Annals of African Medicine Vol. 11, No. 2; 2012

Original Article

Access this article online

Quick Response Code:

Website: www.annalsafrmed.org

DOI: 10.4103/1596-3519.93530

Left ventricular structure and function in black normotensive type 2 diabetes mellitus patients

Igben F. Aigbe, Philip M. Kolo, Ayodele B. Omotoso

Department of Medicine, University of Ilorin Teaching Hospital, Ilorin, Nigeria

Correspondence to: Dr. I. F. Aigbe, Department of Medicine, University of Ilorin Teaching Hospital, P.M.B. 1459, Ilorin, Nigeria. E-mail: igbenf@yahoo.com

Abstract

Background: Relationship between type 2 DM and cardiovascular disease (CVD) is well known, with CVD being the most common cause of mortality in diabetics. Significant myocardial injury before overt CVD in DM can be identified early using echocardiography. This study therefore aimed at evaluating left ventricular structure and function of patients with type 2 DM.

Materials and Methods: One hundred and fifty adult type 2 DM patients were recruited with 150 age- and sex-matched controls. Patients and subjects with systemic hypertension, pregnancy, sickle cell disease and structural heart disease were excluded from the study. Participants were evaluated clinically; had anthropometric parameters and electrocardiogram taken. Echocardiograms were obtained according to the American Society of Echocardiography (ASE) recommendations.

Results: Mean age of the patients (55.4±11.6 years) was similar to that of the control (54.2±9.6 years) (*P*=0.348) and the duration of DM was 4.53 years. Left ventricular (LV) systolic function was normal in both groups but was higher in patients than controls (ejection fraction=70.3±10.7% and 64.4 ± 9.4%, *P* =0.001 respectively). The prevalence of LV diastolic dysfunction (LVDD) was 72% in the patients compared with 6% in controls (*P*=0.001). Patients' age, body weight, duration of DM, LV mass index and left atrial dimension were positive correlates of LVDD while patients' age, weight and left atrial dimension were independent predictors of LVDD.

Conclusion: There is high prevalence of alterations in LV structure and function in normotensive type 2 DM; and there is a need for early intervention to prevent overt LV dysfunction.

Keywords: Black normotensive patients, left ventricular function, type 2 DM

Résumé

Fond: Relation entre tapez 2 DM et de maladies cardiovasculaires (MCV) sont bien connu, avec les maladies cardiovasculaires sont la cause la plus fréquente de mortalité chez les diabétiques. Lésion myocardique significative avant CVD manifeste en DM peut être identifiée au début à l'aide d'échocardiographie. Cette étude donc vise à évaluer à gauche ventriculaire structure et la fonction des patients avec type DM 2.

Matériaux et procédés: Les patients DM 2 de type adulte à cent cinquante ont été recrutés avec 150 contrôles appariés selon l'âge et le sexe. Les patients et les sujets atteints d'hypertension systémique, grossesse, drépanocytose et structurelle de maladie cardiaque étaient exclus de l'étude. Les participants ont été évalués sur le plan clinique; paramètres anthropométriques et l'électrocardiogramme avaient pris. Échocardiogrammes ont été obtenus selon la société américaine d'échocardiographie (ASE) recommandations.

Résultats: Moyen âge des patients (55.4±11.6 ans) était semblable à celle du contrôle (54.2±9.6 ans) (*P*= 0.348) et la durée de DM était 4,53 ans. Gauche fonction systolique ventriculaire (LV) était normale dans les deux groupes, mais est plus élevée chez les patients que les contrôles (fraction=70.3±10.7% d'éjection et 64,4 ± 9,4%, *P* = 0,001 respectivement). La prévalence de la dysfonction diastolique LV (LVDD) était de 72% chez les patients comparés à 6% chez les témoins (*P*= 0,001). L'âge des patients, poids corporel, durée de DM, indice de masse LV et dimension auriculaire gauche étaient positifs corrélats de LVDD alors que l'âge, de poids et de dimension auriculaire gauche

des patients étaient des prédicteurs indépendants de LVDD.
Conclusion: Il y a forte prévalence de la fonction et des altérations dans la structure de LV dans normotensif type 2 DM; et il existe un besoin pour une intervention précoce à empêcher les manifestes LV dysfonctionnement.

