Original Article

Assessment of Left Ventricular Geometry in Normotensive Type II Diabetic Patients

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Abstract

Background: Abnormalities of glucose metabolism are associated with abnormal left ventricular geometry (LV) independent of atherosclerosis. Abnormal LV geometry, a predictor of premature cardiovascular events, indicates presence of subclinical target organ damages. Screening for abnormal LV geometry in diseases of abnormal glucose metabolism is desirable as part of their management protocol. **Aim:** To assess the left ventricular geometry in normotensive type II diabetic patients. Cross-sectional, descriptive, hospital-based study. One hundred normotensive type II diabetic patients drawn from the Endocrinology and Family Medicine Clinics of a tertiary hospital were age- and gender-matched with 100 apparently healthy controls. Participants meeting the criteria and informed consent proceeded for clinical evaluation, biochemical assessment, electrocardiography, and echocardiography using the American Society of Echocardiography guideline. **Materials and Methods:** Data were analyzed using the Statistical Package for Social Sciences [SPSS] version 25.0 (Chicago Illinois, USA). **Results:** Mean age of study and control groups was (55.56 ± 9.89 versus 55.47 ± 10.7) years ($\chi 2 = 0.062$, $P = 0.951$). The mean duration of diabetes illness was 6.57 ± 6.26 years. Prevalence of abnormal LV geometry was 51% (study) versus 18% (control) FT, $P < 0.001$). Concentric remodeling was the predominant geometry in 36% of study versus 11% of controls, followed by eccentric hypertrophy in 11% (study) versus 4% (control) and concentric hypertrophy in 4% (study) versus 3% (control). Geometry was normal in 49% of study against 82% in the controls (FT, $P < 0.001$). Significant association existed between LV geometry and duration of diabetes ($\chi 2 = 10.793$, $P = 0.005$). **Conclusion:** Abnormal LV geometry is highly prevalent in normotensive diabetic patients.

Keywords: *Diabetes, echocardiography, left ventricular geometry, normotension*

Introduction

Left ventricular (LV) geometry is the relationship between left ventricular wall thickness indexed to body surface area[1] or height. A strong link exists between diabetes mellitus (DM) and greater risk for the onset of cardiovascular disease (CVD)[2] which is a common complication of DM.[3] Left ventricular hypertrophy (LVH) could be one of the links between DM and non-obstructive coronary heart disease (CHD)[4,5] and in the presence of DM, can be subclinical predicting an increased incidence of cardiac events, mortality, and total mortality.[6]

There is no systematic screening for abnormal LV geometry in DM patients in most of endocrinology clinics in Nigeria unless there are overt cardiovascular complications at which point it may not be reversible. The main aim of this study is to assess the LV geometric patterns in normotensive type II diabetic patients, and the specific aim is to determine the echocardiographic

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prevalence of LVH in normotensive type II diabetic patients. We hypothesize that DM causes an abnormal LV geometry in the absence of co-existing hypertension.

DM in the absence of hypertension causes an abnormal LV geometry, of which the predominant pattern is concentric remodeling that may occur early in the course of the disease. Screening is recommended earlier in the course of their management as some category of abnormal geometry is reversible with aggressive treatment, thereby yielding significant long-term benefits.

Subjects and Methods

Study was carried out in a tertiary, multispeciality, referral hospital located in the southeastern part of the country. It was a descriptive, cross-sectional study. Minimum sample size was derived with the formulae Z^2pq/d^2 using the prevalence of DM in Nigeria at 5.77%.[7] Approval was obtained from the Ethics Committee of the hospital with reference no NHREC/05/01/2008B-FWA00002458-1RB00002323 on July 23, 2018.

One hundred normotensive type II diabetic patients aged 30–70 years were recruited consecutively from the Endocrinology and Family Medicine Clinics of the hospital. Diagnosis of DM was made according to the American Diabetes Association (ADA)[8] criteria when fasting blood glucose (FBG) was ≥126 mg/dl (7 mmol/l). Exclusion criteria included patients with hypertension defined as blood pressure (BP) ≥130/80 mmHg[9] or on any antihypertensive agent, pregnant women, patients on insulin therapy, terminal illnesses, stroke, current smoking history, established heart failure or valvular heart disease, clinical and or electrocardiographic (ECG) evidence of myocardial infarction (MI), ECG evidence of atrial fibrillation, and laboratory evidence of chronic kidney disease (eGFR <60 ml/min/1.73 m^2).

