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Original Research Article

Incidence of Left Ventricular Dysfunction in Normotensive Type 2 Diabetic Patients

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Abstract

Aim: To determine the incidence and prevalence of left ventricular dysfunction in normotensive Type 2 diabetic patients and establish the correlation between glycemic control, metabolic parameters, and cardiac function abnormalities.

Materials and Methods: A cross-sectional observational study was conducted on 300 normotensive Type 2 diabetic patients (150 males, 150 females) aged 35-70 years and 150 age-matched, gender-matched normotensive healthy controls. Comprehensive echocardiography with Doppler imaging was performed to assess left ventricular systolic and diastolic function. Blood pressure monitoring confirmed normotensive status (<140/90 mmHg). Glycemic parameters (HbA1c, fasting glucose), lipid profile, high-sensitivity C-reactive protein (hs-CRP), and anthropometric measurements were recorded. Exclusion criteria included history of hypertension, smoking, significant alcohol intake, valvular heart disease, renal insufficiency, and thyroid disorders.

Results: Left ventricular dysfunction was detected in 180 (60%) diabetic patients compared to 18 (12%) controls ($p < 0.001$). Diastolic dysfunction was present in 156 (52%) diabetics versus 12 (8%) controls. Systolic dysfunction (LVEF <50%) was identified in 54 (18%) diabetics and 6 (4%) controls. Mean LVEF was 61.8% ± 8.9% in diabetics versus 68.4% ± 7.2% in controls ($p < 0.001$). Left ventricular mass index was significantly elevated in diabetics (98.5 ± 26.3 g/m²) compared to controls (84.2 ± 19.1 g/m²) ($p = 0.002$). HbA1c showed inverse correlation with LVEF ($r = -0.456$, $p < 0.001$) and positive correlation with left ventricular mass index ($r = 0.512$, $p < 0.001$). hs-CRP levels positively correlated with diastolic dysfunction severity ($r = 0.478$, $p < 0.001$).

Conclusion: Normotensive Type 2 diabetic patients demonstrate significantly high incidence of subclinical left ventricular dysfunction despite the absence of hypertension and cardiac symptoms. Early detection through echocardiography and optimal glycemic control are essential for preventing progression to overt heart failure.

Keywords: Left ventricular dysfunction; Type 2 diabetes mellitus; Echocardiography; Diastolic dysfunction; Normotensive diabetic patients.

Introduction

Diabetes mellitus has emerged as a pandemic affecting over 500 million individuals globally, with Type 2 diabetes accounting for approximately 90-95% of all diabetic cases [1]. India has the highest burden of Type 2 diabetes, with prevalence estimated at 80-100 million individuals, significantly impacting public health infrastructure and clinical outcomes [2]. While the macro-vascular and micro-vascular complications of diabetes are well-established, the cardiac manifestations have gained considerable clinical significance in recent years. Diabetic cardiomyopathy, defined as myocardial dysfunction in the absence of coronary artery disease and hypertension, is recognized as an independent complication of diabetes mellitus [3].

This entity has emerged as one of the leading causes of morbidity and mortality in diabetic patients, contributing to approximately 50-60% of deaths among individuals with Type 2 diabetes [1].

The pathophysiology involves multiple mechanisms including chronic hyperglycemia, insulin resistance, increased oxidative stress, myocardial fibrosis, and systemic inflammation [4].

Left ventricular (LV) dysfunction represents an early manifestation of diabetic cardiomyopathy that often precedes the clinical onset of heart failure. While hypertension frequently coexists with diabetes, contributing to LV dysfunction through increased afterload, recent evidence suggests that normotensive diabetic patients also develop subclinical left ventricular dysfunction, indicating that hyperglycemia itself exerts direct deleterious effects on myocardial architecture and function

This study was designed to investigate the incidence and prevalence of left ventricular dysfunction in a cohort of normotensive Type 2 diabetic patients using comprehensive echocardiographic assessment and to establish correlations between glycemic parameters, inflammatory markers, and indices of ventricular dysfunction.

Materials and Methods

Study Design and Setting A cross-sectional observational study was conducted over a period of 18 months (January 2024 to June 2025). The study was approved by the Institutional Ethics Committee, and informed written consent was obtained from all participants.

Study Population A total of 300 consecutive normotensive Type 2 diabetic patients (150 males and 150 females) aged between 35 and 70 years were enrolled. An equal number of age-matched, gender-matched, normotensive, non-diabetic healthy controls (150 individuals) were selected from the hospital staff and community.

