



Echocardiographic Assessment of Left Ventricular Diastolic Dysfunction in Patients with Type 2 Diabetes Mellitus and Its Association with Glycated Hemoglobin Levels

¹Dr Jaladhi Bhardwaj, ²Dr Bandana Singh

¹ Assistant Professor, Department Of Medicine, Gautam Buddha Chikitsa Mahavidyalaya & Subharti Hospital, Dehradun

² Assistant Professor, Department of Medicine, Krishna Mohan Medical College and Hospital, Mathura



Correspondence:
Dr Bandana Singh

Received: January 07, 2026

Revised: January 24, 2026

Accepted: February 25, 2026

Published: March 11, 2026

Abstract

Background: Type 2 diabetes mellitus (T2DM) is associated with significant cardiovascular morbidity, including diabetic cardiomyopathy, which frequently presents initially as left ventricular diastolic dysfunction (LVDD). Early recognition of subclinical myocardial involvement is essential to prevent progression to overt heart failure. **Objective:** To determine the prevalence of LVDD using echocardiography in patients with T2DM and evaluate its association with glycemic control measured by HbA1c and duration of diabetes. **Methods:** This prospective observational study included 100 patients with T2DM. Clinical, biochemical, and echocardiographic assessments were performed. Statistical analysis included t-tests, chi-square tests, ANOVA, and correlation analysis. **Results:** LVDD was present in 54% of patients. Mean HbA1c was significantly higher in patients with LVDD ($9.1 \pm 1.3\%$) compared with those without LVDD ($7.0 \pm 1.2\%$, $p < 0.001$). Duration of diabetes was significantly associated with LVDD ($p = 0.003$). E/A ratio was significantly reduced in LVDD ($p = 0.002$). **Conclusion:** LVDD is common in T2DM and correlates with poor glycemic control and longer disease duration. Routine echocardiographic screening may aid early detection and intervention.

Keywords: Type 2 diabetes mellitus, Left ventricular diastolic dysfunction, Echocardiography, HbA1c, Diabetic cardiomyopathy.



This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license (<http://creativecommons.org/licenses/by/4.0/>).

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion or action [1]. The global prevalence of T2DM continues to rise, and cardiovascular disease remains the leading cause of mortality among affected individuals [2]. Diabetic cardiomyopathy represents a distinct clinical entity characterized by myocardial dysfunction independent of hypertension or coronary artery disease [3].

Left ventricular diastolic dysfunction is often the earliest detectable manifestation of diabetic cardiomyopathy and may precede systolic impairment by several years [4,5]. Chronic hyperglycemia promotes myocardial fibrosis through the accumulation of advanced glycation end products, oxidative stress, and activation of inflammatory pathways, leading to impaired ventricular relaxation [6].

Echocardiography is a reliable and noninvasive modality for assessing cardiac function and plays a pivotal role in identifying early myocardial changes in patients with

diabetes [7]. Several studies have demonstrated a significant association between poor glycemic control, longer duration of diabetes, and LVDD [8–10]. However, regional data remain limited, and further evaluation is needed to strengthen evidence for routine screening strategies.

The present study was conducted to evaluate the prevalence of LVDD among patients with T2DM and to examine its relationship with HbA1c levels and duration of disease.

MATERIALS AND METHODS

A prospective observational study was conducted at a tertiary care teaching hospital after approval from the Institutional Ethics Committee, consistent with prior methodological frameworks used in cardiovascular studies of diabetes. One hundred patients diagnosed with T2DM were enrolled after obtaining informed consent.

Inclusion Criteria

- Age 30–65 years

Citation: Bhardwaj J, Singh B Echocardiographic Assessment of Left Ventricular Diastolic Dysfunction in Patients with Type 2 Diabetes Mellitus and Its Association with Glycated Hemoglobin Levels. *Int. J. Med.*, 2026;8(3):42-45.