The control group consisted of apparently healthy individuals who had no hypertension or DM or any medical or surgical condition. They were recruited from among staff of the hospital, patients' relatives, and two elderly peoples' homes. Both groups were matched for age and gender in a 1:1 ratio.

The sociodemographic and clinical data were obtained with an interviewer administered questionnaire. Information obtained included biodata, diabetes treatment and complications history, hypertension, and smoking history as well as history of angina pectoris and MI.

Blood pressure (BP) measurements were obtained using a digital Omron upper arm blood pressure monitor HEM-7130. It was checked on both arms of which the arm with the higher reading was used for further BP checks. An average of the three readings measured more than 24 hours apart on three occasions were used for the study. The patients were instructed to avoid caffeine containing drinks 30 minutes before BP check. They were also seated quietly for minimum of 30 mins before checks.

Weight and height measurements were taken with the patients standing on a standard weighing scale and stadiometer with no shoes, heavy clothing, or head gear. Both readings were reported to the nearest kilogram and centimeter.

A 12-hr overnight FBG levels were measured using the glucose oxidase +4 aminoantipyrine (GOD-PAP) and hexokinase method.[10] $HBA1_C$ was done using the immunoturbidimetric method.[11] Body mass index (BMI) was calculated using the formula: weight (kg)/height^2 (cm).

Echocardiography was performed using GE Healthcare LOGIQ e/Vivid-e echocardiograph machine with a 3.5 MHz transducer. It was performed with the patients in the left lateral decubitus position with the left arm in flexed position and supporting the head in order to spread the intercostal spaces.

One cardiologist with an oversea training in echocardiography performed the echocardiography using the American Society of Echocardiography (ASE) guideline,[12] in the standard parasternal long, short axis and apical four chamber views.[13] A two-dimensional (2D)-guided direct measurement of the interventricular septal wall thickness at end diastole (IVSd), LV internal diameter at end diastole (LVIDd), and the posterior wall thickness (PWTd) at end diastole measurements were obtained according to ASE guideline.[12] The machine automatically calculated the ejection fraction (EF) using the Teichholz's formula,[13] LV mass (LVM) and the LV mass index (LVMI) using the cubed formula[13] LVMI = {0.8 × [1.04 × (LVIDd + LVPWd + IVSd) 3 – LVIDd)]} + 0.6g/BSA. LV mass indexed to BSA of >95 g/m^2 was considered abnormal for females and >115 g/m^2 for males, hence the presence of an increased LVMI. LVM was also indexed to height raised to allometric power of 2.7 of which values >44 g/$ht^{2.7}$ for females and >48 g/$ht^{2.7}$ for males were considered abnormal,[14] signifying an elevated LVMI. The relative wall thickness (RWT) was calculated with the formula[12,14]: RWT = 2 × PWTd/LVIDd.

Calculation of RWT enables the categorization of LV geometry as[12,14]:

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- Concentric hypertrophy when RWT was greater than 0.42 plus an increased LV mass index of greater than 95 g/m^2 for women and 115 g/m^2 for men.
- Eccentric hypertrophy when RWT was less than or equal to 0.42 plus an increased LV mass index of greater than 95 g/m^2 for women and 115 g/m^2 for men.
- Concentric remodeling when RWT was greater than 0.42 plus a normal LV mass index of less than or equal to 95 g/m^2 for women and 115 g/m^2 for men.
- Normal geometry when RWT was less than or equal to 0.42 plus a normal LV mass index of less than or equal to 95 g/m^2 for women and 115 g/m^2 for men.

Statistical analysis

Frequency tables, percentages, mean, and standard deviation were used to provide a descriptive summary of the study participants. Student's t-test was used to compare means of numerical variables, while Pearson's Chi-square test was used for categorical variables. Associations between categorical variables were also cross-tabulated and tested using Chi-square. Fisher's exact test was applied when more than 25% of values were less than 5. Where data were skewed, a Wilcoxon test was applied. *P* value of less than or equal to 0.05 was considered significant.

