Innovative Journal of Medical and Health Science
Received 2 Feb 2021 | Revised 23 Apr 2021 | Accepted 3 May 20210 | Published Online 5 May 2021

DOI: https://doi.org/101550.ijmhs.123
Inno J of Med Health Sci 11 (05), 1–8 (2021)

ISSN (O) 2589-9341 | (P) 2277-4939 IF:1.6

**ORIGINAL RESEARCH ARTICLE**

# PATTERN OF LEFT VENTRICULAR ABNORMALITIES IN PEOPLE WITH PREDIABETES

Mohd Abass Bhat[1] | Nazir A Pala[2*] | Manzoor Syed | Anderabi[3] | Shahnawaz Hassan[4]

[1]Senior Resident Department of Medicine, Goverrnment Medical College, J & K, Srinagar, India

[2]Assistant Professor Department of Medicine, Goverrnment Medical College, J & K, Srinagar, India

[3]Associate Professor Department of Medicine, Goverrnment Medical College, J & K, Srinagar, India

[4]Senior Resident Department of Medicine, Goverrnment Medical College, J & K, Srinagar, India

Abstract

**Purpose of study:** Left ventricular dysfunction in people with diabetes mellitus has been demonstrated by various studies in past. However, there is little and conflicting data with regard to Left Ventricular abnormalities in people with Pre-diabetes. In view of Scarcity of data, we designed a study to investigate the early abnormalities in left ventricular function in normotensive people with prediabetes compared to healthy controls, using Doppler Echocardiography

**Methods:** Prospective hospital based case-control study, conducted at a tertiary care hospital in north India. 100 people with prediabetes without known cardiovascular disease, with equal number of matched controls were enrolled in the study. A detailed medical history, clinical examination, biochemical profile and echocardiographic studies were performed in all study subjects.

**Results:** Early diastolic wave (E;0.693±0.092m/s), Early diastolic wave (E)/late diastolic wave (A) ratio (E/A;0.887±0.095), left ventricular end systolic diameter (LVESD; 24.78±1.640mm) were significantly lower in people with prediabetes, whereas isovolumetric relaxation time (IVRT;87.45±8.148ms), sphericity index (0.595±0.028) and left atrial diameter (35.50±1.693mm) were significantly higher in people with pre-diabetes compared to matched controls. However, no correlation was observed with regard to anthropometric variables (body-mass index (BMI), waist circumference) and lipid profile

**Conclusion:** Prediabetes is associated with subclinical left Ventricular systolic and diastolic abnormalities as evaluated on conventional and pulse-wave echocardiography.

Keywords: Echocardiography, Left Ventricular function, Prediabetes PATTERN OF LEFT VENTRICULAR ABNORMALITIES IN PEOPLE WITH PREDIABETES

## 1 | INTRODUCTION

Prediabetes is an intermediate state of hyperglycemia with glycemic parameters above normal but below the diabetes threshold (1). Prediabetes is considered a progenitor for progression to diabetes and is associated with and micro and macro-vascular complications (cardiovascular disease) in a way similar, although to a lesser extent compared to diabetes mellitus (2, 3) .People with pre-diabetes have 20% increased risk of developing cardiovascular disease compared to normoglycemic subjects. Although the exact mechanism of prediabetes leading to increased risk of cardiovascular disease is not clear, however, pathophysiological disturbances same as in diabetes like insulin resistance, impaired beta cell function and decreased compliance of the central arterial system( arterial stiffness) (2–4) have been proposed as possible mechanisms. Similarly various other factors like, neurohumoral pathways, cytokines, immune factors, oxidative stress and the accumulation of advanced glycosylation end products (AGEs) leading to protein cross-linking have also been found to be associated with cardiovascular and other complications related to hyperglycemia (5–7). Although diabetes mellitus is known to be related to left ventricular dysfunction in normotensive people without known coronary artery disease, the data with regard to the left ventricular abnormalities in people with pre-diabetes is conflicting and scare. In view of scarcity and conflicting data, we aimed to investigate the possible impact of Prediabetes on both LV systolic and diastolic function by using conventional echocardiographic techniques including pulse-wave echocardiographic meaurements.

