Section: General Medicine

Original Research Article

TO STUDY THE CORRELATION BETWEEN HbA1C AND LEFT VENTRICULAR DIASTOLIC DYSFUNCTION IN NEWLY DIAGNOSED TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is associated with various cardiovascular complications, among which left ventricular diastolic dysfunction (LVDD) is an early and often asymptomatic manifestation. Early detection of LVDD may help in preventing progression to overt heart failure. Glycemic control, reflected by HbA1C levels, is believed to play a significant role in the development of diabetic cardiomyopathy. The objective is to determine the incidence of left ventricular diastolic dysfunction in newly diagnosed type 2 diabetes mellitus patients and to evaluate the correlation between LVDD and HbA1C levels.

Materials and Methods: This hospital-based prospective observational study was conducted at BARC Hospital, Mumbai, among patients attending dispensaries, medical outpatient departments, and inpatient wards. A total of 110 newly diagnosed T2DM patients were included. Demographic data, anthropometric measurements, biochemical parameters, and echocardiographic findings were recorded. LVDD was assessed using standard echocardiographic criteria. Statistical analysis was performed to evaluate associations between LVDD and clinical as well as biochemical parameters.

Results: The mean age of participants was 56.9 ± 12.6 years, with a male predominance (57.3%). The mean BMI was 25.6 ± 2.6 kg/m². LVDD was observed in 53 patients (48.2%). No statistically significant association was found between LVDD and age or gender. However, LVDD showed a significant association with higher fasting blood sugar, postprandial blood sugar, HbA1C levels, microalbuminuria, total cholesterol, and triglycerides. The diagnostic accuracy of HbA1C for predicting LVDD was 92.2%, which was statistically significant.

Conclusion: Nearly half of newly diagnosed type 2 diabetes patients had LVDD. Poor glycemic control, as indicated by elevated HbA1C, was strongly associated with LVDD, highlighting the importance of early cardiovascular evaluation and strict glycemic control in newly diagnosed T2DM patients.

Keywords: Diabetes mellitus (DM), T2DM, LVDD.

INTRODUCTION

Diabetes Mellitus is a metabolic disorder characterized by chronic hyperglycemia and disruptions in carbohydrate, fat, and protein metabolism caused by abnormal insulin secretion and utilization. Micro and macroangiopathy are caused by genetic, environmental, and HLA variables that are modified. Diabetes mellitus (DM) is becoming more common in both developed and developing countries, and it is currently considered an epidemic. On the other hand, diabetes early and late consequences have become a global problem. India is a key player in the global diabetes epidemic, with the world's second-largest diabetes population (69 million as of 2015). If current trends continue, India will have 123.5 million diabetics by 2040.[1] After China, India has the world's second-largest diabetes population (69.2 million) (109.7 million). Furthermore, in India, approximately 52 percent of adults with diabetes are undiagnosed. Diabetes prevalence in urban India, particularly in large

metropolitan centers, has risen from 2% in the 1970s to over 20% now, with rural areas catching up quickly.[1,2]

Diabetes is a metabolic disease that affects the heart, eyes, kidneys, skin, and peripheral nerves, among other organs. Because of decreased physical activity and increased fast food consumption, the prevalence of type 2 diabetes is increasing at a quicker rate than type I diabetes. Rubler described the first case of diabetic cardiomyopathy in 1972. It's characterized as the onset of congestive heart failure symptoms in a diabetic patient who doesn't have hypertension or any structural or congenital heart disease.

Fasting plasma glucose of 126 mg/dl or higher, random plasma glucose of 200 mg/dl or higher, indications of hyperglycemia or hyperglycemic crises, and an HbA1C level of 6.5 percent or more are all used to diagnosis type 2 diabetes. Hyperglycemia causes the formation of advanced glycation end products and free radicals in people with poorly managed diabetes. Both can produce increased collagen deposition in the myocardium, which can lead to myocardial fibrosis and affect the heart's contractility and relaxation. As a result, poor glycemic control has been linked to an increased risk of left ventricular hypertrophy and diastolic dysfunction (LVDD). Diabetic cardiomyopathy is becoming more common as the frequency of diabetes mellitus rises. Myocardial fibrosis, dysfunctional remodeling, and related diastolic dysfunction are the first signs of diabetic cardiomyopathy, followed by systolic dysfunction, and finally clinical heart failure.[3-5]