Mots-clés: Les patients normotendus noirs, quitté la fonction ventriculaire, tapez 2 DM

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder resulting from defects in insulin secretion, action or both leading to inefficient metabolism of sugars and other substrates by the body tissues; causing both acute and chronic life-threatening complications. Presently, about 2.8% of the world population (about 177 million people) is afflicted by DM and it is expected to almost double by 2030.[1,2] In Nigeria, the prevalence of type 2 DM was about 2.2% in 1997.[3]

Among many complications of DM, cardiovascular involvement, frequently manifesting as peripheral vascular disease, coronary artery disease (CAD), systemic hypertension, congestive heart failure and stroke, is associated with worse clinical outcome.[4] However, prior to, or simultaneously with these overt presentations, more subtle and insidious myocardial damage may be ongoing which become manifest only when significant myocardial injury has occurred. Previous studies in DM patients which assessed cardiac function were predominantly done in those with systemic hypertension.[5,6] A combination of DM and systemic hypertension is known to be associated with poor health and premature death.[7] This study aimed at evaluating left ventricular structure and function of individuals with type 2 DM who are free of systemic hypertension using non-invasive imaging modality.

Materials and Methods

This cross-sectional study was carried-out at a University Teaching Hospital in Nigeria. One hundred and fifty adult type 2 DM patients were randomly selected with 150 age- and sex-matched normal non-diabetic subjects. Type 2 DM patients and subjects with systemic hypertension, pregnancy, sickle cell disease and structural heart disease were excluded from the study. All the study participants were evaluated clinically and anthropometric parameters such as height (m), weight (kg), waist and hip circumference (cm); body mass index (BMI) and waist-hip ratio (WHR) were assessed. The following laboratory investigations were done:; fasting serum lipid profile, fasting plasma (venous) glucose, glycosylated hemoglobin (HbA1c), serum electrolytes, urea and creatinine. Electrocardiogram (ECG) was also done in all the subjects.

Esaote Megas CVX Echocardiography machine which has facility for two-dimensional, m-mode, continuous wave, pulsed wave and color Doppler was used to assess the heart according to the American Society of Echocardiography (ASE) recommendations.[8] Indices measured included÷ left ventricular internal dimension in diastole (LVIDd) and in systole (LVIDs), interventricular septum in diastole (IVSd) and systole (IVSs) and the posterior wall dimension in diastole (PWd) and systole (PWs). Others were aortic (AOD) and left atrial dimension (LAD). LV ejection fraction (LVEF), fractional shortening (FS) and left ventricular mass index (LVMI) were derived from the earlier measurements. The LVMI, EF and FS were determined as follows:

$$LVMI = \frac{1.04(IVSTd + LVIDd + PWTd)^3 - LVIDd^3}{BSA}$$

where IVSd - interventricular septal thickness in diastole
LVDd - left ventricular wall dimension in diastole
PWTd - posterior wall thickness in diastole
BSA - body surface area
Relative wall thickness (RWT) was calculated by the formula:[9] $2 \times PWd/LVIDd$.
The pattern of LV remodeling was determined using LVMI and RWT.
Increased RWT was present if RWT was ≥0.45[9]
LV geometric pattern was classified using RWT and LVMI as follows:
Normal geometry = normal LVMI and RWT
Concentric remodeling= normal LVMI and RWT ≥0.45
Eccentric LV hypertrophy= increased LVMI and RWT <0.45
Concentric LV hypertrophy= increased LVMI and RWT ≥0.45

$$EF = \frac{LVIDd^3 - LVIDs^3}{LVIDd^3} \times 100$$

$$FS = \frac{LVIDd - LVIDs}{LVIDd} \times 100$$

EF=Ejection fraction
FS=Fractional shortening
LVIDd=Left ventricular internal dimension in diastole
LVIDs=Left ventricular internal dimension in systole
The left ventricular diastolic function was assessed

using Doppler modalities. The Doppler variables measured included peak velocity of early mitral filling (E-wave), peak velocity of atrial contraction (A-wave), and E/A ratio. Others included isovolumic relaxation time (IVRT) and the deceleration time (DT). Pulmonary venous flow (PVF) velocity recordings, which included peak systolic (S), diastolic (D) flow velocities ratio of S/D and the peak atrial reversal (Ar) were obtained.

