



ASSESSMENT OF EPICARDIAL ADIPOSE TISSUE AND LEFT VENTRICULAR PERFORMANCE INTERACTION IN TYPE 2 DIABETES MELLITUS USING 2D SPECKLE-TRACKING ECHOCARDIOGRAPHY

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is a major risk factor for diabetic cardiomyopathy, often presenting with subclinical left ventricular (LV) dysfunction before overt heart failure. Epicardial adipose tissue (EAT), a metabolically active fat depot, is increasingly implicated in cardiac dysfunction. **Aim:** To assess the interaction between EAT thickness and LV performance in patients with T2DM using 2D speckle-tracking echocardiography (2D-STE). **Methods:** An Observational study was conducted on T2DM patients. Participants were stratified based on global longitudinal strain (GLS) into two groups: those with preserved systolic function (GLS $\geq 18\%$) and those with impaired systolic function (GLS $< 18\%$). All subjects underwent comprehensive echocardiography, including 2D-STE for GLS measurement and standard 2D echocardiography for assessing LV structure, diastolic function, and EAT thickness. **Results:** The mean & SD of age among our participants is 47.5 ± 12.7 years. Patients with impaired GLS (GLS $< 18\%$) had a significantly longer duration of diabetes, higher HbA1c and BMI compared to those with preserved GLS. The impaired GLS group had significantly lower LVEF (58.3% vs. 61.6%, $p=0.0001$), elevated LV filling pressures (E/e' 13.3 vs. 10.13, $p=0.0001$), and altered diastolic filling (E/A 1.28 vs. 1.14, $p=0.0006$). Crucially, EAT thickness was significantly greater in the impaired GLS group (6.46 mm vs. 4.36 mm, $p=0.0001$). **Conclusion:** GLS is significantly impacted by LV systolic and diastolic dysfunction, with increased EAT thickness linked to impaired GLS, suggesting a potential link between pericardial fat and diabetic cardiomyopathy.

KEYWORDS : Global longitudinal strain, EAT and Cardiac dysfunction.

INTRODUCTION:

Millions of people worldwide suffer from type 2 diabetes mellitus (T2DM), which raises the risk of cardiovascular morbidity and death dramatically [1]. One well-known consequence of type 2 diabetes is diabetic cardiomyopathy, a unique type of myocardial dysfunction that manifests without the presence of valvular disease, hypertension, or coronary artery disease [2]. This condition has a complex pathophysiology that includes inflammation, microvascular dysfunction, and metabolic abnormalities. The existence of epicardial adipose tissue (EAT), a visceral fat depot situated between the myocardium and the visceral pericardium, is a crucial element of this intricate pathophysiology [3]. A number of pro-inflammatory cytokines and adipokines are released by EAT, which is now thought to be a metabolically active endocrine organ. These substances can have direct paracrine effects on the underlying myocardium, possibly leading to cardiac dysfunction [4].

For the early identification and treatment of cardiac dysfunction in patients with type 2 diabetes, evaluation of left ventricular (LV) function is essential. Despite their widespread use, conventional echocardiographic metrics like left ventricular ejection fraction might not be sensitive enough to detect subclinical alterations in myocardial contractility [5]. An advanced, non-invasive imaging technique called two-dimensional speckle-tracking echocardiography (2D STE) offers a quantitative and objective evaluation of myocardial deformation (strain), making it possible to identify minute alterations in global and regional left ventricular function before overt clinical symptoms manifest [6]. This approach is a useful tool in the assessment of high-risk groups, including those with type 2 diabetes, because it can detect myocardial dysfunction in its early stages.

The precise mechanisms and direct interaction by which EAT contributes to impaired LV performance, as measured by the sensitive and sophisticated technique of 2D STE, are still unclear despite mounting evidence linking both T2DM and EAT to cardiovascular disease. Determining new biomarkers and therapeutic targets for the prevention and treatment of

diabetic cardiomyopathy requires an understanding of this relationship. With this background we aimed to use 2D STE to assess the interaction among EAT & LV performance in individuals with T2DM.

Methodology:

This study was designed as a prospective observational study conducted at Chettinad Super Speciality Hospital at department of Cardiology in Kelambakkam.