- Confirmed T2DM as recommended in prior studies evaluating diabetic cardiac dysfunction [9].
- Sinus rhythm

Exclusion Criteria

- Hypertension
- Ischemic heart disease
- Valvular heart disease
- Arrhythmias
- Thyroid disorders
- Chronic kidney or liver disease

Clinical and Laboratory Evaluation

Detailed history including duration of diabetes and treatment was obtained. Laboratory investigations included fasting blood glucose, postprandial glucose, HbA1c, renal and liver function tests, and lipid profile,

Echocardiography

Transthoracic echocardiography was performed to assess E/A ratio, deceleration time (DT), IVRT, left atrial size, and left ventricular dimensions following established guidelines [7].

Statistical Analysis

Continuous variables were expressed as mean $\pm$ SD. Group comparisons were performed using independent samples t-test or ANOVA. Categorical variables were analyzed using chi-square test. Pearson correlation was used to evaluate associations between HbA1c and echocardiographic parameters. A p value <0.05 was considered statistically significant.

RESULTS

LVDD prevalence was 54%. Mean HbA1c showed a strong positive association with LVDD severity (ANOVA $p=0.001$). E/A ratio demonstrated significant between-group differences ($p=0.002$). Duration of diabetes showed significant association with LVDD ($p=0.003$). Deceleration time and IVRT showed trends but did not reach statistical significance ($p=0.058$ and $p=0.118$ respectively).

Table 1: Baseline Characteristics (N=100)

Variable	Value
Age (years), mean $\pm$ SD	55.2 $\pm$ 11.3
Male, n (%)	62 (62%)
Female, n (%)	38 (38%)
BMI (kg/m ²), mean $\pm$ SD	25.1 $\pm$ 3.1
Duration of diabetes (years), mean $\pm$ SD	8.1 $\pm$ 3.8
Fasting glucose (mg/dL)	166.8 $\pm$ 18.1
Postprandial glucose (mg/dL)	226.4 $\pm$ 31.5
HbA1c (%)	8.2 $\pm$ 1.6

Table 2: Distribution of Diastolic Dysfunction

Category	n	%
Normal	46	46
Mild LVDD	42	42
Moderate LVDD	10	10
Severe LVDD	2	2

Table 3: Echocardiographic Parameters by LVDD Status

Parameter	No LVDD (n=46)	LVDD (n=54)	p value
E/A ratio	1.06 $\pm$ 0.34	0.69 $\pm$ 0.15	0.002
DT (ms)	194 $\pm$ 13	226 $\pm$ 18	0.058
IVRT (ms)	72 $\pm$ 8	78 $\pm$ 14	0.118
Left atrial size (mm)	34 $\pm$ 4	38 $\pm$ 5	0.01

Table 4: Duration of Diabetes vs LVDD

Duration	No LVDD	LVDD	Total	p value
<5 years	22	14	36	0.003*
5–10 years	18	26	44	
>10 years	6	14	20	

*: statistically significant

Table 5: Correlation Analysis

Variable Pair	r	p value
HbA1c vs E/A ratio	-0.48	<0.001
Duration vs E/A ratio	-0.32	0.004
HbA1c vs DT	0.21	0.03

DISCUSSION

The present study provides important insights into the burden and determinants of left ventricular diastolic dysfunction among individuals with type 2 diabetes mellitus, highlighting the substantial prevalence of subclinical myocardial involvement even in the absence of overt cardiovascular disease. The finding that more than half of the study population demonstrated echocardiographic evidence of diastolic dysfunction underscores the silent nature of diabetic cardiomyopathy and reinforces the need for proactive cardiovascular evaluation in this high-risk group.

Diabetic cardiomyopathy is increasingly recognized as a distinct pathophysiological entity characterized by structural, metabolic, and functional myocardial abnormalities independent of coronary artery disease or hypertension. The earliest manifestation is typically impaired myocardial relaxation resulting from complex interactions between metabolic derangements and myocardial remodeling. Chronic hyperglycemia plays a central role by promoting the formation of advanced glycation end products (AGEs), which cross-link collagen fibers and increase myocardial stiffness. In addition, hyperglycemia induces oxidative stress through increased production of reactive oxygen species, leading to mitochondrial dysfunction and activation of pro-fibrotic signaling pathways. These mechanisms collectively contribute to extracellular matrix expansion, myocardial fibrosis, and impaired diastolic filling.