Systolic function was considered as normal if LVEF>50% and FS of greater than 25%.

Diastolic function was categorized according to its progression into grades:

(i) Normal diastolic function was taken as E/A between 1 and 2, IVRT of between 80 and 110 ms and DT of between 150 and 240 ms.
(ii) Impaired relaxation, the E/A should be <I, IVRT >110 ms, DT >240 ms,
(iii) Pseudonormalization, E/A between 1 and 2, IVRT 80-110 ms, DT 150-220 ms and the PVF S/D <1.
(iv) Restrictive pattern, the E/A is> 2, and DT <150 ms.[10]

Data Analysis

Data obtained was analyzed using statistical package for social sciences (SPSS 15) computer software. Data were expressed as mean ± standard deviation (SD) and frequencies were expressed as percentages. Means of proportion were compared using the Chi-square while Student's t-test was used for continuous variables. Correlates of LV function were determined using the Pearson's rank correlation and predictors were assessed using multiple regressions. A *P*-value of less than or equal to 0.05 was considered as statistically significant.

Results

0ne hundred and fifty type 2 DM patients consisting of 65 males and 85 females were studied. Their age range was from 26 to 80 years. The mean age of the patients (55.4±11.6 years) was similar to that of the control subjects (54.2±9.6 years) (P=0.348) and mean duration of DM in the patients was 4.53 years.

Table 1 displays the anthropometric characteristics of the study group. Mean BMI, weight, and height were significantly (P=0.001, P=0.002, P=0.001) higher in the control subjects than the type 2 DM patients. However, the WHR and SBP were higher in the diabetic subjects (P=0.001, P=0.001). The patients' FBS and HbA1c were also significantly higher than controls (P=0.01 and P=0.01) [Table 1].

Significant higher proportion of patients (49%) had abnormal ECG pattern compared with 30% of the controls (P=0.001). The prevalence of LVH was significantly (P=0.003) higher in the former than the latter. More diabetic patients had left atrial enlargement (LAE) and left bundle branch block (LBBB) than controls (P=0.006 and 0.006 respectively). Abnormalities of ST segment and Q-wave suggestive of myocardial ischemia were significantly higher (P=0.005) in diabetic cohort than the control. About 17% of diabetics and 5.5% 0f controls had LVH in combination with other abnormalities, such as complete LBBB, PVCs and LAE [Table 2].

Table 1: Anthropometric and glycemic indices of patients and control

Variable	Patients (mean ± SD) *n* = 150	Control (mean ± SD) *n* = 150	*P*
Age (years)	55.4±11.6	54.2±9.6	0.348
Dur of DM (years)	4.53±4.54	-	-
Sex: Male	65 (43.3%)	65 (43.3%)	0.908
Female	85 (56.7%)	85 (56.7%)	0.908
BMI (kg/m^2)	25.2±5.2	27.4±3.9	0.001
WHR	0.99±.07	0.90±.05	0.001
SBP (mmHg)	119.6±10.56	115.0 ±10.68	0.001
DBP (mmHg)	74.1±6.87	73.5±8.04	0.479
Weight (kg)	67.7 ± 14.97	73.07± 15.22	0.002
Height (m)	1.63 ± .08	1.68±.07	0.001
HbA1c	5.96±2.1	4.0±1.1	0.010
FBS	8.80±4.1	4.2±1.2	0.010

Dur of DM – duration of DM, BMI-body mass index, SBP- systolic blood pressure, DBP- diastolic blood pressure, WHR - waist/hip ratio, FBS- fasting blood sugar, HbA1c – glycated hemoglobin.