A total of 50 consecutive individuals diagnosed with T2DM, systemic hypertension (SHTN), and dyslipidemia were included based on predefined inclusion and exclusion criteria. The inclusion criteria comprised T2DM, hypertension, and dyslipidemia, while exclusion criteria included type 1 diabetes mellitus (T1DM), pregnancy-related diabetes, valvular heart disease, congenital heart disease (CHD), and coronary artery disease (CAD).

The study procedure involved two-dimensional echocardiography using a Philips Affinity 50C machine, with standard echocardiographic views such as parasternal long-axis (PLAX), parasternal short-axis (PSAX), apical four-chamber (A4CH), and apical two-chamber (A2CH).

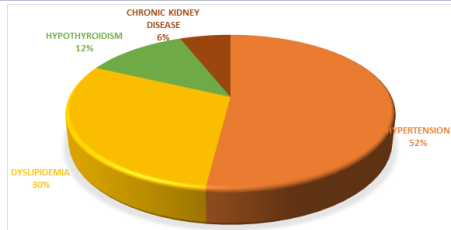
EAT thickness was measured in PLAX and PSAX views. LV systolic function was evaluated using speckle-tracking echocardiography (STT), while LV diastolic function was assessed through mitral valve (MV) inflow and tissue Doppler imaging.

RESULTS & OBSERVATION:

Table 1: Characteristics Of Patients

Variable	No of Patients (n = 50)	
Age (years)	47.51 ± 12.71	
Gender	Male	29 (62)
	Female	21 (38)

The mean & SD of age among our study participants is 47.51 ± 12.71 years and the majority of the participants were male.



Among 50 patients 52% had hypertension, 30% had dyslipidemia, 12% had hypothyroidism and followed by chronic kidney disease among 6%.

Table 2: Analyzing baseline characteristics in individuals with GLS less than 18% and those with GLS more than or equal to 18%

PARAMETERS	GLS < 18% PATIENTS (N = 20)	GLS ≥ 18% PATIENTS (N = 30)
HbA1c (%)	7.50 ± 0.62	7.24 ± 0.53
T2DM Duration	9.15 ± 4.22	4.5 ± 2.47
BMI	26.75 ± 1.57	25.6 ± 1.46

Patients in this group GLS < 18% had a significantly longer duration of Type 2 Diabetes (9.15 vs. 4.5 years) and also exhibited marginally poorer long-term glycaemic control (HbA1c of 7.50% vs. 7.24%) and a higher average body weight (BMI of 26.75 vs. 25.6), indicating that worse glucose management and higher BMI may act as synergistic risk factors that contribute to the development of diabetic cardiomyopathy over time.

Table 3: Comparison Of The Attributes Of Conventional Echocardiography In Individuals With GLS Less Than 18% And More Than Or Equal To 18%

VARIABLES	GLS < 18% PATIENTS (N = 20)	GLS ≥ 18% PATIENTS (N = 30)	p-value
LA DIAMETER (mm)	31.15 ± 3.11	31.5 ± 3.34	0.7108
IVSD (mm)	9.65 ± 1.08	9.46 ± 7.58	0.9121
LVPWD (mm)	10.55 ± 0.88	10.13 ± 6.43	0.7736
LVEDD (mm)	40.95 ± 3.57	40.5 ± 3.14	0.6405
LVESD (mm)	29.05 ± 2.76	30.1 ± 3.26	0.2422
LVEF (%)	58.3 ± 1.9	61.6 ± 1.28	0.0001*
GLS (%)	16.65 ± 1.13	19.96 ± 1.32	0.0001*
E/A	1.28 ± 0.15	1.14 ± 0.12	0.0006*
E/e'	13.3 ± 1.3	10.13 ± 1.38	0.0001*
EAT (mm)	6.46 ± 1.3	4.36 ± 1.32	0.0001*

