IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 16, Issue 9 Ver. XI (September. 2017), PP 64-70
www.iosrjournals.org

The Predictors of Left Ventricular Dysfunction Among Hypertensive And Diabetic - Hypertensive Patients in A Tertiary Hospital in Nigeria

*Vincent Maduka Uhegbu[1,2], Victor Okon Ansa[1, 2], Henry Ohem Okpa[1, 3], Ofem Egbe Enang[1, 4]Clement Osita Odigwe[1, 2]

[1]*Department of Internal Medicine, College of Medical Sciences, University of Calabar, Nigeria*
[2] *Cardiology Unit, Department of Internal Medicine, University of Calabar Teaching Hospital, Calabar, Nigeria*
[3]*Renal Unit, Department of Internal Medicine, University of Calabar Teaching Hospital, Calabar, Nigeria*
[4]*Endocrine and Metabolism Unit, Department of Internal Medicine, University of Calabar Teaching Hospital, Calabar, Nigeria*
Corresponding Author: Vincent Maduka Uhegbu

Abstract: *There is currently an ongoing global epidemiological transition of disease patterns from communicable diseases (CD) to non communicable diseases (NCD), and Sub – Saharan Africa is not spared. Majority of NCD related deaths occur in low and middle income countries such as Nigeria. Diabetes mellitus and systemic hypertension represent the greatest burden of the NCDs in the African region, often occurring together with common risk factors. To determine the predictors of left ventricular diastolic function in type 2 diabetic patients with or without hypertension at the University of Calabar Teaching Hospital. This was a cross sectional study carried out at the cardiac and endocrinology clinics of the University of Calabar Teaching Hospital (UCTH) for a period of 6 months from Jan to July 2013. Data was analysed using SPSS version 15. One hundred and four (56.5%) of the participants were males while 80 (43.5%) were females. The study showed that the prevalence of Left ventricular diastolic dysfunction (LVDD) is 63.0% and 67.4% in normotensive diabetic and hypertensive patients respectively, with a higher prevalence of 76.1% in hypertensive – diabetic patients. The predictors of LVDD were older age, higher waist-hip ratio, diastolic blood pressure, fasting blood sugar and glycated hemoglobin. In conclusion, the combination of hypertension and diabetes mellitus has a greater effect on LVDD.*

Keywords*: Diabetic, Hypertensive, Left ventricular dysfunction, Predictors*

Date of Submission: 01 -09-2017 Date of acceptance: 30-09-2017

I. Introduction

There is an ongoing global epidemiological transition of disease patterns from communicable diseases (CD) to non communicable diseases (NCD). According to the World Health Organization (WHO), non communicable diseases cause about 38 million deaths annually, with 80% of these deaths occurring in low and middle income countries [1]. Diabetes mellitus and systemic hypertension represent the greatest burden of the NCDs in the African region, often occurring together with common risk factors. Dhakshinamurthy et al in their study of risk stratification and predictors of cardiovascular events in type 2 diabetes mellitus reported the following risk factors including sex (male), age, obesity, alcohol, smoking, stress, dyslipidemia and hypertension [2].Diabetes mellitus and hypertension had the highest cardiovascular morbidity and mortality. Diabetes mellitus (DM) is an endocrinopathy resulting in chronic hyperglycemia due to relative or absolute deficiency of insulin production and or secretion, decreased peripheral utilization of insulin (insulin resistance) and increased hepatic production of glucose [3, 4, 5, 6, 7].The International Diabetes Federation (IDF) reported a global prevalence of diabetes with or without hypertension to be 8.3% in 2011 and projected an increase of 9.9% by 2030 [8].Prolonged elevation of blood pressure (BP), which often occurs with type 2 diabetes mellitus leads to accelerated changes in the myocardial structure, premature coronary artery disease, conduction system disease, diastolic and systolic dysfunction of the left ventricle, and these often lead to congestive cardiac failure (CCF).The Framingham study was the first to demonstrate an increased risk of heart failure in diabetic patients, while Russo et al reported the independent and combined effects of diabetes and hypertension on diastolic function, independent of the effects of over-weight and obesity [9, 10]. In Nigeria, Danbauchi et al in Zaria in their study showed that hypertensive diabetic patients had higher cholesterol level, significantly lower ejection fractions and diastolic dysfunction as well as higher left ventricular mass (LVM) than controls [11]. The LVM

correlated significantly with diastolic blood pressure, systolic blood pressure, and pulse pressure. They concluded that the coexistence of hypertension in diabetes increases the risk of developing diabetic cardiomyopathy [11].

