|| ISSN(online): 2589-8698 || ISSN(print): 2589-868X ||
International Journal of Medical and Biomedical Studies
Available Online at www.ijmbs.info
NLM (National Library of Medicine ID: 101738825)
Index Copernicus Value 2019: 79.34
Original Research Article
Volume 5, Issue 1; January: 2021; Page No. 115-119

ECHOCARDIOGRAPHIC EVALUATION OF LEFT VENTRICULAR DIASTOLIC DYSFUNCTION IN TYPE 2 DIABETES MELLITUS PATIENTS

Dr. Anand Sagar[1], Dr. Neha Chaturvedi[2], Dr. Unique Sagar[3], Dr. Mustafa Malvi[4] Dr Vipin Porwal[5]
[1] Asst. Prof. Department of Medicine R D Gardi Medical College, Ujjain
[2]Tutor, Department of Pathology L N Medical College, Bhopal
[3]Junior Resident, Department of Pediatrics Index Medical College & Research Centre, Indore
[4]Senior Resident. Department of Medicine R D Gardi Medical College, Ujjain
[5] Assoc. Prof. Department of Medicine R D Gardi Medical College, Ujjain

Article Info: Received 08 December 2020; Accepted 11 January 2021
DOI: https://doi.org/10.32553/ijmbs.v5i1.1622
Corresponding author: Dr. Neha Chaturvedi
Conflict of interest: No conflict of interest.

Abstract
The study was done to find out the left ventricular diastolic dysfunction in type 2 diabetes mellitus patients.
Methods After ethical consideration, Observation based Cross–Sectional study was conducted at R D Gardi Medical College, Ujjain in 75 cases of type 2 diabetes mellitus. All the patients were subjected for routine investigations along with HbA1c and 2D echocardiography for evaluation of the left ventricular function.
Results: Among 75 study cases, forty one (54.7%) had left ventricular diastolic dysfunction. The cases with left ventricular diastolic dysfunction had a mean HbA1c of 8.48.There was significant correlation of left ventricular diastolic dysfunction with HbA1c (p value of 0.001), With rise in HbA1c level there was increased involvement of the myocardium with subsequent increase in severity of left ventricular diastolic dysfunction.
Conclusions: This study concludes that myocardial damage occurs before the appearance of clinical manifestations. Left ventricular diastolic function is affected earlier than the systolic function and is an early marker of diabetic cardiomyopathy. Early intervention and strict control of diabetes should be advised to all diabetes patients to prevent the early involvement of the myocardium.

Introduction

Diabetes is known to mankind for the past 2500 years and day by day it is increasing in an exponential manner becoming the pandemic of modern era. India will have the highest number of diabetic patients in the world by 2025 becoming the Diabetic capital of the world.[1]

The long–term effects of diabetes include development of cardiomyopathy, retinopathy, nephropathy, neuropathy, increased risk of peripheral vascular disease and cerebrovascular accidents.[10]

Coronary artery disease is not the sole cause of cardiac death in diabetic patients. Left ventricular dysfunction and left ventricular hypertrophy are also implicated and unlike coronary artery disease and are the ideal targets for screening.[2,3]
Ruber et al in 1972 first quoted about the diabetic cardiomyopathy which was also supported by Framingham study that showed heart failure being more common in diabetics due to diabetic cardiomyopathy.[4]
The left ventricular diastolic dysfunction is considered as a precursor of diabetic cardiomyopathy and has been described as an early sign of diabetic heart muscle involvement prior to the systolic dysfunction.[5]

Assessment of Diastolic Function Using Doppler

Doppler flow pattern in the left ventricular inflow tract just beyond the mitral valve superficially resembles an, M-mode tracing of the anterior mitral leaflet. There is rapid inflow in the early diastole, decreased flow in the mid diastole and subsequent increased in inflow with atrial systole.[6]

Doppler echo has been used to evaluate left ventricular diastolic function. M-mode techniques have been used to record the rate of relaxation of the left ventricular cavity.

T.V. Strok et al and Nishimura R.A. et al demonstrated that Doppler echocardiography is the primary non-invasive technique used for evaluating left ventricular diastolic function in diabetes. Early diastolic flow is reduced and the velocity following atrial contraction is increased in diabetes.[7]

This phenomena has been quantitated in several ways. The simple technique is to take a ratio of the peak velocity with early filling or 'E' point and the peak velocity with atrial filling or 'A' point. Normally the velocity at the 'E'' point is significantly higher than at the 'A' point.[8]

Regardless of age, however, normal diastolic function can be characterized as the complete and efficient filling of the left ventricle at physiologic pressures. This implies that an abnormally high left atrial pressure is not required and that the left ventricle can fill completely without an associated abnormal increase in pressure during filling.

