

Clinical Profile and Predictors of Silent Left Ventricular Systolic Dysfunction in Patients with Long-Standing Type 2 Diabetes Mellitus

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Abstract:

Background: Type 2 diabetes mellitus (T2DM) is associated with structural and functional abnormalities of the myocardium and an increased risk of heart failure. Left ventricular (LV) dysfunction may develop before the appearance of clinically overt cardiovascular disease and may remain clinically silent. Echocardiography provides a non-invasive method for detecting abnormalities in LV systolic and diastolic function.

Objective: To evaluate the clinical and echocardiographic profile and identify predictors of silent left ventricular systolic dysfunction among patients with long-standing type 2 diabetes mellitus.

Materials and Methods: A hospital-based cross-sectional observational study was conducted among 800 adults with T2DM of at least 5 years' duration attending Patna Medical College & Hospital, Patna, between July 2025 and June 2026. Participants underwent clinical assessment, biochemical investigations and transthoracic echocardiography. Silent LV systolic dysfunction was operationally defined as LV ejection fraction (LVEF) <50% in the absence of clinically overt heart failure. Clinical, biochemical and echocardiographic parameters were compared between participants with and without LV systolic dysfunction. Logistic regression analysis was performed to identify independent predictors.

Results: Among 800 participants, 242 (30.3%) had silent LV systolic dysfunction. Participants with LV systolic dysfunction were older, had a longer duration of diabetes, higher body mass index (BMI), higher systolic blood pressure and higher glycated haemoglobin (HbA1c). Albuminuria and diabetic retinopathy were also more frequent in this group. Mean LVEF was significantly lower among participants with LV systolic dysfunction than among those without dysfunction ($46.9 \pm 2.8\%$ vs $59.3 \pm 3.7\%$, $p < 0.001$). Global longitudinal strain was less negative in participants with LV systolic dysfunction ($-15.3 \pm 1.7\%$ vs $-19.0 \pm 1.6\%$, $p < 0.001$). On multivariable logistic regression, age, diabetes duration, HbA1c, systolic blood pressure, BMI and albuminuria remained independently associated with silent LV systolic dysfunction.

Conclusion: Silent LV systolic dysfunction was present in approximately one-third of patients with long-standing T2DM. Increasing age, longer diabetes duration, poorer glycaemic control, higher systolic blood pressure, greater BMI and albuminuria were independently associated with LV systolic dysfunction. Echocardiographic assessment, including myocardial deformation imaging, may help identify diabetic patients with otherwise clinically unrecognized myocardial dysfunction.

Keywords: Type 2 Diabetes Mellitus; Diabetic Cardiomyopathy; Left Ventricular Systolic Dysfunction; Echocardiography; Global Longitudinal Strain; HbA1c; Albuminuria; Predictors.

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Introduction

Type 2 diabetes mellitus is an established risk factor for cardiovascular morbidity and mortality and is associated with an increased risk of heart failure. Diabetes may adversely affect myocardial structure and function through metabolic abnormalities, oxidative stress, advanced glycation, inflammation, microvascular dysfunction and myocardial fibrosis. These changes may occur independently of clinically apparent coronary artery disease and have been described within the spectrum of diabetic

cardiomyopathy. [1]

Left ventricular dysfunction may develop before the appearance of symptomatic heart failure. Previous echocardiographic studies have demonstrated that patients with diabetes may have abnormalities of systolic or diastolic function despite the absence of overt cardiovascular disease. The SHORTWAVE study specifically demonstrated the increasing detection of asymptomatic LV dysfunction among

patients with T2DM without overt cardiac disease. [2]

The DYDA study evaluated 751 patients with T2DM and reported LV dysfunction in 59.9% of the study population. Older age and higher HbA1c were among the independent predictors of LV dysfunction, supporting the importance of cumulative metabolic exposure in the development of early cardiac abnormalities. [3]