Results

The mean age of study/controls was 55 ± 9.89/55.47 ± 10.7 years. Study had patients with a mean duration of DM illness of 6.57 ± 6.26 years. The mean FBG was 200.70 mg/dl (11.15 mmol/l) ± 86.15 mg/dl (11.14.79 mmol/l). Ninety-two percent of the study had uncontrolled DM (glycated hemoglobin ($HbA1_c$) ≥ 6.5%).

Table 1 shows the demographic and clinical characteristics of participants. Sixty-nine percent of study participants were in the age range of 51–70 years.

Table 2 shows the LV geometric patterns of participants. An abnormal geometry existed in (51) 51% of study versus (18) 18% of controls (FT, $P < 0.001$).

Table 3 shows the relationship between left ventricular geometry and duration of DM of the study group. Thirty-three percent of the study group had DM diagnosed in ≤ one year, out of which 22 (66.7%) had an abnormal geometry. A significant relationship existed between LV geometry and duration of DM ($\chi^2 = 10.793$, $P = 0.005$).

Table 4 shows the derived echocardiographic parameters between the two groups. The RWT differed significantly between the two groups (Wilcoxon, $P < 0.001$). The RWT in 62% of the study group was ≤0.42, while 38% had >0.42 ($\chi^2 = 14.969$, $P < 0.001$). Though systolic function was normal between both groups, it was significantly lower in the normotensive type II diabetic group (t = 3.904, $P < 0.001$).

Table 5 shows the predictors of abnormal LV geometry in the study group. Obesity and DM duration of 2-4 years were significant predictors of abnormal geometry.

Table 1: Demographic and clinical characteristics of participants

Variable	Normotensive type II diabetics *n*=100	Normal controls *n*=100	Test statistics	*P*
Age of Respondents (yrs)				
31-40	10 (10.0%)	10 (10.0%)	FT	1.000
41-50	21 (21.0%)	21 (21.0%)		
51-60	35 (35.0%)	35 (35.0%)		
61-70	34 (34.0%)	34 (34.0%)		
Gender				
Male	32 (32.0%)	32 (32.0%)	FT	1.000
Female	68 (68.0%)	68 (68.0%)		
Weight (kg)				
Mean (± SD)	72.32±15.16	71.89±13.61	0.211*	0.833
Height (cm)				
Mean (±SD)	164.23±7.78	162.08±8.82	1.827*	0.069
BMI (kg/m^2)				
Mean (±SD)	26.78±5.38	28.59±9.69	1.628*	0.105
Systolic BP (mmHg)				
Mean (±SD)	115.72±11.29	119.94±10.28	2.763*	0.006
Diastolic BP (mmHg)				
Mean (±SD)	5.01±7.89	73.26±8.61	1.496*	0.136

*Student's *t*-test; FT - Fisher's exact test; BP - Blood pressure; BMI - Body mass index

Table 2: Left ventricular geometric patterns of participants

Variable	Normotensive type II diabetics *n*=100	Normal controls *n*=100	Test statistics	*P*
LV geometry				
Normal geometry	49 (49.0%)	82 (82.0%)	FT	<0.001
Concentric remodeling	36 (36.0%)	11 (11.0%)		
Concentric hypertrophy	4 (4.0%)	3 (3.0%)		
Eccentric hypertrophy	11 (11.0%)	4 (4.0%)		

LV - Left ventricle; FT - Fisher's exact test

Table 3: Relationship between left ventricular geometry and duration of diabetes

Variable	Duration of diagnosis of DM n=100			χ^2	*P*
	≤1 year	2-4 years	≥5 years		
LV geometry					
Normal geometry	11 (33.3%)	12 (85.7%)	26 (49.1%)	10.793	0.005
Abnormal geometry	22 (66.7%)	2 (14.3%)	27 (50.9%)		