Table 2: Electrocardiographic (ECG) findings in patients and controls

ECG pattern	Patients (%) n=150	Control (%) n=150	*P*
Normal	51	70	0.001
LVH	19	14	0.032
RBBB	1.3	1.2	0.736
LAE	4.0	2.7	0.006
PVC	2.0	2.0	0.860
PAC	3.3	3.0	0.564
LBBB	2.6	1.3	0.006
LVH/RBB	0.7	0.7	0.867
LVH/LAE	5.2	1.6	0.035
LVH/PVC	4.6	2	0.046
ST-Q wave	5.3	1.0	0.005
LVH/LBBB	1.0	0.5	0.043
LAE/PVC	0.7	0	0.001

LVH- left ventricular hypertrophy, RBBB-right bundle branch block, LAE- left atrial enlargement, PVC- premature ventricular contraction, PAC-premature atrial contraction, LBBB-left bundle branch block, ST-Q - ST-Q wave abnormality.

The left ventricular geometric patterns are as shown in Table 3. Abnormal LV geometric patterns (concentric LVH and eccentric LVH) were commoner in the diabetics than the controls (P=0.001)

The left ventricular dimensions (LVIDd, LVIDs) were significantly (P=0.005, P=0.001 respectively) higher in the controls than the patients. However, the diabetic cohort showed significantly higher LVMI (139.20±40.20 vs 96.46±15.78, P=0.001) [Table 4].

Comparison of parameters of LV systolic function (ejection fraction and fractional shortening) between patients and control revealed that these parameters were within normal range for both groups but were significantly better in patients than controls (EF% 70.3±10.69 *vs* 64.4 ± 9.36, P =0.001; FS% 41.1±10.45 *vs* 33.22±5.45, P= 0.001 respectively) [Table 4].

The indices of LV diastolic function showed that the mitral EA ratio was significantly lower in diabetic cohort as compared with controls (0.92±0.38 *vs* 1.32±0.27 P=0.001). Similarly, the DT was lower (P=0.001) in diabetic than controls. However, the IVRT was higher in the diabetic patients (P=0.001) than the controls. The PVF parameters did not show any statistically significant differences between patients and control. However, the diabetics had lower PVF S/D wave ratio [Table 5].

Table 6 displays patterns and prevalence of various forms of LV diastolic dysfunction in study subjects. Majority (72%) of the patients had LVDD while 6% of controls had LVDD (P=0.001). Sixty five percent of diabetic cohort had impaired form of LV diastolic dysfunction against 6% of control (P=0.001). On the other hand, 4% of the patients had pseudonormalization type of LV diastolic dysfunction while 3% had restrictive pattern of LV diastolic dysfunction but none of the control subjects had the latter two forms of LVDD.

Correlation of severity (grades 1-4) of LVDD with patients' clinical, laboratory and echocardiographic parameters showed significantly positive association with age, weight, LAD and LVMI [Table 7]. However, no correlation was observed between LVDD and duration of DM, BMI, WHR, HbA1c, FBS, DBP and SBP.

Using multiple regression analysis to define determinants of LV diastolic function revealed that, patients' age, LAD and body weight were

Table 3: Left ventricular geometric configuration of patients and control

LV configuration	Patients (%) n=150	Control (%) n=150	*P* value
Normal LV geometry	11	73	0.001
Concentric LVH	55	0	0.001
Concentric LV remodeling	29	270.785	
Eccentric LVH	5	0	0.001

LV- left ventricle, LVH -left ventricular hypertrophy.

