Original Research

CORRELATION OF HBA1C WITH LEFT VENTRICULAR DIASTOLIC DYSFUNCTION IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a major global health concern with rising prevalence, particularly in developing countries like India (1). Chronic hyperglycemia contributes significantly to cardiovascular complications, including left ventricular diastolic dysfunction (LVDD), an early and often clinically silent marker of diabetic cardiomyopathy (2,3). Because LVDD frequently precedes overt systolic dysfunction and heart failure with preserved ejection fraction (HFpEF), identifying an accessible biochemical marker that flags patients at risk is of considerable clinical value. Glycated hemoglobin (HbA1c) reflects long-term glycemic control and may serve as such a predictor of cardiac dysfunction (1).

Objective: To evaluate the correlation between HbA1c levels and left ventricular diastolic dysfunction in patients with T2DM, and to examine how age and duration of diabetes modify this relationship.

Methods: A cross-sectional, hospital-based observational study was conducted on 100 patients with T2DM at LLRM Medical College, Meerut. Patients aged 30–60 years fulfilling WHO criteria for diabetes were included. Clinical evaluation, laboratory investigations including HbA1c, and two-dimensional transthoracic echocardiographic assessment of LVDD were performed. Patients with known cardiovascular or pulmonary disease were excluded to isolate the effect of glycemic status on diastolic function. Statistical analysis was used to assess associations between HbA1c, age, duration of diabetes, and LVDD.

Results: LVDD was present in 46% of patients, with Grade I (mild) dysfunction predominating, consistent with early, largely asymptomatic disease. Mean HbA1c was significantly higher in patients with LVDD (7.7 ± 0.90%) compared to those without LVDD (7.13 ± 0.64%). Patients with LVDD were older (52.47 ± 5.59 years vs 44.98 ± 6.15 years) and had a longer duration of diabetes (8.35 years vs 3.4 years) than those without LVDD.

Conclusion: Elevated HbA1c, increasing age, and longer duration of diabetes are significantly associated with LVDD. Routine echocardiographic screening in patients with poor glycemic control, particularly those with long-standing disease, may allow early detection, and optimal glycemic control may help prevent progression to overt heart failure.

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia and is a major global health concern, especially in India, which carries one of the largest burdens of T2DM worldwide (1).

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Cardiovascular complications remain the leading cause of morbidity and mortality among diabetic patients, accounting for a substantial proportion of diabetes-related hospitalizations and deaths (2).

Diabetic cardiomyopathy is a well-recognized clinical entity that develops independently of hypertension and coronary artery disease, driven instead by the direct metabolic consequences of chronic hyperglycemia on the myocardium. Left ventricular diastolic dysfunction (LVDD) represents the earliest functional manifestation of this condition. Because impaired ventricular relaxation and reduced compliance can exist for years without overt symptoms, LVDD is frequently under-recognized in routine diabetes care (3). If left undetected and untreated, it may progress through

worsening grades of dysfunction to heart failure with preserved ejection fraction (HFpEF), a syndrome that carries significant morbidity and is often more difficult to manage once established than earlier, subclinical stages (4).

The pathophysiology linking hyperglycemia to LVDD is multifactorial. Chronic hyperglycemia promotes non-enzymatic glycation of myocardial structural proteins and accumulation of advanced glycation end-products, which cross-link collagen and stiffen the ventricular wall. In parallel, hyperglycemia drives oxidative stress, mitochondrial dysfunction, and low-grade inflammation, while microvascular dysfunction reduces capillary density and impairs myocardial perfusion reserve. Together these processes result in interstitial fibrosis and impaired active relaxation of the left ventricle, culminating in the functional picture of diastolic dysfunction seen on echocardiography (5).

Glycated hemoglobin (HbA1c) is a reliable, widely available marker of glycemic control over the preceding two to three months and has been consistently associated with both microvascular complications (retinopathy, nephropathy, neuropathy) and macrovascular complications (coronary artery disease, stroke, peripheral arterial disease) of diabetes (1,6). Because HbA1c is already measured routinely in diabetes follow-up, establishing a robust correlation with LVDD would give clinicians a simple, low-cost way to flag patients who may benefit from earlier cardiac screening, without requiring additional biochemical testing.

Despite growing recognition of diabetic cardiomyopathy, data specifically correlating HbA1c with echocardiographically defined LVDD in Indian tertiary-care populations remain limited. This study was therefore designed to evaluate the correlation between HbA1c levels and LVDD in patients with T2DM, and to examine the contribution of age and duration of diabetes to this relationship, with the broader aim of supporting evidence-based screening recommendations in routine diabetes care.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based, cross-sectional, observational study conducted in the Department of Medicine at a tertiary care teaching hospital (Lala Lajpat Rai Memorial Medical College, Meerut). A total of 100 patients with type 2 diabetes mellitus attending the medicine outpatient and inpatient services were enrolled consecutively over the study period.