Ogah et al in their study at Ibadan on left ventricular diastolic function in normotensive type 2 diabetic subjects found a strong association between left ventricular diastolic function and type 2 diabetes mellitus independent of obesity and hypertension [12]. Harry in Enugu reported a similar finding in his study [13]. The aim of this study therefore is to determine the predictors of left ventricular diastolic function in type 2 diabetic patients with or without hypertension at the University of Calabar Teaching Hospital.

II. Methodology

This was a cross sectional study carried out at the cardiac and endocrinology clinics of the University of Calabar Teaching Hospital (UCTH) for a period of 6 months from Jan to July 2013. Adult aged 18 years and above with type 2 diabetes mellitus with or without hypertension presenting to the medical out patients' clinic of the hospital and aged matched control were recruited for the study. Ethical approval for the study was obtained from the Ethics Committee of the UCTH.

A total of 184 patients who met the inclusion criteria with complete data were included as follows:

- 46 Normotensive-diabetic patients (D.M)
- 46 Hypertensive patients (HTN)
- 46 Diabetic-hypertensive patients (DM/HTN)
- 46 Control subject (CS)

2.1. Assessment Of Clinical Parameters

Blood pressure was measured using Accouson Mercury sphygmomanometer to determine the brachial artery systolic and diastolic blood pressures using the first and fifth korotkoff sounds respectively. Each patient was seated for at least 5 minutes in a chair with arm supported at heart level and feet on the floor. An appropriate cuff size (cuff bladder encircling 70 to 80% of the arm) was used. Both the palpation and auscultatory methods were used. The cuff was deflated during auscultation at the rate of 2mmHg per second to determine diastolic pressure, at least two measurements were taken and the average recorded and this is done at least 5 minutes apart. Blood pressure of $\geq$ 140/90mmhg was taken as hypertension using JNCVII definition [14]. Exercise, smoking, caffeine were avoided at least 30 minutes prior to BP measurement [15].

Body mass index (BMI) was calculated by dividing weight in kilograms by the square of the height in meter (kg/m^2). Height was measured using a height-in-metre instrument with the subject standing feet together without shoes or headgear, back and heel together against a vertical ruled bar to which a movable attached horizontal bar is brought to the vertex of the patients head and reading taken to the nearest 0.5 centimetre. Weight was taken using a weighing scale with subject wearing only light clothing. The scale was be standardized against a fixed weight at every ten readings to the nearest kilogram (0.1kg). Body mass index of $\geq$ $30kg/m^2$ was taken as obesity using WHO classification 1997 [16].

Waist circumference was taken at the part of the trunk located midway between the costal margin and the iliac crest while the subject stood with feet about 25-30cm apart.The tape was fitted snugly without compressing underlying soft tissue and circumference was measured to the nearest 0.5cm at the end of a normal expiration [17]. Hip circumference was taken at the level of the greater trochanters'. The waist to hip ratio was also calculated. Waist hip ratio $\geq$ 0.90 for men and 0.85 for female were taken as obesity using WHO classification 1997 [17].

2.2. Blood Chemistry

Fasting blood sugar was done with fasting blood sample from the subjects at least eight hours after the last meal, and this was done using the glucose oxidase method. Fasting lipid profile was done using the esterase method.

Serum electrolyte was obtained using ion selective electrodes while serum urea and creatinine was determined by the colorimetric method.