Table 5: Echocardiographic left ventricular diastolic function parameters of patients and controls

Variable	Patients mean ±SD *n* = 150	Control mean ±SD *n* = 150	*P* value
EDT (ms)	154.6±66.47	177.13±40.53	0.001
IVRT (ms)	138.54±52.61	84.10 ±15.43	0.001
E/A RATIO	0.92±0.38	1.32± 0.27	0.001
PVF S wave (m/s)	37.96±9.15	41.10±11.61	0.355
PVF D wave (m/s)	33.93±9.55	34.75±9.55	0.777
PVF S/D wave	1.16±0.28	1.25 ±0.35	0.295
PVF Ar wave (m/s)	22.13±5.61	24.03 ±5.57	0.191

IVRT-isovolumic relaxation time, EDT-deceleration time, E/A ratio-early/atrial filling ratio, Ar-atrial reversal, PVF-pulmonary venous flow, S/D-systolic and diastolic wave ratio. Plus-minus values are means ± SD. P< 0.05 is significant

Table 4: Echocardiographic parameters of left ventricular systolic function of patients and controls

Variable	Patients' mean ±SD *n* = 150	Control mean ±SD *n* = 150	*P* value
IVSDd (cm)	1.25±0.35	1.00± 0.20	0.001
LVIDd (cm)	4.35 ± 0.70	4.56 ± 0.52	0.005
PWDd (cm)	1.25 ±0.40	0.93 ± 0.14	0.001
IVSDs (cm)	1.86± 0.45	1.33 ±0.29	0.001
LVIDs (cm)	2.60 ±0.62	3.07±0.49	0.001
PWDs (cm)	1.83±0.39	1.24±0.22	0.001
RWT (cm)	0.62 ±0.48	0.41± 0.09	0.001
EF%	70.3± 10.69	64.4 ± 9.36	0.001
FS%	41.1±10.45	33.22± 5.45	0.001
LVMI (g/m^2)	139.20±40.20	96.46± 1578	0.001

IVSDd-interventricular septal thickness in diastole, LVIDd-left ventricular dimension in diastole-, PWDd-posterior wall thickness in diastole, IVSDd-interventricular septal thickness in systole, LVIDs- left ventricular dimension in systole, PWDd-posterior wall thickness in systole, EF-ejection fraction, FS-fractional shortening, LAD-left atrial dimension, RWT-relative wall thickness. Plus-minus values are means±SD. P< 0.05 is significant.

Table 6: Pattern and prevalence of grades of left ventricular diastolic dysfunction among patients and controls

LV diastolic function	Patients n=150 %	Controls n=150 %	*P* value
Normal	27.81	94.0	0.001
Impaired relaxation	64.90	6.0	0.001
Pseudonormalization	3.97	-	-
Restrictive pattern	3.31	-	-
Total	100	100	

Table 7: Correlation of severity of left ventricular diastolic dysfunction with clinical parameters

Parameters	Correlation coefficient	*P* value
Age (years)	0.399	0.001*
Dur of DM (years)	0.090	0.273
Weight (kg)	0.185	0.023*
BMI (g/m^2)	-0.120	0.141
WHR	0.053	0.521
HbA1c%	0.077	0.600
LAD (cm)	0.247	0.002*
FBS (mmol/l)	0.119	0.146
SBP (mmHg)	0.015	0.853
DBP (mmHg)	-0.060	0.467
LVMI (g/m^2)	0.273	0.003*

BMI-body mass index, Dur of DM –duration of DM, SBP-systolic BP, DBP- diastolic BP, WHR-waist/hip ratio, HbA1c-glycated hemoglobin, FBS –fasting blood sugar, LAD- left atrial dimension. *Statistically significant.

independent predictors of LV diastolic function [Table 8]. However, LVMI was not.

Discussion

The main finding of this study is high prevalence of LVDD in black normotensive type 2 DM patients. The study showed that 72% of the patients evaluated had one form of LVDD or the other. Similar studies in USA involving both White and Black type 2 DM individuals reported comparable prevalence of LVDD.[11,12] In the same vein, Osunkwo and Okeahialam had found high prevalence of LVDD in DM patients seen in Jos, Nigeria.[13] However, Danbauchi *et al.,* and Masugata *et al.,* did not observe any significant difference in the prevalence of diastolic dysfunction between diabetic and control subjects in Zaria, Nigeria and Japan respectively.[4,14] The differences in reported prevalence may be due to the use of different instruments and parameters in assessing diastolic function.