Inclusion Criteria

- Diagnosed Type 2 diabetes mellitus (minimum 1 year duration)
- Normotensive status (systolic blood pressure <140 mmHg and diastolic blood pressure <90 mmHg on three separate occasions)
- Age between 35 and 70 years
- Both males and females
- Written informed consent

Exclusion Criteria

- History of hypertension or antihypertensive medication use
- Smoking or significant alcohol consumption (>20 grams/day)
- History of coronary artery disease or prior myocardial infarction
- Presence of significant valvular heart disease

Clinical Assessment Detailed history including diabetes duration, current medications, family history of premature coronary artery disease, and cardiovascular symptoms was recorded. Blood pressure was recorded in triplicate using a standard sphygmomanometer after 5 minutes of rest in sitting position. Anthropometric measurements including height, weight, and waist circumference were recorded for all subjects.

Laboratory Investigations

Echocardiographic Assessment

Observation Tables

Table 1: Demographic and Anthropometric Characteristics of Study Population

Characteristics	Diabetic Patients (n=300)	Control Subjects (n=150)	P-value
Age (years)	52.3 ± 11.2	51.8 ± 10.9	0.625
Gender (M/F)	150/150	75/75	1.000
Body Mass Index (kg/m²)	27.4 ± 3.8	26.8 ± 3.5	0.084
Waist Circumference (cm)	94.2 ± 8.7	89.5 ± 7.2	<0.001
Systolic BP (mmHg)	128.4 ± 9.3	122.1 ± 8.6	<0.001
Diastolic BP (mmHg)	82.3 ± 6.8	78.9 ± 6.2	<0.001
Diabetes Duration (years)	8.7 ± 6.3	NA	NA
Heart Rate (bpm)	76.4 ± 8.2	73.2 ± 7.9	0.002

Table 2: Glycemic and Metabolic Parameters

Parameters	Diabetic Patients	Control Subjects	P-value
Fasting Glucose (mg/dL)	154.8 ± 42.3	92.1 ± 8.7	<0.001
HbA1c (%)	8.2 ± 1.8	5.3 ± 0.4	<0.001
Total Cholesterol (mg/dL)	218.5 ± 46.2	178.3 ± 35.1	<0.001
LDL Cholesterol (mg/dL)	142.3 ± 38.7	108.2 ± 28.4	<0.001
HDL Cholesterol (mg/dL)	38.2 ± 8.9	52.4 ± 9.7	<0.001
Triglycerides (mg/dL)	186.4 ± 74.3	98.2 ± 42.1	<0.001
hs-CRP (mg/L)	5.8 ± 3.2	1.2 ± 0.8	<0.001
Fasting Insulin (μU/mL)	18.4 ± 8.9	8.2 ± 3.5	<0.001

Table 3: Echocardiographic Parameters Comparing Diabetic Patients and Control Subjects

Echocardiographic Parameters	Diabetic Patients	Control Subjects	P-value
LVEF (%)	61.8 ± 8.9	68.4 ± 7.2	<0.001
LVEDD (mm)	50.2 ± 4.8	48.1 ± 3.9	<0.001
LVES D (mm)	34.3 ± 5.2	31.8 ± 4.1	<0.001
Left Ventricular Mass Index (g/m²)	98.5 ± 26.3	84.2 ± 19.1	0.002
E/A Ratio	0.82 ± 0.26	1.08 ± 0.24	<0.001
Deceleration Time (ms)	248 ± 62	195 ± 48	<0.001
E/e' Ratio	9.8 ± 2.4	6.2 ± 1.8	<0.001
Peak Systolic Strain (%)	-17.2 ± 3.4	-21.8 ± 2.9	<0.001

Table 4: Prevalence of Left Ventricular Dysfunction and Correlation with Hba1c Levels

HbA1c Range (%)	Number of Patients	LV Dysfunction n(%)	Diastolic Dysfunction n(%)	Systolic Dysfunction n(%)	Mean LVEF (%)
<6.5% (Controlled)	24	3 (12.5%)	2 (8.3%)	1 (4.2%)	67.3 ± 6.8
6.5-7.5% (Good Control)	78	28 (35.9%)	22 (28.2%)	6 (7.7%)	65.2 ± 7.9
7.6-8.5% (Moderate)	114	76 (66.7%)	68 (59.6%)	18 (15.8%)	62.1 ± 9.2
>8.5% (Poor Control)	84	73 (86.9%)	64 (76.2%)	29 (34.5%)	58.4 ± 10.1
Total	**300**	**180 (60%)**	**156 (52%)**	**54 (18%)**	**61.8 ± 8.9**