*p = <0.05 considered as significant

Our analysis revealed a critical finding, while conventional echocardiographic measures of cardiac structure—including left atrial size (LA Diameter), wall thickness (IVSD, LVPWD), and chamber dimensions (LVEDD, LVESD)—show no significant differences between the groups, all functional and hemodynamic parameters are markedly impaired in patients with a GLS of less than 18%. This group exhibits a significantly lower, though still technically "normal," LVEF (58.3% vs. 61.6%, p=0.0001), confirming that GLS detects subtle systolic dysfunction earlier than traditional ejection fraction. Furthermore, the impaired GLS group demonstrates clear evidence of diastolic dysfunction, with significantly elevated left ventricular filling pressures (E/e' 13.3 vs. 10.13, p=0.0001) and an altered diastolic filling pattern (E/A 1.28 vs. 1.14, p=0.0006). The significantly greater epicardial adipose tissue (EAT) in this group (6.46 mm vs. 4.36 mm, p=0.0001) suggests a potential link between pericardial fat and subclinical myocardial impairment.

DISCUSSION:

The study reveals that patients with Type 2 Diabetes Mellitus experience significant interplay between metabolic markers,

epicardial adipose tissue (EAT), and subclinical cardiac dysfunction. This impairment is linked to higher body weight, poorer glycaemic control, longer diabetes duration, and thicker EAT, potentially mediating the onset of early diabetic cardiomyopathy.

We found that even with a technically "normal" left ventricular ejection fraction (LVEF) of 58.3%, GLS is compromised, which is in line with an increasing amount of research. GLS is a more sensitive marker for identifying subtle myocardial dysfunction earlier than LVEF, which frequently stays intact until later stages of the disease, according to studies by Sade et al. [7] and Yin Y et al. [8]. This emphasises the importance of early risk stratification in asymptomatic T2DM patients using speckle-tracking echocardiography. It is consistent with earlier studies showing diastolic dysfunction as an early and prevalent sign of diabetic cardiomyopathy that the impaired GLS group also showed obvious signs of diastolic dysfunction, as evidenced by elevated E/e' and an altered E/A ratio [9,10].

The central finding of a significant increase in EAT thickness in the group with impaired cardiac function corroborates the hypothesis that EAT is not merely an inert fat depot but a metabolically active organ influencing the adjacent myocardium. Independent of overall obesity, research from multiple groups indicates a direct correlation between EAT volume or thickness and compromised cardiac mechanics [7, 11]. According to a review by Zhu et al., EAT can release pro-inflammatory cytokines that cause myocardial alterations like fibrosis and cardiomyocyte dysfunction because of its close proximity to the myocardium and shared microvasculature [12]. According to our findings, the impaired group's mean EAT thickness of 6.46 mm falls well within the range of values that have been connected to subclinical LV dysfunction in comparable patient populations. Additionally, the synergistic risk factors of BMI, glycaemic control, and diabetes duration are clinically validated by our study. Patients with impaired GLS had higher average BMIs, worse long-term glycaemic control (higher HbA1c), and a noticeably longer duration of diabetes. These results are consistent with those of other studies that have separately connected each of these variables to heart failure in people with type 2 diabetes. For instance, studies by Di Pino et al. and Singh A et al. have shown a strong positive relationship between the presence and severity of left ventricular diastolic dysfunction and elevated HbA1c levels [9, 13].

These variables' convergence in our impaired-GLS cohort points to a progressive and cumulative damage pathway, in which lipotoxicity and chronic hyperglycemia fuel the growth and metabolic dysregulation of EAT, ultimately resulting in the myocardial impairment that has been observed.

Despite the compelling nature of our findings, it is important to acknowledge certain limitations. A larger sample size would be advantageous for future research, and the measurement of epicardial adipose tissue (EAT) using echocardiography is subject to operator variability and may not capture total EAT volume as accurately as advanced imaging modalities such as cardiac MRI or CT. Speckle-tracking echocardiography, while sensitive for detecting early myocardial dysfunction, is also operator-dependent and can be influenced by technical factors, patient anatomy, and image quality.

CONCLUSION:

Our study shows that early subclinical left ventricular dysfunction, which can be identified by decreased global longitudinal strain prior to changes in conventional measures, is closely associated with increased epicardial adipose tissue in type 2 diabetes. Higher BMI, longer duration of diabetes, and poor glycaemic control all contribute to this cardiac impairment. These results highlight the value of metabolic

control and sophisticated imaging in detecting and possibly preventing diabetic cardiomyopathy early on.

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Conflict Of Interest: Nil

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