More comprehensive echocardiographic assessment has demonstrated that diabetic patients without established cardiovascular disease may exhibit abnormalities involving LV mass, relative wall thickness, left-atrial size and myocardial deformation. The DIACAR cohort, which included patients with T2DM without overt cardiovascular disease, demonstrated abnormalities of cardiac structure and reduced global longitudinal strain in a substantial proportion of participants. [4]

Conventional LVEF remains an important measure of LV systolic function; however, myocardial deformation imaging may identify abnormalities in myocardial performance that are not fully reflected by LVEF. Standardized approaches to echocardiographic chamber quantification and speckle-tracking assessment are therefore important when investigating diabetic myocardial dysfunction. [5,6]

The assessment of diastolic function is also relevant because systolic and diastolic abnormalities may coexist in patients with diabetes. Contemporary echocardiographic recommendations provide standardized approaches for assessment using mitral inflow, tissue Doppler and left-atrial measurements. [7]

Despite increasing recognition of myocardial abnormalities in diabetes, data from Indian tertiary-care populations remain important. Identification of clinical and biochemical characteristics associated with silent LV dysfunction may help identify patients who require more detailed cardiovascular assessment.

Aim: To evaluate the clinical and echocardiographic profile of patients with long-standing T2DM and identify predictors of silent LV systolic dysfunction.

Objectives

1. To determine the prevalence of silent LV systolic dysfunction among patients with long-standing T2DM.
2. To compare clinical and biochemical characteristics between participants with and without LV systolic dysfunction.
3. To compare echocardiographic parameters between the two groups.
4. To identify independent clinical and biochemical predictors of silent LV systolic

dysfunction.

Materials and Methods

Study Design and Setting: This hospital-based cross-sectional observational study was conducted at Patna Medical College & Hospital, Patna, Bihar, India.

The study was conducted over a period of one year from July 2025 to June 2026.

A total of 800 participants fulfilling the eligibility criteria were included in the study.

Study Population: The study population consisted of adult patients with established T2DM attending the cardiology department of Patna Medical College & Hospital.

Long-standing diabetes was defined operationally as a duration of diabetes of at least 5 years.

Inclusion Criteria

Participants were included if they fulfilled all of the following criteria:

1. Age ≥ 18 years.
2. Established diagnosis of T2DM.
3. Duration of diabetes ≥ 5 years.
4. Clinically stable at the time of assessment.
5. Ability to undergo transthoracic echocardiography.
6. Willingness to participate and provision of written informed consent.

Exclusion Criteria

Participants were excluded if they had:

1. Known symptomatic heart failure.
2. Previous myocardial infarction.
3. Previously diagnosed significant coronary artery disease.
4. Significant valvular heart disease.
5. Persistent atrial fibrillation or another rhythm disturbance preventing reliable echocardiographic assessment.
6. Known primary cardiomyopathy.
7. Congenital heart disease.
8. Pericardial disease.
9. Inadequate echocardiographic image quality.
10. Any other condition considered likely to substantially alter LV systolic function independently of diabetes.

Clinical Assessment

A standardized clinical assessment was performed for all participants.

The following variables were recorded:

- Age
- Sex
- Duration of diabetes
- Body weight and height

- Body mass index
- Systolic blood pressure
- Diastolic blood pressure
- Hypertension
- Smoking status
- Diabetes treatment
- Diabetic retinopathy
- Albuminuria
- Other diabetes-related complications

BMI was calculated using the formula:

$$\text{BMI} = \text{weight (kg)} / \text{height}^2 (\text{m}^2)$$

Blood pressure was measured after adequate rest using a validated automated or calibrated sphygmomanometer.

Laboratory Investigations: The following biochemical investigations were performed:

- Fasting plasma glucose
- HbA1c
- Serum creatinine
- Estimated glomerular filtration rate (eGFR)
- Urinary albumin-to-creatinine ratio
- Serum triglycerides
- Total cholesterol
- HDL cholesterol
- LDL cholesterol

Albuminuria was classified according to standard clinical criteria.