Although, the results of the study showed normal LV systolic function in both patient and the control groups, there was an enhancement of LV systolic function in the former. This may be due to the increase in LV wall thickness and mass compared to healthy control,[15] as suggested by Thuesen *et al*. However, Osunkwo and Okeahialam; and other related works did not observe any significant difference in LV systolic function in patients and controls; they hypothesized that, signs of diastolic abnormalities could appear much earlier than systolic abnormalities in diabetics or that they do not live long enough to develop overt LV systolic dysfunction.[13,16,17] But, Marwick[18] argued that systolic dysfunction has been more difficult to find in human studies because of the low sensitivity of standard parameters used to assess LV systolic

Table 8: Predictors of left ventricular diastolic function using multiple (stepwise linear) regressions analysis

Model	Variable	R	R2	*P*
1	Age (years)	0.531(a)	0.282	0.000*
2	Age (years)	0.594(b)	0.353	0.000*
	LAD (cm)			0.030*
3	Age (years)			0.002*
	LAD (cm)	0.664(c)	0.441	0.002*
	Weight (Kg)			0.011

LAD = left atrial dimension, (a) Predictors: (constant), age, (b) Predictors: (constant), age, lad, (c) Predictors: (constant), age, lad, weight

function (for example, ejection fraction). Recently, more sensitive indices of long axis function such as tissue Doppler echocardiographic imaging have provided evidence of disturbances of LV systolic function in diabetes, initially compensated by preservation of radial function.[19] On the other hand, some studies had found lower ejection fraction among type 2 DM patients than normal control.[20-22] The diabetic subjects had higher left ventricular mass index (LVMI) than controls in this study. Danbauchi *et al.,*[4] and Liu *et al.,*[23] made similar observation. The LVMI in the diabetic patients was also positively associated with the grade of diastolic dysfunction in this study. This was also observed by Zabalgoitia *et al*.[11] This may be due to increased apoptosis and necrosis which have been identified in diabetic heart disease, causing increased deposition of collagen in diffuse manner as a result of replacement fibrosis and connective tissue cell proliferation.[24] This ultimately results in increased LV mass and consequently decreased ventricular compliance. However, some studies did not find significant difference in LVMI[13] and relative wall thickness[14] between diabetics and normal controls. Danbauchi *et al.,* reported a positive correlation between LVMI, diastolic and systolic blood pressure which was not seen in our study.[4]

Characterization of LV geometric pattern further refines cardiovascular risk associated with LVH. Diabetics, unlike the control, in the present study had predominantly concentric LVH geometric pattern. This is the pattern that is associated with more severe hemodynamic and structural abnormalities and consequently the most adverse cardiovascular (CV) risk.[25] LVDD correlated positively, on simple analysis with patient's age, LAD and weight in this study. On multiple regression analysis, this relationship was sustained with the patient's age, LAD and weight independently predicting LV diastolic function. However, LVMI did not independently predict grades of LV diastolic dysfunction. These are similar to the findings of other studies; Masugata *et al*.,[14] found LV diastolic

function to be inversely correlated with aging and the duration of type 2 diabetes. Danbauchi *et al.,* found significant correlation between diastolic dysfunction and age, fasting blood glucose, and two-hour postprandial glucose.[4] Ageing understandably causes increased atherosclerosis affecting both the large arteries and the resistant vessels leading to LV hypertrophy and decrease compliance. In contrast, Attali *et al.*, in a study of 49 diabetic patients without known heart disease, reported that LVDD was unrelated to sex, age, duration of diabetes, or the presence of complications.[26]

This study showed that more diabetic patients (49%) had abnormal ECG patterns as compared with the control (30%). The resting ECG of the diabetic patients and controls revealed higher incidence of LVH, LAE, LBBB and ST wave and Q wave changes in the diabetics than controls. This is in agreement with reports from Kaduna, Northern Nigeria which found 20% of diabetics and 1.5% of controls to have ST-T abnormalities.[27] Earlier studies especially among diabetic Caucasians revealed high prevalence of asymptomatic abnormal ST segment depression which was thought to be due to autonomic neuropathy.[28]