## 2 | MATERIAL AND METHODS

Prospective case-control study, was conducted at a tertiary care hospital in north India. 100 people with pre-diabetes with equal number of age, gender and BMI matched controls were enrolled in the study after Institutional ethical committee clearance. Informed consent was taken from all study subjects. People with fasting plasma glucose between 100-125mg/dl and/or glycated hemoglobin (HbA1c) values between 5.7%-6.4% (1), were included in the study. People with hypertension, diabetes mellitus, chronic kidney disease, cardiovascular disease, malignancy, severe obesity (BMI>40kg/m2), heavy smokers, adults > 60 years of age, anemia, or any hemoglobinopathies, taking drugs having effect on glucose metabolism or alcohol intake. Detailed medical history, clinical examination including blood pressure (BP) and body mass index (BMI) was recorded and biochemical profile and echocardiography was performed in all study subjects. The BMI was calculated as weight/height$^2$ (kg/m$^2$) and was used as an estimate of the overall adiposity (8). Central obesity was defined as waist circumference >102 cm in males and >88 cm in females (9).Prediabetes was diagnosed according to the American Diabetes Association (ADA) criteria**.** Blood was drawn after fasting for 8 hours. A fasting plasma glucose (FPG) level below 100 mg/dl and/or glycosylated hemoglobin (HbA1c) <5.7% was considered normal. A FPG level between 100 and 125 mg/dl and/or HbA1c 5.7–6.4% on two separate occasions was labelled as Prediabetes (10).

Echocardiographic studies

Echocardiography has introduced the possibility to evaluate systolic and diastolic function. People with Left ventricular systolic dysfunction have reduced ejection fraction, ejection time and prolongation of isovolumetric contraction time; Where as those with left ventricular diastolic dysfunction have increase in the isovolumetric relaxation time and alteration of the timing of diastolic filling .

In our study, left ventricular structural and functional measurements were performed with ultrasonoscope, using a 2.5 and 3.5 MHz transducer. Measurements of Ventricular septum, Posterior wall and Left ventricular cavity (both end systolic and end diastolic

**Supplementary information** The online version of this article (101550.ijmhs.123) contains supplementary material, which is available to authorized users.

**Corresponding Author:** *Nazir A Pala*
*Goverrnment Medical College, Assistant Professor*
*Department of Medicine*
*Email: nazirpala2@gmail.com*

dimensions) were performed according to the American Society of Echocardiography criteria[15]. Left ventricular mass was calculated from the left ventricular end-diastolic cavity and septal and posterior wall thickness was calculated using the Penn convention and the American Society of Echocardiography guidelines (11)**.** The relative wall thickness (RWT) was measured at the end-diastole as the ratio of posterior wall thickness (PoW), plus Interventricular septal (IVS) thickness divided by left ventricular internal dimension. The transmitral flow velocity profile was recorded from the apical four-chamber view with the pulsed wave Doppler sample volume positioned at the tips of mitral leaflets during diastole. The left ventricular outflow velocity pattern was recorded from the apical long axis view, with the pulsed wave Doppler sample volume positioned just below the aortic valve. Five consecutive beats were measured and averaged for each measurement. Early diastolic peak flow velocity(E), late diastolic peak flow velocity (A), E-wave deceleration time (DT), isovolumetric contraction time (IVCT), ejection time (ET) and isovolumetric relaxation time (IVRT) were measured by transmitral Doppler imaging. The IVRT was measured with the pulsed wave sample volume placed between the mitral inflow and the left ventricular outflow tract (12–14). E/A ratio was calculated. Myocardial performance index (MPI) was calculated by summing of IVRT and IVCT and dividing by ET. The sphericity index (SI) was calculated as the ratio between the greater cross sectional diameter and the greater longitudinal diameter of the LV in end-diastolic apical four-chamber view. This index was used as an indicator of geometry change. The data was collected, coded and organized. The final study results were stated using the SPSS program version $_{14}$. Student-t, correlation coefficient and Chi-square tests were used to evaluate the results. Chi-square test was used for qualitative variables while independent t-test was used for quantitative variables. Correlation analysis was performed using Pearson's correlation coefficient. Statistical significance was considered at a P-value <0.05 and highly significant at a P value <0.001.