Echocardiographic Assessment: Transthoracic echocardiography was performed using a commercially available echocardiographic system by an experienced operator.

LV chamber measurements were performed according to contemporary American Society of Echocardiography/European Association of Cardiovascular Imaging recommendations. [5]

The following echocardiographic parameters were assessed:

- LV end-diastolic volume (LVEDV)
- LV end-systolic volume (LVESV)
- LVEF
- LV mass index
- Relative wall thickness
- Left-atrial volume index (LAVI)
- Mitral E velocity
- Mitral A velocity
- E/A ratio
- Tissue Doppler e' velocity
- E/e' ratio
- Global longitudinal strain (GLS)

Measurement of LVEF: LVEF was calculated using the biplane method of disks:

$$\text{LVEF (\%)} = [(\text{LVEDV} - \text{LVESV}) / \text{LVEDV}] \times$$

100

This approach is consistent with established echocardiographic chamber-quantification recommendations. [5]

Global Longitudinal Strain

Two-dimensional speckle-tracking echocardiography was used to estimate GLS.

Interpretation and terminology were based on established EACVI/ASE recommendations for standardization of two-dimensional speckle-tracking echocardiography. [6]

Because longitudinal strain is conventionally expressed as a negative value, a less negative GLS was considered indicative of impaired longitudinal myocardial deformation.

Definition of Silent LV Systolic Dysfunction: For the present study, silent LV systolic dysfunction was defined as:

LVEF <50% in the absence of clinically overt heart failure symptoms or established symptomatic heart failure.

Statistical Analysis: Continuous variables were expressed as mean $\pm$ standard deviation (SD), while categorical variables were expressed as frequency and percentage.

Continuous variables were compared using the independent-samples t test for normally distributed variables and the Mann-Whitney U test when appropriate.

Categorical variables were compared using the chi-square test or Fisher's exact test.

Logistic regression analysis was performed to identify predictors of silent LV systolic dysfunction.

Results were expressed as odds ratios (ORs), adjusted odds ratios (aORs), 95% confidence intervals (CIs) and p values.

Variables considered clinically relevant and/or associated with the outcome in preliminary analysis were entered into the multivariable model.

A two-sided p value <0.05 was considered statistically significant.

Results

Study Population: A total of 800 participants with long-standing T2DM were included in the final analysis.

Among them, 242 participants (30.3%) met the predefined criteria for silent LV systolic dysfunction, while 558 participants (69.8%) did not have LV systolic dysfunction.

The baseline clinical and biochemical characteristics of the study population are presented in **Table 1**.

Table 1: Baseline clinical and biochemical characteristics of the study population

Variable	Overall (n=800)	No LV systolic dysfunction (n=558)	Silent LV systolic dysfunction (n=242)
Age, years	58.4 ± 8.7	56.8 ± 8.3	62.1 ± 8.1
Male sex, n (%)	456 (57.0)	319 (57.2)	137 (56.6)
Diabetes duration, years	11.2 ± 4.1	10.3 ± 3.7	13.3 ± 4.2
BMI, kg/m ²	27.1 ± 3.5	26.7 ± 3.3	28.0 ± 3.8
Systolic BP, mmHg	138.0 ± 17.0	135.9 ± 16.2	142.8 ± 17.8
HbA1c, %	8.0 ± 1.2	7.8 ± 1.1	8.5 ± 1.3
eGFR, mL/min/1.73 m ²	72.0 ± 19.0	73.6 ± 18.4	68.4 ± 19.7
Albuminuria, n (%)	224 (28.0)	126 (22.6)	98 (40.5)
Hypertension, n (%)	496 (62.0)	335 (60.0)	161 (66.5)
Retinopathy, n (%)	240 (30.0)	143 (25.6)	97 (40.1)

Participants with silent LV systolic dysfunction were older and had a longer duration of diabetes than those without LV systolic dysfunction. They also had higher BMI, systolic blood pressure and HbA1c. Albuminuria and diabetic retinopathy were more frequent among participants with LV systolic

dysfunction (Table 1).