Various abnormalities in diastolic function, e.g. prolonged isovolumic relaxation period, delayed mitral valve opening and impairment in rapid diastolic filling, increased atrial

contribution of LV filling, reduced E/A mitral ratio have been characteristics findings.[9]

In 1993 A.K. Das et al studied type I patients and evaluated diastolic dysfunction in found it in approximately 24% of 38 patients in absence of any CAD. E velocity, E/A ratio and peak filling rate were the most altered parameters.[10]

Airaksinen et al used digitized M-Mode echocardiography to study 36 young women with diabetes mellitus who were free of clinical cardiac disease or hypertension. Prolongation of early rapid filling was noted in 19 of these women (53 %) which is a parameter for diastolic dysfunction.[11]

In 1995 Figorini et al after his study in patients of diabetes mellitus reached to a conclusion that patients who having no evidence of cardiac disease had significant alteration in E/A ratio and isovolumetric relaxation time.[12]

Abnormal diastolic function. [13,14,15]

It is classified into four grades according to the dominant pathophysiology.

Grade	Stage	Dominant pathophysiology
1	Impaired relaxation	Delayed LV early diastolic active relaxation Normal LA pressure Low opening LA-LV pressure gradient Reduced LV suction force
2	Pseudonormalization	Delayed LV early diastolic active relaxation Mildly elevated LA pressure Low opening LA-LV pressure gradient Reduced LV suction force
3	Restrictive filling (reversible)	Noncompliant LV chamber (increased stiffness) Diminished LV suction forces High opening LA-LV pressure gradient Elevated LA pressure (inflow by "pushing" blood) Failing LA contractility Responds positively to preload reduction
4	Restrictive filling (irreversible)	Noncompliant LV chamber (increased stiffness) Diminished LV suction forces High opening LA-LV pressure gradient Elevated LA pressure (inflow by "pushing" blood) Failing LA contractility No improvement with preload reduction

Aims and Objectives

To find out the left ventricular diastolic dysfunction Echocardiographically in study cases.

Material & Methods

This Observation based Cross–Sectional study was done R D Gardi Medical College, Ujjain for a duration of 18 months in 75 cases of Type 2 Diabetes Mellitus.

Inclusion Criteria

Diabetic Patients in the age group 30-60years, with BP < 130/84 mm Hg in sitting posture on ≥ 2 separate occasions
•Without prior history or symptoms suggestive of hypertension, Coronary artery disease, valvular heart disease or congestive cardiac failure.

Exclusion Criteria

- Any kind of acute complication of diabetes.
- •Patients with BP of >130/84mmhg.
- •Patients on antihypertensive medication.
- •Past history of Myocardial infarction, unstable angina.
- •Patients with Rheumatic Heart disease.

Observations & Results

All selected 75 study cases of type 2 DM, were subjected for echocardiography to establish the evidence of left ventricular diastolic dysfunction. In this study of seventy five type 2 DM patients, left ventricular diastolic dysfunction (LVDD) was present in forty one (54.7%) included in **Group A** while in thirty four (45.3%) cases LVDD was absent and were included in **Group B**. In the present study in **Group A**, five (12.10%) cases were in 4th decade and twelve (29.27%) in 5th while maximum twenty four (58.53%) cases were in the 6th decade. Whereas in **Group B** twelve (35.29%) cases each were in the 4rd and 5th decade. while ten (29.42%) cases in the 6th decade. In the present study in **Group A** the mean age is 50.41 yrs. with a standard deviation of 8.02 while 44.59 yrs. in **Group B.** In the present study in **Group A**, twenty two (53.6%) cases were males while nineteen (46.34%) were females whereas in **Group B** twenty (58.82%) cases were males and fourteen (41.18%) cases were females. The male: female ratio in **Group A** was 1.15:1 while that in **Group B** 1.42:1.In the present study in **Group A**, out of forty one (100%) cases two (4.9%) were having HBA1c <6.4% and three (7.3%) were having between 6.5-7% and thirteen (31.77%) were having 7.1-8% and sixteen (39%) were having 8.1-10% and seven(17.1%) were having HBA1c >10% whereas in **Group B** out of thirty four (100%) cases six (17.6%)were having HBA1c <6.4% and thirteen (38.2%) were having between 6.5-7% and nine (26.5%) were having 7.1-8% and six (17.6%) were having 8.1-10% and there was no case having HBA1c >10%.table--In the present study in **Group A**, eleven (26.84%) cases having diabetes duration <5 years and fourteen (34.14%) were having 5-10 years of diabetes duration and sixteen (39.02%) were having diabetic duration of >10 years. Whereas in **Group B** twenty two (64.70%) cases having diabetes duration <5 years and nine (26.47%) were having 5-10 years of diabetes duration and three (8.83%) were having diabetic duration of >10 years.