2.3. Electrocardiography (ECG)

A conventional resting 12 lead ECG was obtained on the subject lying supine at a paper speed of 25mm/s and a calibration of 10mm/mv. The American Heart Association (AHA) recommendation concerning standardization of leads and specification for instruments was used [18], ECG finding was interpreted by the investigator. The ECG parameters that were assessed included:

i. Rate
ii. Rhythm
iii. Axis

iv. Chamber sizes
v. LV hypertrophy
vi. Conduction blocks
vii. Arrythmia

The ECG diagnosis of left ventricular hypertrophy for the study was done using Araoye criteria, $SV_2 + RV_2 > 40$mm in men and $SV_2 + RV > 35$mm for females [19].

2.4. Echocardiography

Transthoracic 2-Dimensional M-mode guided and Doppler echocardiagrams were performed by the investigator on the patients and controls using Chison Echocargraphy machine model- LCD 1502 with D3P64l 3.5MHz Transducer. Measurements were taken in accordance with the American Society of Echocardiology (ASE) guidelines [20].

The echocardiography views that were utilized for the study with the patient in left lateral decubitus position included (a) parasternal long axis, (b) short axis, (c) apical 4 chamber and 5 chamber views. These views and measurements were used to examine wall dimensions, the wall motion abnormalities; chamber dimensions and appearance, the left ventricular mass (LVM), valve morphologies and areas as well as systolic function using ejection fraction and fractional shortening (FS). The LV mass was calculated using the American Society of Echocardiography formula modified by Devereux [21].

LV MASS (g) = $0.8[1.04[ivsd+Lvidd+ivpwd]^3 + 0.6$
IVSTD = interventricular septal thickness in diastole
LVIDD = left ventricular internal diameter in diastole
LVPWD = posterior wall thickness in diastole

Doppler echocardiography recordings of the mitral inflow were obtained from the apical 4 chamber view in order to assess LV filling dynamics. The diastolic function was determined by the ratio E/A which is ratio of peak early transmitral filling velocity (E) and late filling velocity (A), and deceleration time (DCT) as well as Isovolumetric relaxation time (IVRT).

2.5. Statistical Analysis

Statistical analysis was done using the SPSS version 15.0 software (SPSS, Chicago, IL, USA). The values were quoted as means and 95% confidence intervals. Categorical variables were expressed as proportions and percentages while continuous variables were expressed as means $\pm$ standard deviation. Comparisons of continuous variables between groups were performed with the independent t-test.

III. Results

Table 1: Frequency Table For Sex Distribution

Sex	DM No. (%)	HTN No. (%)	DM + HTN No. (%)	Control No. (%)	Chi^2 value	P - value
Male	27 (58.70)	20(43.500)	26 (56.50)	31 (67.40)		
Female	19 (41.30)	26 (56.5)	20 (43.50)	15 (32.60)		
TOTAL	46 (100)	46 (100)	46 (100)	46 (100)	5.485	0.140

Table 1 shows the sex distribution among the 4 groups. A total of 104 males representing 56.50% of the study population and 80 females representing 43.50% of the study population were recruited. The controls had the highest percentage of males (67.40%) while hypertensive had a highest percentage of females (56.50%).However; there was no statistically significant difference in sex distribution among the groups ($p > 0.05$).

Table 2: Age, Anthropometric And Clinical Parameters Of The Subjects And The Conrols

Parameters	DM (N = 46) Mean (±SD)	HTN (N = 46) Mean (±SD)	DM+HTN (N = 46) Mean (±SD)	Control (N = 46) Mean (±SD)	F	P-Value
Age (yrs)	52.89 (±11.50)	52.63 (±12.77)	55.02 (±9.17)	53.11 (±7.02)	0.510	0.676
Body wt (kg)	75.65 (±12.57)	81.78 (±18.51)	76.06 (±11.76)	28.56(±20.57)	1.376	0.256
HT (m)	1.63 (±0.08)	1.62 (±0.07)	1.60 (±0.12)	1.67 (±0.100)	3.34	0.20
BMI (kg/m^2)	28.66 (±4.54)	31.43 (±7.50)	30.01 (±6.34)	28.93 (±6.34)	1.866	0.137
Waist Circ (cm)	94.17 (±12.31)	102.43 (±16.30)	100.26 (±25.67)	92.61 (±14.50)	3.222	0.25
Hip Circ. (cm)	100.61 (±11.21)	109.43 (±13.34)	105.85 (±9.50)	101.76 ±(14.93)	4.837	0.03*
WHR(cm)	0.94 (±0.006)	94.0 (±0.12)	94 (±0.19)	0.92 (±0.11)	0.410	0.746
SBP(mmHg)	117.39 (±10.63)	155.80 (±12.54)	152.39 (±11.54)	117 (±7.23)	181.59	0.001*
DBP(mmHg)	76.09 (±6.82)	93.78 (±7.34)	92.74 (±8.29)	77.08 (±4.8)	88.632	0.001*