Echocardiographic Findings: The echocardiographic characteristics according to LV systolic function are presented in Table 2.

Table 2: Echocardiographic characteristics according to LV systolic function

Echocardiographic parameter	No LV systolic dysfunction	Silent LV systolic dysfunction	p value
LVEF, %	59.3 ± 3.7	46.9 ± 2.8	<0.001
GLS, %	-19.0 ± 1.6	-15.3 ± 1.7	<0.001
LV mass index, g/m ²	105.2 ± 19.5	116.4 ± 20.1	<0.001
LVEDV, mL	91.0 ± 17.0	96.2 ± 18.1	<0.001
LVESV, mL	36.0 ± 9.0	47.0 ± 9.0	<0.001
LAVI, mL/m ²	28.2 ± 5.8	31.5 ± 6.0	<0.001
E/e'	10.5 ± 2.3	11.7 ± 2.4	<0.001

Participants with silent LV systolic dysfunction had significantly lower LVEF and less negative GLS than participants without LV systolic dysfunction (Table 2). LV mass index, LVEDV, LVESV, LAVI and E/e' were also significantly higher in the dysfunction group.

Prevalence of Silent LV Systolic Dysfunction

The prevalence of silent LV systolic dysfunction was:

$$242/800 = 30.3\%.$$

Thus, approximately one-third of the study population had evidence of LV systolic dysfunction despite the absence of clinically overt heart failure.

The distribution of participants according to LV systolic function is illustrated in Figure 1.

Figure 1. Distribution of study participants according to presence or absence of silent LV systolic dysfunction

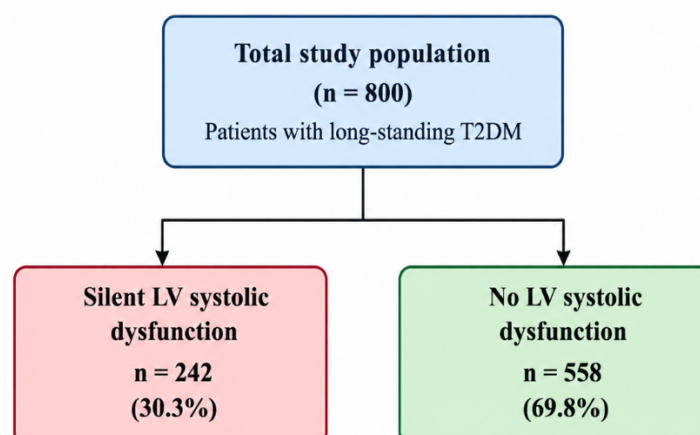


Figure 1: Distribution of study participants according to presence or absence of silent LV systolic dysfunction

Univariable Logistic Regression Analysis:
Potential clinical and biochemical predictors were initially evaluated using univariable logistic

regression.

The results are presented in **Table 3**.

Table 3: Univariable logistic regression analysis of predictors of silent LV systolic dysfunction

Predictor	OR	95% CI	p value
Age, per 10 years	1.61	1.38–1.88	<0.001
Diabetes duration, per 5 years	1.55	1.31–1.83	<0.001
HbA1c, per 1%	1.28	1.16–1.41	<0.001
SBP, per 10 mmHg	1.16	1.08–1.25	<0.001
BMI, per 5 kg/m ²	1.29	1.11–1.50	0.001
eGFR, per 10 mL/min/1.73 m ²	0.93	0.87–0.99	0.026
Albuminuria	2.05	1.45–2.91	<0.001
Hypertension	1.51	1.09–2.10	0.014

OR: odds ratio; **CI:** confidence interval; **SBP:** systolic blood pressure; **BMI:** body mass index.

As shown in **Table 3**, older age, longer diabetes duration, higher HbA1c, higher systolic blood pressure, higher BMI and albuminuria were positively associated with silent LV systolic dysfunction.