## 3 | RESULTS

The baseline characteristics of the study population are presented in [Table1]. Age, gender, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, smoking rate, total cholesterol, low-density lipoprotein (LDL), total cholesterol, high-density lipoprotein (HDL) cholesterol and triglyceride levels were comparable in two study groups. However, fasting plasma glucose (FPG) level (112.04 ± 3.85mg/dl vs 84.75 ± 3.89mg/dl, respectively, $p < 0.001$) and HbA1c level (5.99 ± 0.17 % vs 5.04 ± 0.12%, respectively, $p < 0.001$) were significantly higher in people with prediabetes compared to healthy subjects.

The conventional echocardiographic results of the subjects are presented in [Table 2]. Accordingly, Left Ventricular end-diastolic diameter, thickness of Interventricular septum, Posterior Wall thickness and LV ejection fraction were similar among the groups. However, Left Atrial diameter (35.50 ± 1.693mm vs 33.48 ±1.918mm, respectively, $p< 0.001$) was significantly higher in Prediabetes group than in controls. Whereas LVESD (left ventricular end systolic diameter; 24.78 ± 1.640mm vs 26.11 ± 1.603mm, respectively, $p< 0.001$) was significantly lower in prediabetes group. Pulse-wave echocardiographic measurements of the subjects are demonstrated in [Table 3]. According to these results, no statistically significant difference was seen in A- (late diastolic) wave, IVCT (isovolumetric contraction time), ET (ejection time), IVCT/ET and MPI (myocardial performance index) between two groups. However, E-wave (0.693 ± 0.092 m/s vs 0.891 ± 0.083 m/s, respectively, $p< 0.001$) and E/A ratio (0.887 ± 0.095 vs 1.161 ± 0.075, respectively, $p< 0.001$) were significantly lower in Prediabetic group. Further, IVRT (isovolumetric relaxation time 87.45 ± 8.148ms vs 70.780 ± 9.874ms, respectively, $p< 0.001$), DT (deceleration time 211.0 ± 26.36ms vs 168.24 ± 18.90ms, respectively, $p< 0.001$) and SI (sphericity index 0.595 ± 0.028 vs 0.497 ± 0.026, respectively, $p< 0.001$) were significantly higher in prediabetic group as compared to controls.

**TABLE 1:** Clinical and demographic characteristics of study population

| Variables | Prediabetes(n=100) | Healthy controls (n=100) | P - value |
|---|---|---|---|
| Age(years) | 50.62±3.50 | 50.67±3.67 | 0.969 |
| Gender(M/F) | 45/55 | 43/57 | 0.776 |
| BMI(kg/m$^2$) | 29.91±2.55 | 29.61±2.28 | 0.352 |
| Pulse(beats/min) | 76.31±3.67 | 76.17±3.76 | 0.979 |
| SBP(mmHg) | 121.30±5.44 | 121.28±5.44 | 0.968 |
| DBP(mmHg) | 76.60±3.54 | 76.58±3.57 | 0.791 |
| Smokers (%) | 28 | 31 | 0.642 |
| FBG(mg/dl) | 112.04±3.85 | 84.75±3.89 | < 0.001 |
| HbA1c (%) | 5.99±0.17 | 5.04±0.12 | < 0.001 |
| Total cholesterol (mg/dl) | 191.81±10.93 | 189.42±9.41 | 0.099 |
| LDL cholesterol(mg/dl) | 150.14±41.72 | 158.43±46.38 | 0.185 |
| HDL cholesterol(mg/dl) | 44.31±6.15 | 45.60±6.07 | 0.137 |
| Triglycerides(mg/dl) | 151.03±18.35 | 147.41±18.34 | 0.164 |

