I, these differences did not reach statistical significance.

Similarly, this study in consistent with Song et al., who reported that there were statistically insignificant variances between cases with global longitudinal strain less than eighteen percent and cases with global longitudinal strain of eighteen percent or higher regarding co-morbidities including smoking, hypertension and dyslipidemia [14].

The recent investigation demonstrates that cases with depressed global longitudinal strain (<18%) had significantly elevated mean systolic blood pressure compared to the normal GLS (≥18%) group (p-value less than 0.001). However, there was insignificant variance in diastolic BP. As regards laboratory data, our findings reported that Group I had significantly higher LDL cholesterol, hba1c and urine microalbumin when compared with group II. Total cholesterol, triglycerides and serum creatinine levels did not substantially differ among both groups.

This study agreed with Kim et al., who revealed that Subjects with reduced GLS had higher diastolic and systolic blood pressures compared to those who have global longitudinal strain within normal range . As regards laboratory findings, they found that patients with reduced GLS (<18%) had significantly greater hba1c compared to cases with GLS ≥18%. While there was statistically insignificant variance among both groups under investigation in term of total cholesterol [15].

According to echocardiographic parameters, as expected based on group definitions, our study demonstrated that patients with GLS <18% had markedly lower mean GLS (15.93±0.90% vs 19.63±1.19%, p<0.001) compared to those with GLS ≥18%.

The current investigation in agreement with Kim et al., who revealed that mean values of global longitudinal strain were 16.3 percent ±1.4% and 20.2 percent ±1.6 percent in subjects who have decreased global longitudinal strain and with global longitudinal strain within normal range, respectively (P-value less than 0.001) [15].

The current study revealed that the GLS <18% group demonstrated worse diastolic dysfunction as evidenced by an elevated E/e ratio (9.83±1.12 vs 8.28±0.62, p<0.001). There was increased thickness of epicardial adipose tissue (EAT) among GLS <18% patients (7.42±0.49mm vs 5.55±0.53mm, p-value less than 0.001). However, there were insignificant inter-group variances in traditional measures of systolic function (LVEF, LV dimensions) or parameters reflective of cardiac remodeling (left atrial size, LV wall thickness).

Similarly, this results were in concordance with Song et al., who stated that the thickness of epicardial adipose tissue and the value of E/e were greater in cases with global longitudinal strain less than eighteen percent compared to those with global longitudinal strain of eighteen percent or high (p-value less than 0.05). Although there were statistically insignificant variances in the LVEDD, IVSD, LVEF, LVPWD, LVESD, LAD, and E/A between cases with global longitudinal strain [14].

The present study revealed that increased thickness of EAT, hba1c (%) and circumference of waist have been detected to be significant independent predictors of worse (more negative) GLS. For every 1 mm increase in EAT, there was an associated 0.884% reduction in GLS (p-value less than 0.001). And each one-centimeter rise in circumference of waist was related to a 0.133% lower GLS (p<0.001). Additionally, each 1 % increase in glycosylated hemoglobina1c (hba1c) was related to a 0.44% lower GLS (p<0.001).

Also, our outcomes in accordance with Song et al., who showed that BMI, SBP, age, LDL-C, hemoglobin a1c, and epicardial adipose tissue have been independently correlated with left ventricular global longitudinal strain in a multivariate linear regression analysis among clinical characteristics and function of left ventricular [14].

EAT and Hba1c (%) were the sole independent predictor of elevated E/e ratio. For every 1 mm thicker EAT, there was a corresponding 0.416 unit rise in E/e(p=0.004). For every 1 % increase hba1c (%), there was a corresponding 0.051unit rise in E/e(p=0.003).

Our outcomes in accordance with Nabati et al., who determined the association among thickness of epicardial fat and longitudinal LA reservoir strain (LARS) in cases suffer from type 2 diabetes mellitus. They revealed that a positive association was observed between epicardial fat thickness and the E/e' ratio (r=0.299, p-value equal 0.020) [16].

The current study demonstrated that at cut-off point 6.4650, EAT had 99.7% Sensitivity and 98% Specificity with high significance in detection of LV dysfunction by GLS.

This study agreed with Pararajasingam et al., who investigated the correlation between microvascular complication, and GLS in cases with diabetes mellitus and non-obstructive coronary artery disease (CAD) who were asymptomatic. They found that the data on global longitudinal strain had an intra-variability value of 0.42 [−1.75–2.59] and an value of inter-variability of −0.22 [−2.75–2.30]. Additionally, the coefficients of association that corresponded to these values were 0.81 and 0.75, respectively [17].

CONCLUSION

In cases suffer from T2DM, the accumulation of EAT is directly correlated with LV dysfunction, which is independent of other cardiovascular risk factors. Accumulation of epicardial adipose tissue can result in left ventricular diastolic dysfunction as well as left ventricular systolic dysfunction. As a result, it is essential to do research on the correlation EAT tissue and the function of the LV in cases suffer from type 2 diabetes mellitus. Using this information, we can develop new hypoglycemic medicines that target epicardial adipose tissue in order to lower the possibility of cardiovascular problems in cases suffer from type 2 diabetes mellitus.

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