Multivariable Logistic Regression Analysis:
Variables considered clinically relevant were entered into the multivariable logistic regression model.

The adjusted results are presented in **Table 4**.

Table 4: Multivariable logistic regression analysis of independent predictors of silent LV systolic dysfunction

Predictor	Adjusted OR	95% CI	p value
Age, per 10 years	1.48	1.25–1.76	<0.001
Diabetes duration, per 5 years	1.39	1.16–1.67	<0.001
HbA1c, per 1%	1.22	1.09–1.36	<0.001
SBP, per 10 mmHg	1.12	1.03–1.21	0.006
BMI, per 5 kg/m ²	1.23	1.04–1.46	0.016
eGFR, per 10 mL/min/1.73 m ²	0.96	0.89–1.03	0.226
Albuminuria	1.79	1.23–2.61	0.002
Hypertension	1.18	0.82–1.70	0.369

OR: odds ratio; **CI:** confidence interval; **SBP:** systolic blood pressure; **BMI:** body mass index.

After multivariable adjustment, age, diabetes duration, HbA1c, systolic blood pressure, BMI and albuminuria remained independently associated with silent LV systolic dysfunction (Table 4).

The associations of eGFR and hypertension were attenuated after adjustment.

Discussion

The present study evaluated the clinical and echocardiographic profile and predictors of silent LV systolic dysfunction among 800 patients with long-standing T2DM. Silent LV systolic dysfunction was identified in 242 participants, corresponding to a prevalence of 30.3%.

The presence of LV dysfunction in patients without clinically overt heart failure is consistent with the concept that myocardial abnormalities may precede symptomatic cardiovascular disease in diabetes. The SHORTWAVE study demonstrated a substantial burden of asymptomatic LV dysfunction among patients with T2DM without overt cardiac disease, emphasizing the potential value of echocardiographic assessment in this population. [2]

The DYDA study reported LV dysfunction in 59.9% of 751 patients with T2DM and identified older age and higher HbA1c among important predictors. [3] The prevalence observed in the present study was lower. Differences in study populations, definitions of LV dysfunction, echocardiographic methodology and inclusion criteria may contribute to differences between studies.

Age was an independent predictor of silent LV systolic dysfunction in the present study. For every 10-year increase in age, the adjusted odds of LV systolic dysfunction increased by 48% (aOR 1.48, 95% CI 1.25–1.76; $p < 0.001$). This finding is consistent with previous studies demonstrating an association between increasing age and LV dysfunction in patients with diabetes. [2,3]

Duration of diabetes was also independently associated with LV systolic dysfunction. Each additional 5 years of diabetes duration was associated with a 39% increase in adjusted odds of LV dysfunction (aOR 1.39, 95% CI 1.16–1.67; $p < 0.001$). Longer exposure to hyperglycaemia and associated metabolic abnormalities may contribute to myocardial remodeling through oxidative stress, advanced glycation, altered myocardial metabolism, endothelial dysfunction and myocardial fibrosis. [1]

Poor glycaemic control was another important predictor. HbA1c remained independently associated with silent LV systolic dysfunction, with each 1% increase associated with a 22% increase in adjusted odds (aOR 1.22, 95% CI 1.09–1.36;

$p < 0.001$). This finding is directionally consistent with the DYDA study, in which HbA1c was an independent predictor of early LV dysfunction. [3]

Systolic blood pressure was also independently associated with LV dysfunction. Each 10-mmHg increase in systolic blood pressure was associated with a 12% increase in adjusted odds. Chronic pressure overload may contribute to LV remodeling, increased LV mass and progressive impairment of myocardial function.

BMI was independently associated with silent LV systolic dysfunction. Each 5-kg/m² increase in BMI was associated with a 23% increase in adjusted odds. Obesity may contribute to cardiac dysfunction through haemodynamic loading, insulin resistance, neurohormonal activation, inflammation and adverse ventricular remodeling.