In the present study in **Group A** the mean Peak velocity of early mitral flow E is 69.05 with a standard deviation of 8.71 while 75.29 in **Group B** with a standard deviation of 8.31.

In the present study in **Group A** the mean Peak velocity of late mitral flow A is 65.32 with a standard deviation of 10.10 while 60.32 in **Group B** with a standard deviation of 7.82

In the present study in **Group A** the mean E/A Ratio is 10.8 with a standard deviation of 0.25 while 1.3 in **Group B** with a standard deviation of .193

In the present study in **Group A** the mean IVRT is 80.29 with a standard deviation of 7.15 while 77.26 in **Group B** with a standard deviation of 3.02.

In the present study in **Group A** the mean LA Size is 3.14 with a standard deviation of 0.16 while 2.34 in **Group B** with a standard deviation of 0.80

Table 1: Age wise distribution of cases

	Group A	Group B		
Age In Years	No. of cases	Percentage	No. of cases	Percentage
30-40	5	12.10	12	35.29
41-50	12	29.27	12	35.29
51-60	24	58.53	10	29.42
Total	41	100	34	100
Chi-square		df	p value	
8.06		2	0.018	

Table 2: Gender wise distribution of cases

	Group A		Group B	
Gender	No. of cases	Percentage	No. of cases	Percentage
Males	22	53.66	20	58.82
Females	19	46.34	14	41.18
	41	100	34	100
Chi-square		df	p value	
0.2		1	0.6	

Table 3: Duration of Diabetes in Group A and Group B

	Group A		Group B	
Duration Of Diabetes	No	%	No	%
<5 Yrs.	11	26.84	22	64.70
5-10 Yrs.	14	34.14	9	26.47
>10Yrs	16	39.02	3	8.83
Total	41	100	34	100
Chi-square	df	p value		
13.10	2	0.01		

Table 4: Comparison of Hba1c in Group A and Group B.

	Group A		Group B	
HBA1c	No	%	No	%
<6.4%	2	4.9	6	17.6
6.5-7%	3	7.3	13	38.2
7.1-8%	13	31.7	9	26.5
8.1-10%	16	39	6	17.6
> 10%	7	17.1	0	0
	41	100	34	100
Chi-square	df		p value	
20.04	4		<0.001	

Table 5: Mean and SD of Echocardiographic Parameters of LV Diastolic Dysfunction

Variable	Group A		Group B		Significance levels		
	Mean	SD	Mean	SD	Mean diff	t value	p Value
E	69.05	8.71	75.29	8.31	6.245	3.15	0.002
A	65.32	10.10	60.32	7.82	-4.99	-2.35	0.021
E/A Ratio	1.08	0.25	1.3	0.26	.193	3.22	0.002
IVRT	80.29	7.15	77.26	5.5	3.02	2.01	0.04
LA Size	3.14	0.16	2.34	0.31	0.80	14.05	<0.001