Fewer patients (5%) in this study had ST segment changes and Q-wave abnormalities but the prevalence was higher in the diabetic group than control. This may be due to the low incidence of CAD in sub-Saharan Africans even among diabetics. In this regard, a review of ECG of diabetic Asian migrants in the UK showed ST-T wave abnormalities to be the most common finding[29] and ST-T wave abnormalities on ECG at rest have been shown to strongly correlate with silent ischemia.[30] LVH is the most frequent ECG abnormality, present in about 34% of diabetics which is similar to the reports of other studies.[16] However Bello-Sani and Anumah reported that only 7% of their diabetic cohort had LVH.[27]

In conclusion, there is a high prevalence of LVDD in normotensive type 2 DM patients in our environment and this suggests cardiac involvement even in asymptomatic individuals. We suggest that cardiac assessment using non-invasive imaging modality should be a part of routine evaluation of type 2 DM patients. Early lifestyle modifications to achieve normal body weight in this group of patients will prevent or delay onset of overt cardiovascular disease.

References

1. Diabetes Action Now: An Initiative of the World Health Organization (WHO) and International Diabetes Federation. A WHO Publication; 2004. p. 4.
2. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes estimates for the year 2000 and projections for 2030. Diabetes Care 2004;27:1047-53.
3. Akinkugbe OO, editor. Non-communicable Disease in Nigeria: Final Report of National Survey. Lagos: Federal Ministry of Health and Social Service; 1997. p. 64-90.
4. Danbauchi SS, Anumah FE, Alhassan MA, David SO, Onyemelukwe GC, Oyati IA. Left ventricular function in type 2 diabetes patients without cardiac symptoms in Zaria, Nigeria. Ethn Dis 2005;15:635-40.
5. Palmieri V, Bella JN, Arnett DK, Liu JE, Oberman A, Schuck MY, *et al*. Effect of type 2 diabetes mellitus on left ventricular geometry and systolic function in hypertensive subjects: Hypertension Genetic Epidemiology Network (HyperGEN) Study. Circulation 2001;103:102-7.
6. Hildebrandt P, Wachtell K, Dahlöf B, Papademitriou V, Gerdts E, Giles T, *et al*. Impairment of cardiac function in hypertensive patients with Type 2 diabetes: A LIFE study. Diabet Med 2005;22:1005-11.
7. UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macro vascular and micro vascular complications in type 2 diabetes (UKPDS 38). BMJ 1998;317:705-13.
8. Henry WL, DeMaria A, Gramiak R, King DL, Kisslo JA, Popp RL, *et al*. Report of the American Society of Echocardiography Nomenclature and Standards in two-dimensional Echocardiography. Circulation 1980;62:212-5.
9. Savage DD, Garrison RJ, Kannel WB, Levy D, Anderson SJ, Stokes J, *et al*. The spectrum of left ventricular hypertrophy in a general population sample: The Framingham Study. Circulation 1987;75:126-33.
10. Yamamoto K, Redfield MM, Nishimura RA. Analysis of left ventricular diastolic function. Heart 1996;75:27-35.
11. Zabalgoitia M. Prevalence of diastolic dysfunction in normotensive, asymptomatic patients with well-controlled type 2 diabetes mellitus. Am J Cardiol 2001;87:320-3.
12. Boyer JK, Thanigaraj S, Schechtman KB, Pérez JE. Prevalence of ventricular diastolic dysfunction in asymptomatic, normotensive patients with diabetes mellitus. Am J Cardiol 2004;93:870-5.
13. Osunkwo DA, Okeahialam BN. Left ventricular function in Nigerians with non–insulin-dependent diabetes mellitus. Am J Cardiol 2001;87:1026-8.
14. Masugata H, Senda S, Goda F, Yoshihara Y, Yoshikawa K, Fujita N, *et al*. Left ventricular diastolic dysfunction in normotensive diabetic patients in various age strata. Diabetes Res Clin Pract 2008;79:91-6.
15. Thuesen L, Christiansen JS, Mogensen CE, Henningsen P. Cardiac hyperfunction in insulin dependent diabetic patients developing microvascular complications. Diabetes 1988;37:851-6.
16. Shehadeh A, Regan TJ. Cardiac consequences of diabetes mellitus. Clin Cardiol 1995;18:301-5.
17. Fiorini G, Scotti LA, Parmigiani ML, Ferrari M, Pezzoli P, Bignotti G. An echocardiographic study of left ventricular diastolic function in patients with diabetes mellitus type. G Ital Cardiol (Rome) 1995;25:17-25.
18. Marwick TH. Diabetic heart disease. Postgrad Med J 2008;84:188-92.
19. Andersen NH, Poulsen SH. Evaluation of the longitudinal contraction of the left ventricle in normal subjects by Doppler tissue tracking and strain rate. J Am Soc Echocardiogr 2003;16:716-23.
20. Annonu AK, Mokhtar MS, Ghareeb S, Elhendy A. Left