Albuminuria demonstrated one of the strongest independent associations in the multivariable model. Participants with albuminuria had 1.79-fold higher adjusted odds of silent LV systolic dysfunction. Albuminuria may represent a marker of systemic endothelial and microvascular injury and may therefore identify patients with a greater burden of diabetes-related target-organ damage.

The echocardiographic findings provide additional evidence of structural and functional myocardial involvement. Participants with silent LV systolic dysfunction had significantly lower LVEF and less negative GLS than those without dysfunction. GLS was $-15.3 \pm 1.7\%$ in the dysfunction group compared with $-19.0 \pm 1.6\%$ in participants without dysfunction ($p < 0.001$).

The DIACAR cohort similarly demonstrated structural and myocardial deformation abnormalities among patients with T2DM without overt cardiovascular disease, including reduced GLS in approximately one-fifth of participants. [4] These findings support the use of myocardial deformation imaging as an adjunct to conventional assessment of LV function.

The increase in LV mass index observed in participants with LV dysfunction is also clinically relevant. Diabetes, hypertension and obesity may interact to promote LV hypertrophy and adverse remodeling. Standardized measurement of LV mass, chamber volumes and related parameters is therefore important when assessing diabetic cardiac involvement. [5]

LAVI and E/e' were also significantly higher in the LV dysfunction group. These findings suggest that systolic impairment may coexist with abnormalities of filling pressure and diastolic function. Current recommendations emphasize an integrated assessment of diastolic function using mitral inflow, tissue Doppler and left-atrial measurements. [7]

The present findings have potential clinical implications. Patients with long-standing T2DM who are older, have longer diabetes duration, poor glycaemic control, elevated systolic blood pressure, higher BMI or albuminuria may represent a subgroup with greater likelihood of silent myocardial dysfunction. However, the clinical utility and cost-effectiveness of routine echocardiographic screening of all asymptomatic patients with diabetes require further prospective evaluation.

Strengths

The major strengths of the study include:

1. Relatively large sample size of 800 participants.
2. Assessment of clinical, biochemical and cardiovascular risk factors.
3. Standardized echocardiographic assessment.
4. Inclusion of GLS as an additional measure of myocardial function.
5. Evaluation of multiple potential predictors using multivariable logistic regression.
6. Assessment of albuminuria as a marker of diabetic target-organ involvement.

Limitations

The study has several limitations.

First, the cross-sectional design prevents determination of temporal or causal relationships between diabetes-related risk factors and LV dysfunction.

Second, this was a single-centre study, which may limit the generalizability of the findings to other populations.

Third, echocardiographic measurements may be subject to interobserver and intraobserver variability.

Fourth, residual confounding may remain despite multivariable adjustment.

Finally, the operational definition of silent LV systolic dysfunction was based on LVEF <50%; patients with preserved LVEF but abnormal myocardial deformation may represent an earlier stage of myocardial dysfunction and were not included in the primary endpoint.

Conclusion

Silent LV systolic dysfunction was identified in 30.3% of patients with long-standing T2DM in the present study.

Older age, longer duration of diabetes, higher HbA1c, higher systolic blood pressure, greater BMI and albuminuria were independently associated with silent LV systolic dysfunction.

Participants with LV dysfunction also demonstrated significantly lower LVEF, less negative GLS and

adverse structural and functional echocardiographic characteristics.

These findings suggest that clinically silent myocardial dysfunction may be relatively common among patients with long-standing T2DM and that selected clinical characteristics may help identify individuals at increased risk. Echocardiographic assessment, including myocardial deformation imaging, may provide additional information beyond conventional clinical assessment.

Prospective multicentre studies are required to determine whether early identification and targeted management of silent LV dysfunction can reduce subsequent heart failure and cardiovascular events in patients with T2DM.

Conflict of Interest: The authors declare that they have no conflict of interest.

Informed Consent: Written informed consent was obtained from all participants before enrolment in the study.

Data Availability: The data supporting the findings of this study are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

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