Many researchers believe that the first sign of cardiac remodeling in DM is left ventricular diastolic dysfunction (LVDD).6 DM and HF have a bidirectional effect on each other in terms of cause and effect. T2DM is seen in 19% of patients with heart failure, and the presence of T2DM increases the risk of HF by 2- to 8-fold. As a result, detecting LVDD early in type 2 diabetes patients may have clinical, prognostic, and economic ramifications by avoiding or delaying the onset of overt HF. The goal of this study is to see if HbA1C levels may detect left ventricular diastolic dysfunction in type-2 diabetes patients, because poor glycemic control has been linked to LVDD.[6,7]

Hemoglobin A1c (HbA1C) is a serological measure that has been recommended for periodic glycemic control. Independent of other cardiovascular (CV) factors, a rise of 1% in HbA1C is associated with an 8% higher risk of HF. In addition, HF reduces quality of life and alters the therapeutic impact of hypoglycemic medicines. The measurement of glycosylated Hb provides the foundation for monitoring glycemic management throughout time. By a constant slow non-enzymatic process, glucose is irreversibly bonded to hemoglobin, resulting in glycosylated hemoglobin. There is an increase in non-enzymatic glycosylation of hemoglobin when glucose levels in the plasma are regularly raised. HbA1C levels in the blood are linked to mean blood glucose levels. About 3-6 percent of Hb in a healthy person is glycosylated. The average blood glucose level over the previous three months is reflected in the HbA1C level.[8]

Microalbuminuria is defined as unusually high albumin excretion in the range of 30-299 mg/g. It's a sign that your endothelium system isn't working properly. Microalbuminuria is thought to be the result of renal and systemic trans vascular albumin leakage caused by low heparan sulfate levels in vessel walls. The leakiness of collagen, cholesterol, and advanced glycated end products has been described in the myocardium of human hearts as a result of this widespread increase in vascular permeability. Increased end-diastolic myocardial stiffness can result from these tissue changes. Cardiomyopathy may be more common as a result of endothelial dysfunction in the myocardium. Microalbuminuria can be extrapolated as a sign of endothelial dysfunction in the heart because it is a marker of endothelial dysfunction in the glomerulus, which is an arteriole. As a result, early detection and management of myocardial dysfunction in the diabetic population before the onset of overt HF is strongly advised. The incidence of LV diastolic dysfunction and the relationship between HbA1C and LVDD in newly diagnosed T2DM patients were studied in this study.

MATERIALS AND METHODS

The Study was conducted at the in patients attending medical opd and admitted to the wards in BARC Hospital, Mumbai.

Study Design: It was a hospital-based, prospective observational study.

Study Period: 2 years in the academic course of DNB

Sample Size Calculation:

Sample size n =z2×p(1-p)/e2 Where n is sample size Z is confidence interval taken as 95% so value of Z is 1.96 p- Estimated prevalence as obtained from previous studies. e- maximum tolerable error for prevalence taken as +/-0.05 Prevalence of DM is 8.9.[8]

Substituting values in above formula

N= 1.96×1.96×0.089(1-0.089)/0.055×0.055

N= 3.84×0.089×0.911/0.0025=0.311/0.0030=103.66

% of attrition:5% Total sample size:110

Study Population: patients attending dispensaries, medical OPD and admitted to the wards in BARC Hospital

Inclusion Criteria

Newly diagnosed Type 2 diabetes mellitus patients (HbA1C>6.5%), absence of any cardiovascular symptoms, absence of any abnormality in ECG

Exclusion Criteria

Old diagnosed diabetic patients on drugs, presence of any cardiovascular disease clinically, overt proteinuria, thyroid dysfunction and hypertensive patients

Sampling Technique: All consecutive patients who fulfilled the inclusion and exclusion criteria were taken up to complete the sample size in the stipulated time period.

Methodology: Informed consent was obtained from the subjects. Detailed medical history was collected from each patient. Patients were subjected for clinical examination followed by relevant investigations.

The patients undergo all basic investigations and Doppler Echo is done in each patient and 3-4 cardiac cycles was analysed to get best phase for better outcome of results.

In Doppler Echo study following values are being evaluated.

- E-peak velocity of early mitral flow (N :50-90 cm / sec)
- peak velocity of late mitral flow (N: 30-70 cm / sec)
- E/A ratio(N:1-2)
- Left atrial size (N:3–4cms)5.EF(N:>60%)

Reduction in peak velocity of early mitral flow(E) velocity increase over peak velocity of late mitral flow(A) velocity with E/A ratio of <1 and increase in left atrial (LA) size with preserved ejection fraction (EF) were considered as the evidence of left ventricular diastolic dysfunction. Patients were sent for 2-D ECHO and ECG for every patient was done and according to different criteria of ECG to diagnose LVH were noted.