Results

The study comprised 300 normotensive Type 2 diabetic patients (mean age 52.3 ± 11.2 years, 150 males and 150 females) and 150 healthy controls (mean age 51.8 ± 10.9 years, 75 males and 75 females). The groups were well-matched for age, gender, and body mass index (p >0.05). Diabetic patients demonstrated significantly elevated fasting glucose levels (154.8 ± 42.3 mg/dL vs 92.1 ± 8.7 mg/dL, p <0.001) and HbA1c levels (8.2 ± 1.8% vs 5.3 ± 0.4%, p <0.001) compared to controls. Lipid profile analysis revealed significantly higher total cholesterol, LDL cholesterol, and triglycerides in diabetic patients, while HDL cholesterol was significantly lower (p <0.001 for all comparisons).

Left ventricular ejection fraction (LVEF) was significantly reduced in diabetic patients compared to controls (61.8 ± 8.9% vs 68.4 ± 7.2%, p <0.001). Systolic dysfunction (LVEF <50%) was identified in 54 (18%) diabetic patients compared to only 6 (4%) controls (p <0.001). Among those with systolic dysfunction, 36 (12%) had mild dysfunction (LVEF 40-49%), 14 (4.7%) had moderate dysfunction (LVEF 30-39%), and 4 (1.3%) had severe dysfunction (LVEF <30%). All controls with reduced LVEF had mild systolic dysfunction.

Diastolic dysfunction was the predominant abnormality identified in this study. Among 300 diabetic patients, 156 (52%) had evidence of impaired diastolic function compared to only 12 (8%) control subjects (p <0.001). Left ventricular mass index was significantly elevated in diabetic patients (98.5 ± 26.3 g/m² vs 84.2 ± 19.1 g/m², p = 0.002). Among those with LVH, concentric hypertrophy was identified in 84 (28%) and eccentric hypertrophy in 54 (18%) diabetic patients. A strong inverse correlation was observed between HbA1c levels and LVEF (r = -0.456, p <0.001). Patients with poor glycemic control (HbA1c >8.5%) had significantly higher prevalence of LV dysfunction (73 of 84, 86.9%) compared to those with good control (HbA1c <6.5%, 3 of 24, 12.5%).

Duration of diabetes showed positive correlation with degree of LV dysfunction. Patients with diabetes duration >10 years had significantly higher prevalence of diastolic dysfunction (68.4%) and systolic dysfunction (24.3%) compared to those with duration <5 years (34.2% and 6.8% respectively, p <0.001). However, subclinical LV dysfunction was also identified in recently diagnosed diabetic patients (<2 years), suggesting that hyperglycemia itself contributes to early myocardial dysfunction independent of disease duration.

Statistical Analysis: Data were analyzed using Statistical Package for Social Sciences version 25.0 (SPSS Inc., Chicago, USA). Continuous variables were expressed as mean ± standard deviation, and categorical variables as percentages. Student's t-test was used for comparing continuous variables between groups, while chi-square test was applied for categorical variables. Pearson's correlation coefficient was used to establish relationships between glycemic parameters and echocardiographic indices. Binary logistic regression analysis was performed to identify

independent predictors of LV dysfunction. A p-value <0.05 was considered statistically significant.

Discussion

The present study demonstrates a strikingly high prevalence of left ventricular dysfunction (60%) in normotensive Type 2 diabetic patients, with diastolic dysfunction (52%) being the predominant abnormality and systolic dysfunction (18%) occurring in a significant minority. This finding is particularly clinically relevant as it challenges the traditional notion that LV dysfunction occurs primarily in hypertensive diabetic patients. The results align with recent large-scale epidemiological studies from India and globally, which have consistently demonstrated prevalence rates of LV dysfunction ranging from 30% to 80% in diabetic populations.

The Framingham Heart Study and subsequent prospective cohort studies established that diabetic individuals have 2-4-fold higher risk of developing heart failure compared to non-diabetic individuals. However, most of these studies included cohorts with mixed hypertensive and normotensive status. The novelty of the present investigation lies in the selective enrollment of strictly normotensive diabetic patients, thereby isolating the direct effects of hyperglycemia and metabolic dysfunction on myocardial structure and function independent of the confounding effects of elevated blood pressure.