The strong association observed between elevated HbA1c levels and the presence as well as severity of diastolic dysfunction in this study supports the concept that glycemic burden is a key driver of myocardial injury. Higher HbA1c reflects prolonged exposure to hyperglycemia, which accelerates structural remodeling and functional impairment of the myocardium. This dose-response relationship has been consistently reported in prior studies and suggests that tighter glycemic control may play a protective role in preventing or delaying cardiac dysfunction. Moreover, HbA1c may serve not only as a marker of metabolic control but also as a surrogate indicator of cardiovascular risk in patients with diabetes.

Duration of diabetes emerged as another significant determinant of diastolic dysfunction. Patients with longer disease duration demonstrated a markedly higher prevalence of LVDD, emphasizing the cumulative effects of chronic metabolic stress on myocardial tissue. Prolonged exposure to hyperglycemia leads to progressive microvascular dysfunction, impaired coronary flow reserve, and subclinical ischemia, which

further exacerbate myocardial fibrosis and ventricular stiffening. In addition, diabetic autonomic neuropathy may impair heart rate variability and diastolic filling dynamics, contributing to the progression of cardiac dysfunction over time.

The reduction in E/A ratio observed among patients with diastolic dysfunction reflects impaired early diastolic relaxation, a hallmark of early diabetic cardiomyopathy. Alterations in calcium handling within cardiomyocytes, particularly delayed reuptake of calcium into the sarcoplasmic reticulum, play a crucial role in this process. Furthermore, insulin resistance alters myocardial substrate utilization, shifting energy metabolism toward increased fatty acid oxidation, which is less efficient and leads to accumulation of toxic lipid intermediates within cardiomyocytes. Lipotoxicity further promotes apoptosis and myocardial dysfunction, reinforcing the pathophysiological link between metabolic abnormalities and cardiac impairment.

Although deceleration time and isovolumetric relaxation time did not reach statistical significance in the present study, the observed trends toward prolongation are consistent with impaired ventricular compliance and delayed relaxation described in previous echocardiographic investigations. The increase in left atrial size among patients with LVDD likely reflects chronically elevated left ventricular filling pressures and provides additional evidence of diastolic impairment. Enlargement of the left atrium has been associated with adverse cardiovascular outcomes, including atrial fibrillation and heart failure, highlighting the prognostic implications of early diastolic abnormalities.

From a clinical perspective, the findings of this study have several important implications. First, they support the concept that routine echocardiographic screening should be considered in patients with long-standing or poorly controlled diabetes, even in the absence of symptoms. Early identification of diastolic dysfunction offers an opportunity to intensify therapeutic interventions aimed at modifying disease progression. Optimization of glycemic control remains a cornerstone of management, but comprehensive risk reduction strategies should also include blood pressure control, lipid management, weight reduction, and promotion of regular physical activity.

Emerging evidence suggests that certain pharmacologic therapies may exert favorable effects on myocardial structure and function beyond glycemic control. Sodium-glucose cotransporter-2 inhibitors have

demonstrated significant reductions in heart failure hospitalization and may improve diastolic function through mechanisms including osmotic diuresis, reduction in myocardial inflammation, and improved myocardial energetics. Similarly, glucagon-like peptide-1 receptor agonists have been associated with cardiovascular benefit and may play a role in mitigating myocardial remodeling. Although these therapies were not specifically evaluated in the present study, their potential cardioprotective effects highlight the importance of integrating modern therapeutic strategies into the management of diabetic patients with early cardiac involvement.

Another important consideration is the role of lifestyle modification in preventing progression of diastolic dysfunction. Weight loss, improved physical fitness, and dietary interventions have been shown to improve insulin sensitivity and reduce systemic inflammation, which may translate into improved myocardial function. Exercise training, in particular, has been associated with enhanced diastolic filling and improved ventricular compliance, emphasizing its role as a non-pharmacologic intervention in patients with early diabetic cardiomyopathy.