χ^2 - Chi-square test; DM - Diabetes mellitus; LV - Left ventricular

Table 4: Derived Echocardiographic parameters

Variable	Normotensive type II diabetics *n*=100	Normal controls *n*=100	Test statistics	*P*
LVM (g)				
Mean (±SD)	143±41.83	137±39.44	1.003*	0.317
LVM/BSA (g/m^2)				
Mean (± SD)	79.31±21.67	76.45±19.22	0.987*	0.325
LVM/ht2.7 (g/ht 2.7)				
Mean (± SD)	31.67±11.88	37.16±9.2	0.340*	0.734
RWT				
Mean (± SD)	0.44±0.28	0.33±0.08	W†	<0.001
EF (%)				
Mean (± SD)	62.12±11.63	67.47±7.23	3.908*	<0.001

*Student's *t*-test; †Wilcoxon test; LVM - Left ventricular mass; LVM/BSA - Left ventricular mass indexed to body surface area, LVM/ht 2.7. Left ventricular mass indexed to height 2.7; RWT - Relative wall thickness; EF - Ejection fraction

Discussion

Type II DM is associated with an abnormal LV geometry in the absence of co-existing hypertension. This is congruent with previous similar studies[15-18] in Nigeria where the presence of abnormal LV geometry in persons with diabetes but without co-existing hypertension ranged between 50 and 89%. The predominant geometry noted in this study was concentric remodeling at 36%. While some studies[18-21] had a similar finding of concentric remodeling as the most predominant, some[15,17] did not, but found concentric hypertrophy as the most predominant. This pattern of concentric remodeling in our study, rather than concentric hypertrophy as seen in other studies, may be due to the lower cut off blood pressure we used to define hypertension in our study participants. We also postulate that the pattern of LV geometry is dependent on the duration of DM. Patients with type II DM tend to have hyperinsulinemia, especially in the early years of diagnosis, and this may also be contributory to the LV geometry we obtained; however, measurement of insulin or C peptide levels was not done and may be subject to future research.

A third of individuals in this study with a post DM diagnosis of ≤ one year had an abnormal LV geometry and may be in the early phase of diabetic cardiomyopathy. It further adds weight to the hypothesis that cardiac hypertrophy being a structural change resulting from yet to be elucidated pathway involving insulin signaling, a complex interplay of insulin resistance and compensatory hyperinsulinemia, may actually precede the diagnosis of DM[22] or may be subclinical. We had expected that as the duration of DM increases, so will the magnitude of myocardial structural abnormalities.[22] However, we found that only two (14.3%) of study participants had an abnormal LV geometry at two–four years post DM diagnosis but on logistic regression, patients who had DM lasting for two to four years were about six times more likely (significant) to have abnormal geometry when compared

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Table 5: Predictors of abnormal left ventricular geometry

Variable	Left ventricular geometry *n*=100		*P*[a]	AOR (95% CI)
	Abnormal (*n* %)	Normal (*n* %)		
Age				
≤56 years	23 (46.0)	27 (54.0)	0.317	NA
>56 years	28 (56.0)	22 (44.0)		
Gender				
Male	16 (50.0)	16 (50.0)	0.891	NA
Female	35 (51.5)	33 (48.5)		
Obesity				
Present	27 (43.5)	35 (56.5)	0.057	2.80 (1.12-7.00)
Absent	24 (63.7)	14 (36.8)		1
HbA1c				
Uncontrolled DM	48 (52.2)	44 (47.8)	0.426	NA
Controlled DM	3 (37.5)	5 (62.5)		
LDL-C dyslipidaemia				NA
Present	47 (50.5)	46 (49.5)	0.736	
Absent	4 (57.1)	3 (42.3)		
Duration of DM				
≤1 year	22 (66.7)	11 (33.3)	0.005	0.42 (0.16-1.08)
2-4 years	2 (14.3)	12 (85.7)		6.20 (1.22-31.42)
≥5 years	27 (50.9)	26 (49.1)		1

to those whose DM had lasted for ≥ five years. It is unclear why this percentage reduced, with progression of diabetes, before peaking again as about half of those with diabetes for more than 5 years had abnormal LV geometry. Although this can be explained by difficulty in actually ascertaining the exact duration of type II DM in most patients, further studies may be needed to establish if indeed there is a reduction in further remodeling at this intermediate stage. It is also possible that lack of awareness about diabetes and late presentation of DM patients as well as other confounding variables such as age, dyslipidemia, obesity, and ethnicity may have also contributed to this association between the LV geometry and DM duration.