Among our study population of 300 diabetic patients, 180 (60%) demonstrated evidence of LV dysfunction. This high prevalence persists despite blood pressure remaining within normotensive range throughout the study period, suggesting that mechanisms independent of increased afterload from hypertension drive the development of diabetic cardiomyopathy. The fact that 282 (94%) of these patients were completely asymptomatic emphasizes the insidious nature of subclinical myocardial dysfunction and its potential to progress to overt heart failure if left undetected and untreated.

Our finding that diastolic dysfunction (52%) is more prevalent than systolic dysfunction (18%) in this cohort is consistent with the current understanding of the pathophysiology of diabetic cardiomyopathy. Diastolic dysfunction represents the earliest detectable functional abnormality in diabetic myocardium and may persist for years before progression to systolic dysfunction or symptomatic heart failure. Among 156 patients with diastolic dysfunction, the majority (102 patients, 65.4%) demonstrated Grade 1 diastolic dysfunction characterized by impaired relaxation with preserved ejection fraction. This pattern reflects the fundamental pathophysiologic changes occurring in diabetic myocardium: impaired diastolic relaxation due to abnormal calcium handling, increased myocardial stiffness from interstitial fibrosis, and altered mitochondrial function.

Tissue Doppler imaging parameters revealed reduced e' velocity (early diastolic mitral annular velocity) in diabetic patients, which represents a more specific marker of myocardial dysfunction than conventional Doppler parameters. The markedly elevated E/e' ratio (9.8 ± 2.4 vs 6.2 ± 1.8, p <0.001) in diabetic patients indicates increased left ventricular filling pressure, suggesting that despite normal ejection fractions, diabetic patients experience hemodynamic changes characteristic of diastolic dysfunction with elevated ventricular stiffness.

While systolic dysfunction (LVEF <50%) occurred in 18% of our diabetic cohort, this likely underestimates the true prevalence of systolic involvement when more sensitive markers are employed. The mean LVEF in our diabetic group (61.8 ± 8.9%) was significantly lower than in controls (68.4 ± 7.2%), suggesting that subclinical systolic dysfunction exists across the spectrum of diabetic patients, even those with preserved ejection fraction (LVEF ≥50%).

This finding emphasizes that conventional ejection fraction may be insufficiently sensitive for detecting early diabetic cardiomyopathy. Strain imaging, particularly global longitudinal strain assessment, has emerged as a valuable tool for identifying diabetic patients at high risk for progression to overt systolic dysfunction.

The mechanisms underlying systolic dysfunction in diabetic patients include myocardial fibrosis, alterations in energy metabolism, oxidative stress-induced damage to contractile proteins, and abnormal mitochondrial function. The progressive reduction in LVEF with increasing disease severity underscores the importance of early detection and intervention to prevent irreversible myocardial damage.

The development of LVH in normotensive diabetic patients represents a maladaptive response to multiple pathologic stimuli including chronic hyperglycemia, insulin resistance, and systemic inflammation. The pattern of LVH observed in our cohort included both concentric hypertrophy (28%) and eccentric hypertrophy (18%). Concentric hypertrophy, characterized by increased wall thickness with preserved or reduced chamber size, is associated with particularly poor diastolic properties and is a powerful predictor of heart failure development. The presence of concentric LVH in 84 (28%) of our patients indicates significant structural remodeling despite absence of hypertension.

The mechanisms of LV remodeling in diabetes involve activation of renin-angiotensin-aldosterone system (RAAS) even in normotensive individuals due to local tissue RAAS activation, increased sympathetic nervous system activity, enhanced inflammatory cascade, and altered collagen synthesis leading to myocardial fibrosis. These mechanisms explain how LVH develops independent of elevated systemic blood pressure.

A striking finding in our study was the powerful relationship between glycemic control indices and cardiac dysfunction parameters. Patients with poor glycemic control (HbA1c >8.5%) had significantly higher prevalence of LV dysfunction (86.9%) compared to those with good control (HbA1c <6.5%, 12.5%). The mean LVEF progressively deteriorated with increasing HbA1c: from 67.3% in well-controlled patients to 58.4% in poorly controlled patients.