Echocardiographic Grading of Diastolic Dysfunction:

Grade I: Abnormal relaxation pattern, Reversal of E/A ratio, IVRT>100ms, DT>240 ms

Grade II: Pseudonormal filling pattern, elevated atrial filling pressure, E/A ratio return to range of 0.8 to 1.5, IVRT <90ms, DT < 160 ms

Grade III: Reversible restrictive pattern, reversal of diastolic abnormalities on Valsalva manoeuvre, E/A ratio >2, IVRT <80 ms, DT<160 ms,

Grade IV: Fixed restrictive pattern, Most severe form of diastolic dysfunction., No reversibility of diastolic abnormalities on Valsalva manoeuvre, E/A ratio >2.

The questionnaires were initially checked for completeness, and data was cleaned for errors and missing values. The corrected data was then entered into Microsoft Excel after preparing a Master-chart. After data entry of every ten questionnaires, one random form was picked and data entry was re-checked. An independent person verified data entry of two randomly chosen forms after entry of every 10th questionnaire. Data analysis was done using licensed SPSS software version 21.0 (Chicago, Illinois). Univariate analyses were done initially and the results were presented with the help of tables, text, bar-diagrams and pie-charts. Descriptive statistics were used to calculate frequencies of categorical variables, and measures of central tendencies and dispersion were used to describe continuous variables. Bi-variate analyses were done using the Chi square test/Fisher's Exact test for qualitative variables. For quantitative variable independent t test was used. Accuracy of HbA1C was calculated by plotting ROC curve. P value <0.05 was considered as statistically significant.

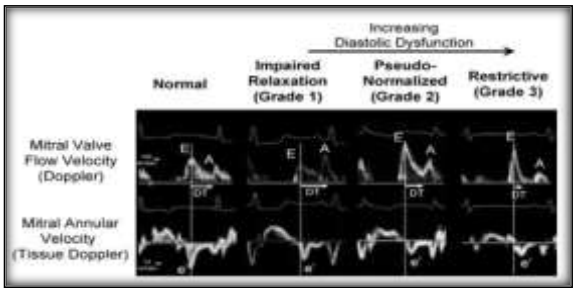

Figure 1: The stages of diastolic dysfunction recognized by changes in LV filling dynamics.

Approval from Institutional Ethical Committee of the hospital was taken before the start of the study. Written and informed consent taken from the participants before proceeding the study. Each eligible subject was explained about the purpose of the study by the investigator and an informed consent was obtained, prior to inclusion. They were assured of complete confidentiality of information, and the option of withdrawing from the study at any point of time. The study did not involve any method that puts the subjects, family members or the investigator at risk.

RESULTS

In the present study, mean age of study participants was 56.9 years with SD of 12.6. mean BMI of study participants was 25.6±2.6 kg/m2. Mean Hb and HCT of study participants was 14.0±1.3 gm/dl and 42.7% respectively.

Table 1: Distribution of participants according to age group

Age group	Frequency	Percent
18-40 years	10	9.1
41-60 years	59	53.6
>60 years	41	37.3
Total	110	100.0
Gender		
Female	47	42.7
Male	63	57.3

In our study, out of 110 patients, maximum 59 were belongs to age 41-60 years followed by 41 participants in >60 years of age. Out of 110 participants, 63 were male and 47 were female.

Table 2: Distribution of participants according to microalbuminuria and urine sugar

Microalbuminuria	Frequency	Percent
Absent	78	70.9
Present	32	29.1
Total	110	100.0
Urine sugar		
1+	31	28.2
2+	12	10.9
3+	7	6.4
4+	3	2.7
Absent	57	51.8

In our study, out of 110 participants, microalbuminuria was present in 32 patients. Out of 110 participants, urine sugar was present in 53 patients.

Table 3: Distribution of basic investigations among study participants

	FBS (mg/dl)	PPBS (mg/dl)	HbA1C	S. Creatinine	S. Uric acid
Mean	174.59	221.22	9.246	1.0422	5.4635
Median	151.50	202.00	8.600	1.0200	5.6850
Std. Deviation	59.834	66.876	2.1710	.20180	1.21287
Minimum	113	137	6.9	.60	2.90
Maximum	340	375	15.7	1.45	7.40

In our study, mean FBS, PPBS and HbA1C of study participants was 174.6±59.8 mg/dl, 221.2± 66.9 mg/dl and 9.2±2.2 % respectively. In our study, mean Serum creatinine and serum uric acid was 1.0±0.20 mg/dl and 5.5±1.2 mg/dl respectively.