M/F: male/female; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; FBG: fasting plasma glucose; HbA1C: glycosylated hemoglobin; LDL: low-density lipoprotein; HDL: high-density lipoprotein

**TABLE 2:** Conventional echocardiographic parameters of study population

| Variable | Prediabetes (n=100) | Healthy controls(n=100) | P value |
|---|---|---|---|
| LVESD (mm) | 24.78 ± 1.640 | 26.11 ± 1.603 | < 0.001 |
| LVEDD (mm) | 44.26 ± 2.045 | 44.85 ± 2.039 | 0.071 |
| LA (mm) | 35.50 ± 1.693 | 33.48 ± 1.918 | < 0.001 |
| PoW (mm) | 9.49 ± 0.561 | 8.80 ± 0.558 | < 0.001 |
| IVS (mm) | 9.16 ± 0.918 | 8.97 ± 0.629 | 0.089 |
| LVEF (%) | 64.91 ± 1.676 | 65.14 ± 1.952 | 0.371 |

LVESD = left ventricular end systolic diameter, LVEDD= left ventricular end diastolic diameter, LA = diameter of left atrium, PoW=Thickness of posterior wall, IVS = thickness of interventricular septum, LVEF= left ventricular ejection fraction

**TABLE 3:** Pulse-wave echocardiographic measurements of study population

| Variable | Prediabetes | Healthy Controls | P – value |
|---|---|---|---|
| E (m/s) | 0.693 ± 0.092 | 0.891 ± 0.083 | < 0.001 |
| A (m/s) | 0.834 ± 0.319 | 0.765 ± 0.260 | 0.095 |
| E/A | 0.887 ± 0.095 | 1.161 ± 0.075 | < 0.001 |
| DT (ms) | 211.0 ± 26.36 | 1.161 ± 0.075 | < 0.001 |
| IVCT (ms) | 64.74 ± 9.494 | 66.95 ± 8.893 | 0.091 |
| ET (ms) | 281.90 ± 16.11 | 277.4 ± 16.24 | 0.141 |
| IVCT/ET | 0.232 ± 0.029 | 0.239 ± 0.024 | 0.185 |
| IVRT (ms) | 87.450 ± 8.148 | 70.78 ± 9.874 | < 0.001 |
| MPI | 0.546 ± 0.269 | 0.501 ± 0.156 | 0.149 |
| Sphericity Index | 0.595 ± 0.028 | 0.497 ± 0.026 | < 0.001 |

E=early diastolic wave, A=late diastolic wave, DT=deceleration time, IVCT= Iso-volumetric contraction time, ET = ejection time, IVRT = isovolumetric relaxation time, MPI = myocardial performance index

## 4 | DISCUSSION

Hyperglycemia has been demonstrated to be the progenitor of advanced glycosylation end-products (AGE), enhancement of progressive loss of cardiomyocytes and increase in fibrosis resulting from oxidative stress and inflammation. Diabetic heart may be affected by way of ventricular hypertrophy, metabolic abnormalities, extracellular matrix remodelling, fibrosis, vascular changes, insulin resistance, oxidative stress and apoptosis leading to abnormalities in myocardial function. As a result, it has been assumed that prediabetic patients may have decreased LV function due to prolonged hyperglycemia (15, 16)**.** In our study, we demonstrated. that patients with Prediabetes have both impaired LV systolic and diastolic function. As we excluded people with other co-morbidities, like hypertension (HTN), coronary artery disease (CAD) and other chronic systemic illnesses, LV abnormalities observed in people with prediabetes probably depend directly on impaired glucose metabolism in these people. The increased prevalence of diastolic dysfunction and its severity in proportion to the degree of hyperglycemia, people with prediabetes has been demonstrated in the past (16) .Hyperglycemia may also stimulate apoptosis, myocyte necrosis with eventual myocardial cell loss (17, 18) which may impair myocardial contractility and lead to systolic dysfunction. LV diastolic dysfunction could represent the earliest pre-clinical manifestation of myocardial involvement in diabetes, prior to any systolic dysfunction (19, 20)· Moreover, myocardial abnormalities in an individual could be demonstrated before detection of hyperglycemia (21), indicating that apart from being a complication of diabetes, it could represent a coexisting condition.