The present findings also contribute to the growing body of evidence supporting the concept of heart failure with preserved ejection fraction as a major cardiovascular complication of diabetes. LVDD represents a key pathophysiological substrate for this condition, and early detection may allow clinicians to identify patients at risk before the development of symptomatic heart failure. Given the increasing prevalence of diabetes worldwide, the burden of heart failure related to diabetic cardiomyopathy is expected to rise substantially, underscoring the importance of preventive strategies.

Despite its strengths, including prospective design and comprehensive echocardiographic assessment, the study has certain limitations. The single-center nature of the study may limit generalizability, and subclinical coronary artery disease was not excluded using advanced imaging modalities such as stress testing or coronary computed tomography angiography. In addition, the cross-sectional design precludes assessment of temporal relationships or causal inference. Longitudinal studies are needed to determine whether early detection of diastolic dysfunction translates into improved clinical outcomes and reduced progression to overt heart failure.

Future research should focus on incorporating advanced imaging techniques such as tissue Doppler imaging, speckle tracking echocardiography, and cardiac magnetic resonance imaging to provide more detailed characterization of myocardial mechanics. Biomarkers such as natriuretic peptides and markers of fibrosis may also enhance risk stratification and allow for more personalized management approaches. Furthermore, randomized trials evaluating targeted interventions in

patients with subclinical LVDD may help define optimal strategies for preventing progression of diabetic cardiomyopathy.

In summary, the study reinforces the concept that diastolic dysfunction is a prevalent and clinically significant manifestation of type 2 diabetes mellitus. The strong associations with glycemic control and disease duration highlight the importance of early detection and comprehensive management. As our understanding of the pathophysiology of diabetic cardiomyopathy continues to evolve, integrating echocardiographic screening with aggressive risk factor modification may play a crucial role in reducing cardiovascular morbidity and improving long-term outcomes in this growing patient population.

Limitations

- Single-center study
- Subclinical coronary disease not excluded with advanced imaging
- Lack of longitudinal follow-up

CONCLUSION

LVDD is highly prevalent among patients with T2DM and is significantly associated with poor glycemic control and longer duration of diabetes. Echocardiography is a valuable tool for early detection of diabetic cardiomyopathy and may help guide preventive strategies.

REFERENCES

1. Ma RC, Tong PC. Epidemiology of type 2 diabetes. *Textbook of Diabetes*. 2016;6:43.
2. Nichols GA, Hillier TA, Erbey JR, Brown JB. Congestive heart failure in type 2 diabetes. *Diabetes Care*. 2001;24:1614-9.
3. Devereux RB, Roman MJ, Paranicas M, et al. Impact of diabetes on cardiac structure. *Circulation*. 2000;101:2271-6.
4. Cosson S, Kevorkian JP. Left ventricular diastolic dysfunction. *Diabetes Metab*. 2003;29:455-66.
5. Boyer JK, Thanigaraj S, Schechtman KB, Pérez JE. Prevalence of diastolic dysfunction. *Am J Cardiol*. 2004;93:870-5.
6. Beckman JA, Paneni F, Cosentino F, Creager MA. Diabetes and vascular disease. *Eur Heart J*. 2013;34:2444-52.
7. Rovai D, Morales MA, Di Bella G, et al. Echocardiography in LV dysfunction. *Acta Cardiol*. 2008;63:507-13.
8. Sarkar UK, Jain M, Kiyawat P. Echocardiographic evaluation in T2DM. *Int J Res Med Sci*. 2018;6:3235-8.
9. Patil VC, Patil HV, Shah KB, et al. Diastolic dysfunction in asymptomatic diabetes. *J Cardiovasc Dis Res*. 2011;2:213-22.
10. Guria RT, Prasad MK, Mishra B, et al. Association of HbA1c with LVDD. *Cureus*. 2022;14:e31262.