Inclusion Criteria

- Age between 30 and 60 years
- Patients fulfilling WHO criteria for diabetes mellitus
- Patients providing written informed consent

Exclusion Criteria

- Known hypertension, ischemic heart disease, or cardiomyopathy
- Chronic respiratory disease
- Congenital heart disease
- Any severe comorbid condition affecting participation

These exclusions were applied specifically to minimize confounding cardiac and pulmonary causes of diastolic dysfunction, so that the association observed could be attributed more confidently to the metabolic effects of diabetes itself.

Methodology

A detailed clinical history and physical examination were performed for every participant, with specific attention to symptoms of diabetes and its complications. Laboratory investigations included HbA1c (measured by high-performance liquid chromatography or an equivalent standardized assay), renal function tests, and a fasting lipid profile. A resting 12-lead electrocardiogram and chest radiograph were obtained as baseline investigations to help exclude overt structural or ischemic heart disease.

All patients underwent two-dimensional transthoracic echocardiography with Doppler assessment of left ventricular diastolic function, performed by a trained operator using standard views. Parameters assessed included the mitral inflow early-to-late diastolic velocity ratio (E/A ratio), deceleration time, isovolumic relaxation time (IVRT), and tissue Doppler-derived e′ velocity with the E/e′ ratio, together with left atrial volume where applicable. Based on these parameters, LVDD was graded according to standard echocardiographic criteria as Grade I (impaired relaxation), Grade II (pseudonormal filling), or Grade III (restrictive filling pattern).

Statistical Analysis

Continuous variables (HbA1c, age, duration of diabetes) are expressed as mean ± standard deviation, and were compared between patients with and without LVDD. Categorical variables, including the presence and grade of LVDD, are expressed as frequencies and percentages. Associations between HbA1c, age, and duration of diabetes with the presence of LVDD were assessed using appropriate statistical tests for continuous and categorical data, with a p-value of <0.05 considered statistically significant.

RESULTS

Of the 100 patients with T2DM evaluated, 46% were found to have echocardiographic evidence of LVDD.

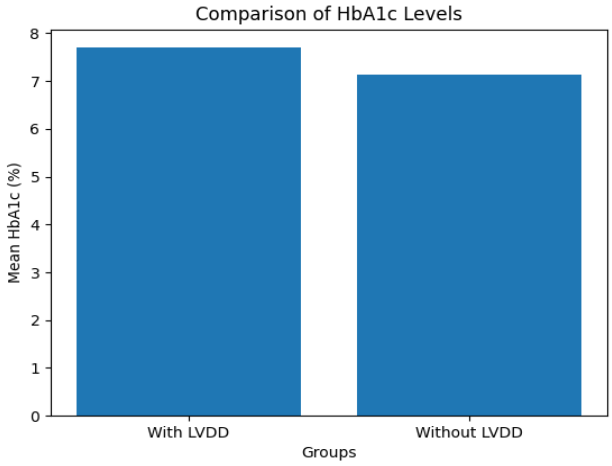

Figure 1: Comparison of mean HbA1c levels in patients with and without LVDD.

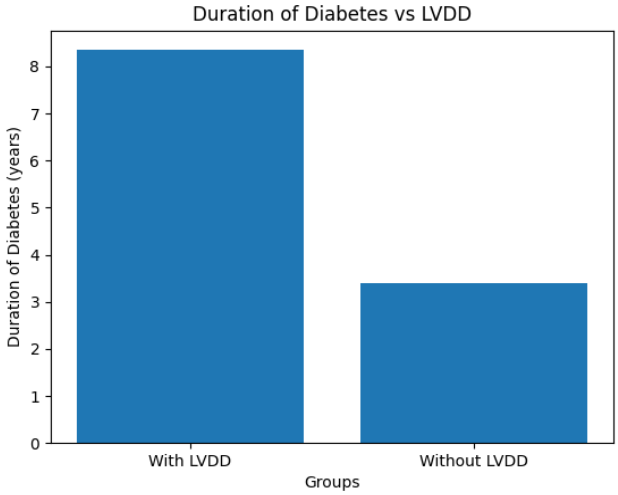

Figure 2: Duration of diabetes in patients with and without LVDD.

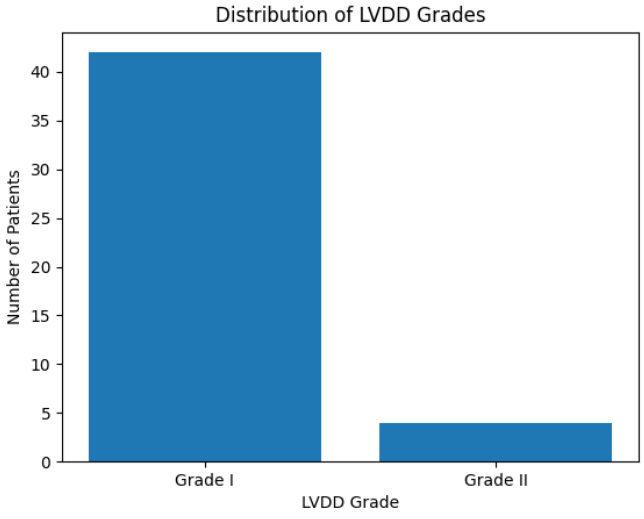

Figure 3: Distribution of LVDD grades among the study population.