The mechanism by which hyperglycemia damages myocardium involves multiple pathways: increased production of advanced glycation end products (AGEs), enhanced oxidative stress through uncoupling of mitochondrial electron transport chain, increased lipid peroxidation, protein kinase C activation, hexosamine pathway upregulation, and poly(ADP-ribose) polymerase activation. Additionally, hyperglycemia impairs endothelial function through reduced nitric oxide bioavailability and impaired vasodilation, leading to microvascular dysfunction and myocardial ischemia.

The importance of glycemic control is highlighted by the multivariate logistic regression analysis in our study, which identified HbA1c as the strongest independent predictor of LV dysfunction. This finding advocates for aggressive glycemic management in all diabetic patients to prevent progression of subclinical to overt cardiac dysfunction.

The finding of high prevalence of subclinical LV dysfunction in normotensive diabetic patients has significant clinical implications. First, it establishes that cardiac screening with echocardiography should be considered in all diabetic patients, regardless of blood pressure status, presence of symptoms, or presence of other cardiovascular risk factors. Guidelines should be modified to recommend baseline echocardiographic assessment in newly diagnosed Type 2 diabetic patients. Second, it provides evidence supporting the cardioprotective benefits of glucose-lowering agents beyond their glycemic effects. SGLT2 inhibitors have demonstrated marked cardiovascular benefits in multiple randomized controlled trials, with benefits occurring independent of glucose lowering and including improvement in diastolic dysfunction, reduction in LV mass, and prevention of heart failure hospitalization. GLP-1 receptor agonists have similarly shown cardioprotective effects. Metformin, the first-line agent for Type 2 diabetes, has been associated with improved LV function in some studies.

Third, it emphasizes the importance of optimal blood pressure control even in patients with normotensive diabetes, as our findings suggest that blood pressures in the upper normotensive range (128.4 mmHg systolic and 82.3 mmHg diastolic) are associated with greater LV dysfunction compared to truly normal blood pressures. Achieving systolic blood pressure <120 mmHg may be beneficial in diabetic patients.

Fourth, it justifies aggressive lipid management and weight reduction in diabetic patients, as dyslipidemia and obesity contribute to myocardial inflammation and dysfunction. Cardiovascular risk reduction through lifestyle modifications should be emphasized.

Limitations of the Study: While this study provides valuable insights, several limitations should be acknowledged. First, the cross-sectional design prevents determination of causality and temporal relationships. Prospective longitudinal studies are needed to track progression of cardiac dysfunction and correlation with clinical outcomes. Second, the study population is limited to a single center in central India, which may limit generalizability to other geographic regions and ethnic populations. Multi-center studies involving diverse populations are warranted.

Third, while echocardiography is a valuable non-invasive imaging tool, it has inherent limitations including operator dependence and inability to assess myocardial tissue composition. Cardiac magnetic resonance imaging with tissue characterization would provide additional information about myocardial fibrosis and infiltration.

Fourth, the study does not include assessment of additional biomarkers of myocardial dysfunction such as natriuretic peptides (BNP, NT-proBNP) or cardiac troponins, which might provide additional prognostic information. Fifth, the absence of long-term follow-up data limits our ability to predict which patients with subclinical LV dysfunction will progress to symptomatic heart failure.

Conclusion

The presence of significant subclinical myocardial dysfunction in asymptomatic normotensive diabetic patients has major clinical implications. Screening for left ventricular dysfunction should be incorporated into routine care of all Type 2 diabetic patients, regardless of blood pressure status. Early

detection enables initiation of cardioprotective therapies including glucose-lowering agents with demonstrated cardioprotective effects (SGLT2 inhibitors, GLP-1 agonists), ACE inhibitors, and angiotensin receptor blockers.

Future research should focus on prospective longitudinal studies tracking progression of subclinical LV dysfunction to symptomatic heart failure, identification of genetic and molecular markers predictive of rapid progression, development of risk stratification models to identify highest-risk patients for intensive intervention, and evaluation of novel pharmacologic agents targeting specific pathogenic mechanisms of diabetic cardiomyopathy.

The findings of this study emphasize that diabetic cardiomyopathy represents a continuum of cardiac dysfunction that progresses from subclinical diastolic abnormalities through systolic dysfunction to symptomatic heart failure. Early detection and aggressive management of metabolic risk factors represent critical opportunities for prevention and modification of this serious cardiovascular complication of diabetes mellitus.

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