In view of cardiovascular repercussions associated with hyperglycemia, a detailed evaluation of cardiovascular alterations in people with prediabetes is mandated. We studied normotensive people with prediabetes to look for any subclinical left ventricular abnormalities in these people. We observed that prediabetes is associated with impaired left ventricular function and that it appears to be independent status of blood pressure, ventricular geometry, glucose tolerance, lipids and adiposity. The E/A ratio exhibited a stepwise decrease from the control group to the prediabetic group, owing to increased A-wave velocity and isovolumic relaxation time (IVRT) was significantly longer in the people with prediabetes. These findings suggest that there is a progressive impairment in left ventricular relaxation during diastole, which is consistent with that of a recently published study in patients without overt type 2 diabetes (22) .In addition; the diastolic changes were associated with an unaltered geometric pattern. These observations indicate that the functional changes in people with hyperglycemia are independent and precede the systolic and structural changes. Similar, observation was made in prior studies in people with obesity, metabolic syndrome and diabetes (23–25). In our study, people with prediabetes exhibited a higher Left Atrial Volume (LAV) with respect to healthy controls, which indicates chronic and long-standing pressure overload on left ventricle in people with hyperglycemia. In agreement with our data, Dinh et al (22) showed an increase in LAV with transition from Prediabetes to overt type 2 diabetes mellitus. In addition to this, the study reported a significant correlation between HbA1c and LAV, indicative of left ventricular diastolic abnormalities with elevated filling pressure, even in people with normoglycemia. In our study population, people with prediabetes had higher Sphericity Index (SI) compared to healthy controls and was independently related with HbA1c levels. These structural and functional abnormalities of left are clinically relevant because abnormal left ventricular remodelling has been associated with worse prognostic outcome (26). In particular, abnormal left ventricular sphericity has also been associated with increased mortality after acute myocardial infarction (27)·as has been previously demonstrated in a population based study, where people with a higher sphericity had increased incidence of Heart failure and Atrial Fibrillation (28). In conclusion, people with prediabetes in our study population had an impaired left ventricular function, which represent a major pattern of cardiovascular disease (CVD). None of our prediabetic adults had history of overt cardiovascular complications or events. Furthurmore, detection of left ventricular abnormalities without clinically apparent heart failure in people with prediabetes in our study indicates that

echocardiographic assessment for all people with prediabetes should be performed as the primary prevention for the development of future CVD.

## 5 | LIMITATIONS OF THE STUDY

Several potential limitations of this study need to be highlighted**.** First, Although People with prediabetes in our study did not have symptomatic CHD; screening for CHD using Coronary angiography was not performed. Second,E/A should be interpreted in conjunction with clinical characteristics and other echocardiographic parameters, although it has important diagnostic and prognostic implications. Thirdly, in of small sample size of people with prediabetes in our study, results cannot be generalised to all people with prediabetes. Fourthly, although LA volume is a more precise indicator of chronic diastolic dysfunction, we have utilized LA diameter.

Conflict of interest: None

## REFERENCES


1. Cheng YJ, Gregg EW, Geiss LS, Imperatore G, Williams DE, Zhangx. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2012;35(1):64–71.