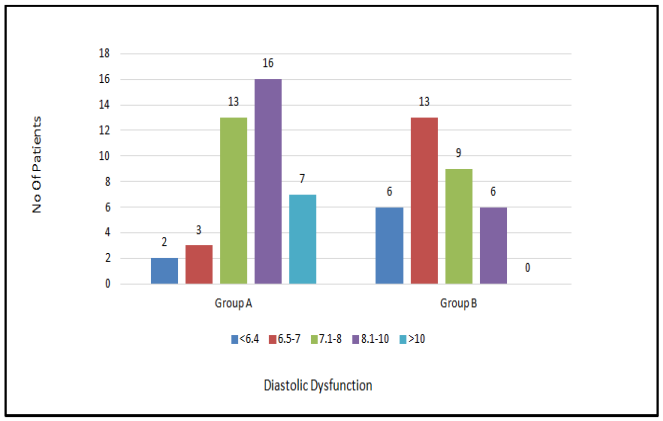

Figure 1: Age wise distribution of cases of Group A and Group B

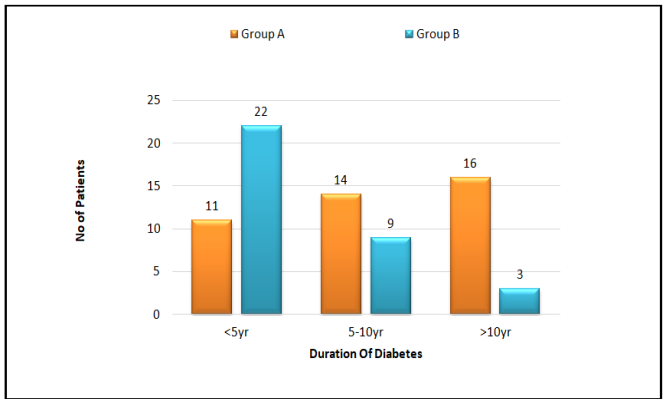

Figure 2: Duration of diabetes of cases of Group A and Group B.

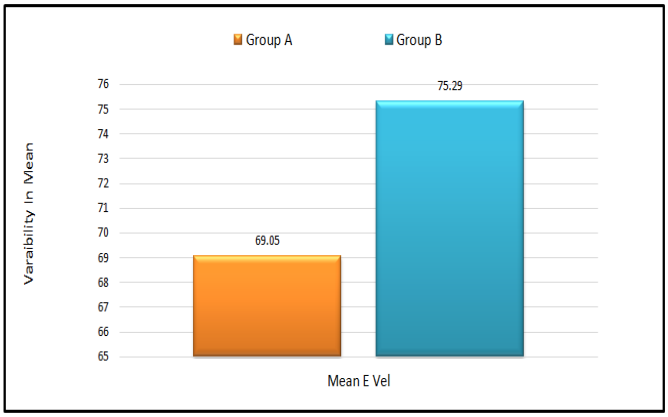

Figure 3: Mean E Velocity of Group A and Group B.

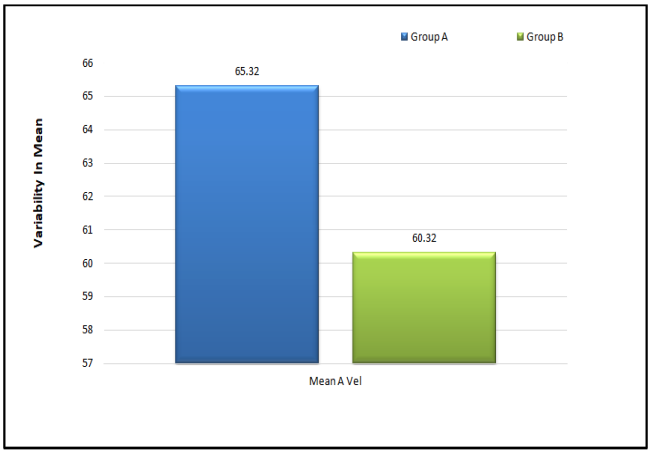

Figure 4: Mean A Velocity of Group A and Group B.

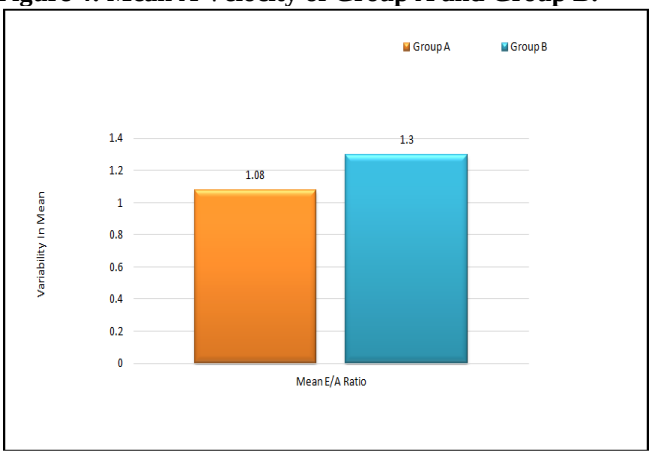

Figure 5: Mean E/A Ratio of Group A and Group B.