ventricular systolic and diastolic functional abnormalities in asymptomatic patients with non-insulin-dependent diabetes mellitus. J Am Soc Echocardiogr 2001;14: 885-91.
21. Bertoni AG, Hundley WG, Massing MW, Bonds DE, Burke GL, Goff DC Jr. Heart failure prevalence, incidence, and mortality in the elderly with diabetes. Diabetes Care 2004;27:699-703.
22. Fang ZY, Schull-Meade R, Downey M, Prins J, Marwick TH. Deteminants of subclinical diabetic heart disease. Diabetologia 2005;48:394-402.
23. Liu JE, Palmieri V, Roman MJ, Bella JN, Fabsitz R, Howard BV, *et al*. The impact of diabetes on left ventricular filling pattern in normotensive and hypertensive adults: The Strong Heart Study. J Am Coll Cardiol 2001;37:1943-9.
24. Weber KT, Brilla CG. Pathological hypertrophy and cardiac interstitium. Fibrosis and renin-angiotensin-aldosterone system. Circulation 1991;83:1849-65.
25. De Simone G. Left ventricular geometry and hypotension in end-stage renal disease: A mechanical perspective. J Am Soc Nephrol 2003;14:2421-7.
26. Attali JR, Sachs RN, Valensi P, Palsky D, Tellier P, Vulpillat M, *et al*. Asymptomatic diabetic cardiomyopathy: A noninvasive study. Diabetes Res Clin Pract 1988;4: 183-90.
27. Bello-Sani F, Anumah FO. Electrocardiographic abnormalities in persons with type 2 diabetes in Kaduna, Northern Nigeria. Abstract of the America Association of Clinical Endocrinologist, 17th Annual Meeting and Clinical Congress; 2008. p. 16-7.
28. Reissell E, Yli-Hankala A, Orko R, Lindgren L. Sudden Cardiorespiratory arrest with diabetic autonomic neuropathy and prolonged QT interval. Acta Anaesthesiol Scand 1994;38:406-8.
29. Zargar AH, Sofi FA, Laway BA, Masoodi SR, Shah NA, Dar FA. Profile of neurological problems in diabetes mellitus retrospective analysis of data from 1294 patients. Ann Saudi Med 1997;17:20-5.
30. Milan study on atherosclerosis and diabetes (MISAD) group. Prevalence of unrecognised silent myocardial ischemia and its association with atherosclerotic risk factors in non-insulin dependent diabetes mellitus. Am J Cardiol 1997;79:134-9.

Cite this article as: Aigbe IF, Kolo PM, Omotoso AB. Left ventricular structure and function in black normotensive type 2 diabetes mellitus patients. Ann Afr Med 2012;11:84-90.
Source of Support: Nil, **Conflict of Interest:** None declared.