2. Standards of medical care in diabetes - 2012. Diabetes Care. 2012;35(1):11–63.

3. Arnett DK, Evans GW, Riley WA. Arterial Stiffness: A New Cardiovascular Risk Factor? American Journal of Epidemiology. 1994;140(8):669–682. Available from: https://dx.doi.org/10.1093/oxfordjournals.aje.a117315. doi:10.1093/oxfordjournals.aje.a117315.

4. Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, et al. Aortic Stiffness Is an Independent Predictor of All-Cause and Cardiovascular Mortality in Hypertensive Patients. Hypertension. 2001;37(5):1236–1241. Available from: https://dx.doi.org/10.1161/01.hyp.37.5.1236. doi:10.1161/01.hyp.37.5.1236.

5. Steinberg HO, Chaker H, Leaming R, Johnson A, Brechtel G, Baron AD. Obesity/insulin resistance is associated with endothelial dysfunction. Implications for the syndrome of insulin resistance. Journal of Clinical Investigation. 1996;97(11):2601–2610. Available from: https://dx.doi.org/10.1172/jci118709. doi:10.1172/jci118709.

6. Singh R, Barden A, Mori T, Beilin L. Advanced glycation end-products: a review. Diabetologia. 2001;44(2):129–146. Available from: https://dx.doi.org/10.1007/s001250051591. doi:10.1007/s001250051591.

7. Arcaro G, Cretti A, Balzano S, Lechi A, Muggeo M, Bonora E, et al. Insulin Causes Endothelial Dysfunction in Humans. Circulation. 2002;105(5):576–582. Available from: https://dx.doi.org/10.1161/hc0502.103333. doi:10.1161/hc0502.103333.

8. Sunyer FX, Ca M, Pi G. Obesity and type 2 diabetes. Endocrinol Metab Clin North Am. 2000;3:521–529.

9. Grundy SM. Multifactorial causation of obesity; Implications for prevention. Am J Clin Nutr. 2003;67:563–72.

10. Dujardin KS, Tei C, Yeo TC, Hodge DO, Rossi A, Seward JB. Standard of medical care in diabetes-2008. Diabetes care. 2008;31.

11. Schiller NB, Shah PM, Crawford M, Demaria A, Devereux R, Feigenbaum H. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr. 1989;2(5):358–67.

12. Laskowski C, Zhan WZ. Myocardial contractility by strain echocardiography: Comparison with physiological measurements in an in-vitro model. Am J. 2003;285:2599–604.

13. Heatlie GJ. Echocardiography and the general physician. Postgraduate Medical Journal. 2004;80(940):84–88. Available from: https://dx.doi.org/10.1136/pmj.2003.010363. doi:10.1136/pmj.2003.010363.

14. Saul G, Myerson L. Left Ventricular Mass: Reliability of M-Mode and2- Dimensional Echocardiographic Formulas. Hypertension. 2002;40:673–681.

15. Singh R, Barden A, Mori T, Beilin L. Advanced glycation end-products: a review. Diabetologia. 2001;44(2):129–146. Available from: https://dx.doi.org/10.1007/s001250051591. doi:10.1007/s001250051591.

16. Stahrenberg R, Edelmann F, Mende M, Kockskämper A, Düngen HD, Scherer M, et al. Association of glucose metabolism with diastolic function along the diabetic continuum. Diabetologia. 2010;53(7):1331–1340. Available from: https://dx.doi.org/10.1007/s00125-010-1718-8. doi:10.1007/s00125-010-1718-8.

17. Cai L, Li W, Wang G, Guo L, Jiang Y, Kang YJ. Hyperglycemia-Induced Apoptosis in Mouse Myocardium: Mitochondrial Cytochrome c-Mediated Caspase-3 Activation Pathway. Diabetes. 2002;51(6):1938–1948. Available from: https://dx.doi.org/10.2337/diabetes.51.6.1938. doi:10.2337/diabetes.51.6.1938.