Table 4: Distribution of lipid profile among study participants

	HDL	LDL	TC	TG
Mean	42.55	120.264	197.082	136.136
Median	42.00	122.000	198.000	134.500
Std. Deviation	10.600	33.2339	26.3791	39.0075
Minimum	19	11.0	99.0	65.0
Maximum	82	197.0	246.0	264.0

In our study, mean HDL, LDL, TC and TG was 42.55±10.6 mg/dl, 120.3± 33.2 mg/dl, 197.1±27.4 mg/dl and 136.1±39.0mg/dl respectively.

Table 5: Distribution of thyroid function tests among study participants

	T3	T4	TSH
Mean	.930	8.635	2.429
Median	.910	8.500	2.060
Std. Deviation	.2257	2.2539	1.4162
Minimum	.6	3.4	.7
Maximum	1.4	13.8	5.4

In our study, mean T3, T4 and TSH was 0.93±0.22 mIU/ml, 8.6± 2.2 mIU/ml and 2.4±1.4 mIU/ml respectively.

Table 6: Distribution of participants according to LVDD

LVDD	Frequency	Percent
GRADE 1 DD	42	38.2
GRADE 2 DD	11	10.0
NO DD	57	51.8
Total	110	100.0

In our study, out of 110 participants, LVDD were found in 53 (48.2%) participants.

Table 7: Association of age with LVDD among study participants

Age	LVDD absent		LVDD present		p value
	Count	%	Count	%	
18-40 years	8	14.0%	2	3.8%	0.158
41-60 years	30	52.6%	29	54.7%	
>60 years	19	33.3%	22	41.5%	
Total	57	100.0%	53	100.0%	
Female	24	42.1%	23	43.4%	0.891
Male	33	57.9%	30	56.6%	
Total	57	100.0%	53	100.0%	

In our study, no statistically significant association was found between age, gender and LVDD.

Table 8: Association of parameters with LVDD among study participants

Microalbuminuria	LVDD absent		LVDD present		p value
	Count	%	Count	%	
Absent	50	87.7%	28	52.8%	0.0001
Present	7	12.3%	25	47.2%	
Total	57	100.0%	53	100.0%	
HB	13.90	1.291	14.19	1.362	0.244
HCT	42.295	3.6776	43.187	4.1456	0.234
FBS	140.30	34.451	211.47	59.590	0.0001
PPBS	181.77	40.833	263.64	63.554	0.0001
HbA1C	7.814	0.928	10.787	2.070	0.0001
HDL	41.98	10.530	43.17	10.741	0.0560
LDL	117.965	33.0422	122.736	33.5766	0.454
TC	191.842	28.9684	202.717	22.1909	0.030
TG	124.842	33.7024	148.283	40.9396	0.001
S. Creatinine	1.0216	.20711	1.0643	.19545	0.269
S. Uric acid	5.6104	1.24930	5.3057	1.16349	0.189
T3	.899	.2418	.964	.2040	0.132
T4	8.718	2.2180	8.547	2.3098	0.694
TSH	2.298	1.3158	2.570	1.5168	0.317

In our study, a statistically significant association was found between microalbuminuria and LVDD. In our study, a statistically significant high FBS, PPBS, HbA1C, TC and TG was found among the patients with LVDD compare to patients without LVDD.

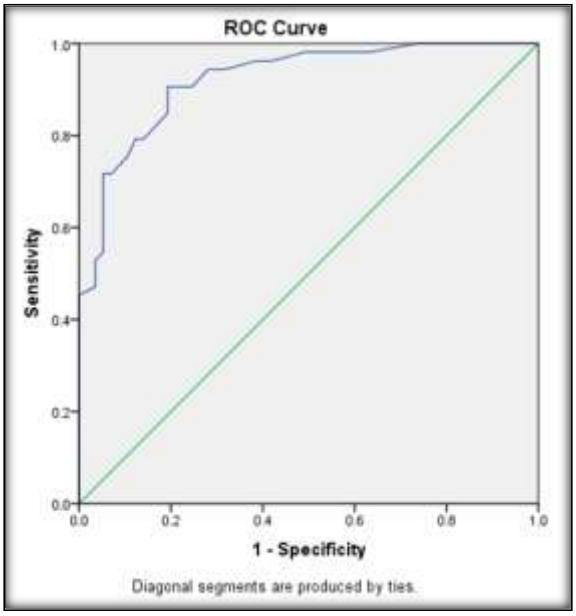

Figure 2: ROC curve HbA1C to diagnose LVDD

In our study, accuracy of HbA1C to diagnose LVDD was found to be 92.2%. it was found to be statistically significant.