Among patients with LVDD, the majority had Grade I diastolic dysfunction (impaired relaxation pattern), consistent with early-stage, largely asymptomatic disease rather than advanced restrictive physiology; this pattern is in keeping with the cross-sectional, outpatient-weighted nature of the cohort, in which patients with overt heart failure had already been excluded.

The mean HbA1c level in patients with LVDD was significantly higher (7.7 ± 0.90%) compared to those without LVDD (7.13 ± 0.64%), indicating that even a modest difference in average glycemic control over the preceding months was associated with detectable differences in diastolic function.

Patients with LVDD were also older (52.47 ± 5.59 years vs 44.98 ± 6.15 years) and had a substantially longer duration of diabetes (8.35 years vs 3.4 years) than those without LVDD. The magnitude of this difference in disease duration — more than double — suggests that cumulative glycemic exposure over time, rather than glycemic control at a single point alone, is an important driver of diastolic impairment.

Taken together, these findings indicate that poor glycemic control, increasing age, and longer disease duration are significant contributors to the development of LVDD (6,7).

DISCUSSION

The present study demonstrates a significant association between elevated HbA1c levels and LVDD in patients with T2DM, adding to a growing body of evidence that routinely measured glycemic markers can serve as practical, low-cost flags for subclinical cardiac involvement in diabetes.

Chronic hyperglycemia leads to structural and functional myocardial changes, including interstitial fibrosis and impaired active relaxation, contributing to diastolic dysfunction (5,8). Mechanistically, sustained elevation of blood glucose promotes the formation of advanced glycation end-products that stiffen myocardial collagen, activates the renin–angiotensin–aldosterone system and pro-fibrotic signaling pathways, and impairs coronary microvascular reserve through endothelial dysfunction and capillary rarefaction. These changes reduce ventricular compliance and slow early diastolic filling well before any decline in ejection fraction becomes apparent, which explains why LVDD can remain clinically silent for years. Higher HbA1c levels, reflecting poorer glycemic control, are associated with an increased risk of diabetic cardiomyopathy and progression to heart failure (6,9), and the findings of this study are consistent with this previously described relationship.

Age and duration of diabetes were also identified as important risk factors in this cohort. Prolonged exposure to hyperglycemia results in cumulative myocardial damage, increasing the likelihood of cardiac dysfunction over time (3,7); the markedly longer disease duration observed among patients with LVDD in this study supports the concept of a cumulative, dose-dependent effect of glycemic exposure on the myocardium, distinct from age-related stiffening alone. Since age itself is an independent contributor to diastolic stiffening, disentangling the relative contributions of chronological aging versus diabetes duration remains an important area for further study, ideally using multivariable analysis in a larger cohort.

From a clinical standpoint, these findings support incorporating echocardiographic screening for diastolic dysfunction into the routine follow-up of patients with T2DM who have suboptimal glycemic control or long-standing disease, even in the absence of cardiac symptoms. Early detection using echocardiography, combined with strict glycemic control, may help prevent progression to overt heart failure (2,10) and allow timely initiation of lifestyle and pharmacological measures known to benefit diastolic function and glycemic control together.

Strengths and Limitations

This study benefits from a well-defined, homogeneous cohort in which major confounding cardiac and pulmonary causes of diastolic dysfunction were carefully excluded, strengthening the inference that the association observed reflects diabetes-related myocardial changes rather than coexisting cardiopulmonary disease. However, several limitations should be considered. The cross-sectional design precludes establishing a causal or temporal relationship between HbA1c and LVDD, and the single-center setting with a modest sample size of 100 patients may limit generalizability to other populations. Additional biomarkers of cardiac strain, such as natriuretic peptides, were not assessed, and echocardiographic grading, while performed to standard criteria, remains subject to some degree of inter-observer variability. Longitudinal follow-up was also not performed, so the rate and predictors of progression from LVDD to overt HFpEF in this population could not be evaluated.

Future Directions

Larger, multicentric, prospective cohort studies with longitudinal echocardiographic and biomarker follow-up are needed to confirm these findings, clarify the independent contributions of HbA1c, age, and diabetes duration using multivariable modelling, and determine whether intensifying glycemic control after detection of early LVDD can slow or reverse progression toward heart failure.

CONCLUSION

This study highlights a strong correlation between HbA1c levels and left ventricular diastolic dysfunction in patients with type 2 diabetes mellitus. Elevated HbA1c, increasing age, and longer

duration of diabetes are important risk factors for LVDD. Routine screening using echocardiography and strict glycemic control should be emphasized in the management of diabetic patients, particularly those with long-standing or poorly controlled disease, to prevent cardiovascular complications. Early diagnosis and timely intervention can significantly improve patient outcomes.

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