18. Bojunga J, Nowak D, Mitrou PS, Hoelzer D, Zeuzem S, Chow KU. Antioxidative treatment prevents activation of death-receptor- and mitochondrion-dependent apoptosis in the hearts of diabetic rats. Diabetologia. 2004;47(12):2072–2080. Available from: https://dx.doi.org/10.1007/s00125-004-1572-7. doi:10.1007/s00125-004-1572-7.

19. Raev DC. Which Left Ventricular Function Is Impaired Earlier in the Evolution of Diabetic Cardiomyopathy?: An echocardiographic study of young type I diabetic patients. Diabetes Care. 1994;17(7):633–639. Available from: https://dx.doi.org/10.2337/diacare.17.7.633. doi:10.2337/diacare.17.7.633.

20. Zabalgoitia M, Ismaeil MF, Anderson L, Maklady FA. Prevalence of diastolic dysfunction in normotensive, asymptomatic patients with well-controlled type 2 diabetes mellitus. The American Journal of Cardiology. 2001;87(3):320–323. Available from: https://dx.doi.org/10.1016/s0002-9149(00)01366-7. doi:10.1016/s0002-9149(00)01366-7.

21. Stahrenberg R, Edelmann F, Mende M, Kockskämper A, Düngen HD, Scherer M, et al. Association of glucose metabolism with diastolic function along the diabetic continuum. Diabetologia. 2010;53(7):1331–1340. Available from: https://dx.doi.org/10.1007/s00125-010-1718-8. doi:10.1007/s00125-010-1718-8.

22. Dinh W, Lankisch M, Nickl W, Scheyer D, Scheffold T, Kramer F, et al. Insulin resistance and glycemic abnormalities are associated with deterioration of left ventricular diastolic function: a cross-sectional study. Cardiovascular Diabetology. 2010;9(1):63–63. Available from: https://dx.doi.org/10.1186/1475-2840-9-63. doi:10.1186/1475-2840-9-63.

23. Alterations in left ventricular structure and function in young healthy obese women: Assessment by echocardiography and tissue Doppler imaging. J Am Coll Cardiol. 2004;43:1399–404.

24. de Simone G, Palmieri V, Bella JN, Celentano A, Hong Y, Oberman A, et al. Association of left ventricular hypertrophy with metabolic risk factors: the HyperGEN study. Journal of Hypertension. 2002;20(2):323–331. Available from: https://dx.doi.org/10.1097/00004872-200202000-00024. doi:10.1097/00004872-200202000-00024.

25. From AM, Scott CG, Chen HH. The Development of Heart Failure in Patients With Diabetes Mellitus and Pre-Clinical Diastolic Dysfunction. Journal of the American College of Cardiology. 2010;55(4):300–305. Available from: https://dx.doi.org/10.1016/j.jacc.2009.12.003. doi:10.1016/j.jacc.2009.12.003.

26. Sutton MGSJ, Sharpe N. Left Ventricular Remodeling After Myocardial Infarction. Circulation. 2000;101(25):2981–2988. Available from: https://dx.doi.org/10.1161/01.cir.101.25.2981. doi:10.1161/01.cir.101.25.2981.

27. Wong SP, French JK, Lydon AM, Manda SOM, Gao W, Ashton NG, et al. Relation of left ventricular sphericity to 10-year survival after acute myocardial infarction. The American Journal of Cardiology. 2004;94(10):1270–1275. Available from: https://dx.doi.org/10.1016/j.amjcard.2004.07.110. doi:10.1016/j.amjcard.2004.07.110.

28. Ambale-Venkatesh B, Yoneyama K, Sharma RK, Ohyama Y, Wu CO, Burke GL. Left ventricular shape predicts different types of cardiovascular events in the general population. Heart; 2016.

**How to cite this article:** Abass Bhat M., Pala N.A., Syed M., Anderabi , Hassan S. **PATTERN OF LEFT VENTRICULAR ABNORMALITIES IN PEOPLE WITH PREDIABETES**. Innovative Journal of Medical and Health Science. 2021;1−8. https://doi.org/101550.ijmhs.123