DISCUSSION

Diabetes mellitus (DM), especially type 2, is a major risk factor for cardiovascular morbidity and mortality. Even in the absence of coronary artery disease (CAD), hypertension, or known structural heart disease, diabetic patients frequently develop heart failure (HF), particularly diastolic heart failure with preserved ejection fraction (HFpEF). In this study, 48.2% of newly diagnosed type 2 DM patients demonstrated left ventricular diastolic dysfunction (LVDD), despite normal LV systolic function. This aligns with accumulating evidence that diabetes contributes to myocardial dysfunction independent of traditional risk factors. The concept of diabetic cardiomyopathy — first described by Rubler et al. and now widely validated — emphasizes that diabetes causes metabolic, microvascular, and interstitial changes in the myocardium that precede overt HF (Seferović & Paulus, 2019; Håkanson et al., 2022).[9,10]

Our finding of nearly half of diabetic patients exhibiting LVDD is consistent with recent literature as Rusak et al,[11] (2021) reported that asymptomatic diabetics have a high prevalence of diastolic dysfunction, even with preserved ejection fraction, suggesting early myocardial involvement. These results reinforce the notion that diastolic impairment is an early marker in diabetic cardiomyopathy before systolic deficits emerge (Shah et al., 2020).[12]

The present study demonstrated a significant association between HbA1c levels and LVDD. This relationship has been confirmed in multiple recent trials. Zhang et al,[13] (2021) showed a strong correlation between elevated HbA1c and impaired diastolic parameters, independent of age or hypertension. Antoniades et al,[14] (2020) found that poor glycemic control predicted increased LV stiffness and reduced early diastolic filling. Furthermore, Perumal et al,[15] (2022) and Patil et al,[16] (2021) reported similar findings, where higher HbA1c was significantly associated with LVDD in asymptomatic diabetics. Taken together, this implies that long-term hyperglycemia directly contributes to myocardial stiffness, fibrotic remodeling, and microvascular dysfunction — all core elements of diastolic dysfunction.

In this study, microalbuminuria, elevated FBS, PPBS, TC, and TG levels correlated significantly with LVDD, reflecting the interconnected nature of diabetic end-organ damage. Microalbuminuria is a validated marker of generalized vascular dysfunction and has been linked to adverse cardiac remodeling in diabetes (Lee et al., 2022).[17]

While this study focused on diastolic abnormalities, previous research points toward subclinical systolic dysfunction even in diabetics with normal EF. Gehan Magdy et al,[18] (2020) used strain imaging to detect early systolic impairment not revealed by conventional EF measurements — an observation supported by Razavian et al (2021).[11] These findings suggest that diastolic dysfunction is accompanied by subtle systolic changes identifiable only through advanced echocardiographic techniques.

Table 9: The findings in this study are consistent with related cross-sectional and prospective studies

Study	Population	LVDD Prevalence	HbA1c Association
Patil et al. (2021).[16]	127 Indian diabetics	High prevalence	Significant
Mishra et al. (2020).[19]	100 T2DM patients	Lower E velocity & FI abnormalities	Correlated
Zhang et al. (2021).[13]	Asymptomatic DM patients	~45–50% LVDD	Strong correlation
Lee et al. (2022).[17]	T2DM with microalbuminuria	Higher diastolic dysfunction	Yes

These studies collectively validate the high burden of subclinical diastolic dysfunction in DM and the significant impact of poor glycemic control.

Chronic hyperglycemia leads to structural and functional myocardial changes through several mechanisms as Advanced Glycation End Products (AGEs) accumulation increased myocardial stiffness. Oxidative stress and inflammation cause microvascular dysfunction. Fibrosis and extracellular matrix remodeling as impaired relaxation. Lipotoxicity and insulin resistance causing metabolic myocardial injury. These pathways have been substantiated by experimental and clinical research (Seferović & Paulus, 2019; Jia et al., 2020; Håkanson et al., 2022).[9,10]

Diabetic patients with LVDD carry an increased risk of progression to HFpEF and poor clinical outcomes. Annual mortality of diastolic HF (5–15%) is comparable to that of systolic HF, underscoring the need for early detection (Shah et al., 2020; Gu et al., 2023).[12,20]

CONCLUSION

Our study concluded that incidence of left ventricular diastolic dysfunction among the newly diagnosed patient was 48.2% and it was significantly associated with high microalbuminuria, high FBS, PPBS, HbA1C, TC and TG. Accuracy of HbA1C to diagnose the LVDD was found to be 92.2%. Diastolic dysfunction is common even in newly diagnosed type 2 DM patients. HbA1c is strongly associated with LVDD, suggesting that glycemic control may influence early cardiac changes. Early identification of LVDD offers an opportunity for preventive cardiometabolic interventions.

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