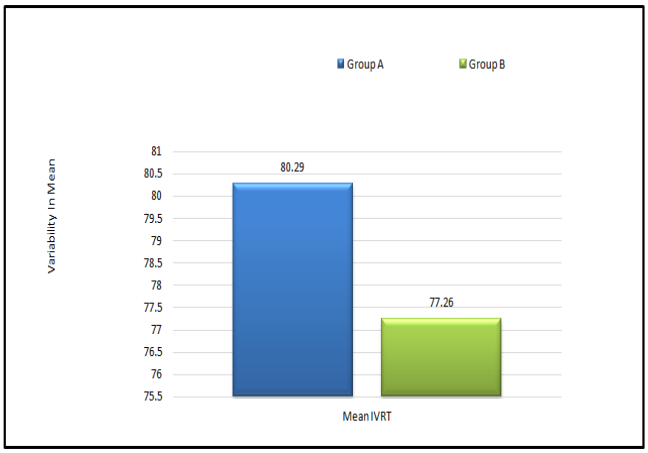

Figure 6: Mean IVRT of Group A and Group B

Conclusions

This study concluded that there is significant myocardial damage in diabetes which occurs before the clinical manifestations appear. Left ventricular diastolic function is affected much earlier in type 2 DM patients than that in normal healthy population and is affected earlier than systolic function and is an early marker of Diabetic Cardiomyopathy. Left ventricular diastolic function was affected by age and more predominant in the 6th decade. Left ventricular diastolic Function was affected more or less equally in both genders. Prevalence of Left ventricular diastolic dysfunction increased gradually with the rise in HbA1c level. Duration of diabetes affects left ventricular diastolic function and is more prevalent in patients of type 2 DM with longer duration of diabetes. The E velocity (p = 0.002), A velocity (p = 0.002), E/A Ratio (p = 0.002), isovolumic relaxation time (p = 0.002) as well as the LA size (p = 0.002) are significantly altered in the patients of diabetic cardiomyopathy.

References

1. National diabetes foundation of India; anoop misra et al indo us study.
2. Ansari M, Alexander M, Tutar A, Massie BM. Incident cases of heart failure in a community cohort: importance and outcomes of patients with preserved systolic function. Am Heart J. 2003; 146: 115–120.
3. Baldwa, Sudhir, Sandeep Mathur K and V.S. Baldwa. 1998. "Silent myocardial ischemia and microvascular disease are associated with LV diastolic dysfunction in NIDDM patients". Jour. Diab Assoc India, 38:55-58.
4. Rubler S, Dlugash J, Yuceoglu YZ, Kumral T, Branwood AW, Grishman A. New type of cardiomyopathy associated with diabetic glomerulosclerosis. AmJ Cardiol,1972; 30, 595-602.
5. Taskiran M, Rasmussen V, Rasmussen B, Fritz-Hansen T, Larsson HB, Jensen GB, Hilsted J. Left ventricular dysfunction in normotensive Type 1 diabetic patients: the impact of autonomic neuropathy. Diabet Med 2004;21:524–530.
6. Yotti R, Bermejo J, Antoranz JC, et al. A noninvasive method for assessing impaired diastolic suction in patients with dilated cardiomyopathy. Circulation 2005; 112:2921-2929
7. .Hurrell D, Nishimura RA, Ilstrup DM, Appleton CP. Utility of preload alteration in assessment of left ventricular filling pressure by Doppler echocardiography: a simultaneous catheterization and Doppler echocardiographic study. J Am Coll Cardiol 1997;30:459-467
8. Hillis GS, Moller JE, Pellikka PA, et al. Noninvasive estimation of left ventricular filling pressure by E/E′ is a powerful predictor of survival after acute myocardial infarction. J Am Coll Cardiol 2004;43:360-367.
9. Nagueh SF, Appleton CP, Gillebert TC, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. J Am Soc Echocardiogr 2008;22:107-133.
10. Das A.K. "Diabetic heart disease, The Indian Scene" in Advances in Endocrinology / metabolism and diabetes, Kochupillai N., New Delhi. MacMiller Indian Ltd., Vol. 2, 1994, 54-60pp.

11. Airaksinen J., Ikaheimo M and Kaila J. 1984. "Impaired left ventricular filling in young female diabetics: An echocardiographic study". Acta Med Scand, 216; 509-516.
12. Fiorini G et al. 1995. "An echocardi'ographic study of left ventricular diastolic function in patients with D.M. type - 2". G.Ital. Cardiol,25,17-28.
13. Melenovsky V, Borlaug BA, Rosen B, et al. Cardiovascular features of heart failure with preserved ejection fraction versus nonfailing hypertensive left ventricular hypertrophy in the urban Baltimore community: the role of atrial remodeling/dysfunction. J Am Coll Cardiol 2007;49:198-207.
14. Takemoto Y, Branes ME, Seward JB, et al. Usefulness of left atrial volume in predicting first congestive heart failure in patients ≥ 65 years of age with well-preserved left ventricular systolic function. Am J Cardiol 2005;96:832-836.
15. Zile MR, Baicu CF, Gaasch WH. Diastolic heart failure: abnormalities in active relaxation and passive stiffness of the left ventricle. N Engl J Med 2004;350:1953-1959.