There was no significant difference between the LVM or LVMI of study when compared with controls. This may be explained by the finding that abnormal myocyte hypertrophy and cardiac steatosis instead of fibrosis may have played a role in the pathogenesis of LVH in stable/early diabetic cardiomyopathy.[23] Interstitial fibrosis has been implicated in the more advanced form of diabetic cardiomyopathy.[23]

RWT allows for the identification of the spectrum of cardiac geometry in addition to the LVM and histologically demonstrates addition of sarcomeres in series (eccentric) or in parallel (concentric). A 1998 study[24] had suggested that concentric remodeling may not be associated with an increased risk of death, but Ochsner studies[25] in 2008 had demonstrated an independent link between increased RWT and reduced coronary flow reserve as well as a predictor of mortality in all populations (obesity and hypertension) studied. It further associated increased RWT to increased levels of catecholamine, aldosterone, and hepatic growth factor which have all been linked to increased cardiovascular events. Furthermore, a systematic review[26] in 2019 showed the adverse prognosis associated with LV concentric remodeling and that its mortality risk is similar to eccentric hypertrophy. A similar study[16] found a significant difference at a mean value of 0.475 ± 0.09 (study)/0.405 ± 0.07 (controls). In contrast, Ojji *et al*.[15] did not find a significant difference at a mean and SD of 0.40 ± 0.06 versus 0.39 ± 0.07 for the controls. These differences could arise from the varying values used as cutoff value. A cutoff value of 0.45 was used by Ojji *et al*.,[15] and this might explain the contrast with the index and Dodiyi-Manuel *et al*.[16] studies. Dodiyi-Manuel *et al*.[16] included patients who were on insulin therapy which may explain the slightly higher value obtained.

The controversy surrounding the best index of LVM in diabetes made the authors to compare both method of indexation in detecting LVH. This study demonstrated a higher prevalence of LVH at 21% when LVM was indexed to height$^{2.7}$ and 17% on indexation to

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BSA. There was a significant difference between the study and the control groups when LVH was indexed to height$^{2.7}$ (17% (study)/7% (control) but not to BSA (21% (study)/20% (control). In our opinion, it may be prudent to index to height in the diabetic population where majority are obese as demonstrated in this study. Whether to index to height alone or raised to allometric power of 1.7 or 2.7 becomes another controversy. Indexation to height raised to an allometric exponent of 2.7, in comparison to BSA or height alone predicted cardiovascular outcome better, detected obesity-related LVH and showed less variability of LVM among normal individuals. However, indexation to BSA is preferred, having been adopted by most imaging bodies even though, it underestimates the effect of obesity on LVM.[27,28] Chirinos *et al*.[29] demonstrated that indexation of LVM to an allometric power of 1.7 was the best method in comparison to BSA and height$^{2.7}$ to identify obesity-related LVH and was more consistently associated with CVD outcomes and all-cause mortality. The low prevalence of LVH when indexed to BSA and height raised to allometric power of 2.7 in this study could be because recruits do not have hypertension and by extension prehypertension was ruled out when most hypertension guidelines were considered. Hypertension causes left ventricular hypertrophy, and co-existence with DM may cause a greater increase in the relative wall thickness and left ventricular mass index.

To the best of our knowledge, this study is the first to evaluate the echocardiographic prevalence of left ventricular hypertrophy with a lower blood pressure of cutoff of <130/80 mmHg in diabetic patients in our locality.

The limitations of this study include inherent limitations in observational studies, the presence of confounders like age, dyslipidemia, ethnicity, and obesity. Ischemic heart disease was ruled out with clinical history and ECG which may miss out subclinical forms of coronary artery disease.

Conclusion

DM in the absence of hypertension alters the LV geometry which worsens the already existing cardiovascular risk established by the presence of the disease alone. Echocardiography should be considered earlier in the management of DM patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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