* Statistically significant; WT = weight, HT = height, BMI = body mass index, SBP = systolic blood pressure, DBP = diastolic blood pressure, WHR=waist hip ratio.

Table 2 shows the age, anthropometric and clinical parameters of the four study groups. The mean age, weight, body mass index, waist circumference and waist hip ratio did not show any significant difference among the groups ($p > 0.05$). However, the hip circumference, systolic blood pressure and diastolic blood pressure showed statistically significant differences within the groups ($p < 0.05$).

Table 3: The Prevalence And Pattern Of Left Ventricular Diastolic Dysfunction Among The Four Groups.

Grades of LVDD	**DM (N = 46)**	**HTN (N = 46)**	**DM + HTN (N = 46)**	**CONTROL (N = 46)**	**F**	**P Value**
	No (%)	**No (%)**	**No (%)**	**No (%)**		
Normal	17 (37.00)	15 (32.60)	11 (23.90)	29 (63.00)	1.146	
Abnormal Pattern						
Impaired Relaxation	21 (45.70)	24 (52.20)	29 (63.00)	6 (13.00)	1.952	
Pseudo Normalisation	0	0	0	0		
Reversible Restrictive Pattern	6(13.00)	3 (6.50)	1 (2.20)	9 (19.60)	2.621	
Fixed Restrictive Pattern	2 (4.30)	4 (8.70)	5 (10.90)	2 (4.30)	2.540	0.001*
Abnormal Total	29(63.00)	31 (67.40)	35 (76.10)	17 (37.00)		
Grand Total (%)	**100**	**100**	**100**	**100**		

* = statistically significant.

Table 3 shows the prevalence and pattern of left ventricular diastolic dysfunction among the four groups. 63.00% of normotensive diabetic patients had left ventricular diastolic dysfunction (LVDD) ,out of which 45.70% had impaired relaxation, 13.00% had reversible restrictive filling and 4.30 % had fixed restrictive filling pattern. Among the hypertensive patients 67.40% had LVDD, of these 52.20% had impaired relaxation and 6.50% had reversible restrictive filling and 8.70% had fixed restrictive filling pattern.

For the diabetic-hypertensives (DM + HTN)76.10% had LVDD,with 63.00% being impaired and 2.20% had reversible restrictive pattern and 10.90% had fixed restrictive filling pattern .For the controls 37.00% had LVDD, with13.00% being impaired relaxation , 19.60% being reversible restrictive filling pattern and 4.30% had fixed restrictive filling. Diabetic-hypertensive patients had the highest prevalence of left ventricular diastolic dysfunction (LVDD). This observed difference was statistically significant ($p < 0.05$).

Table 4: Doppler-Echocardiographic Parameters For Left Ventricular Diastolic Functions In The Subjects And Controls.

PARAMETER	**DM (N = 46) Mean (±SD)**	**HTN (N= 6) Mean (±SD)**	**DM + HTN (N = 46) Mean (±SD)**	**CONTROL (N = 46) Mean (±SD)**	**F**	**P-value**
E/A	1.089 (±0.66)	1.100 (±0.65)	1.085 (±0.88)	2.940 (±1.162)	1.146	0.341
E-wave	60.33 (±19.80)	63.96 (±18.09)	59.57 (±18.17)	68.37 (±21.88)	1.952	0.123
A – wave	63.62 (±18.13)	67.533 (±22.05)	69.02 (±24.39)	58.00 (±12.34)	2.631	0.053
DC –Time	142.13 (±56.21)	146.40 (±60.13)	154.07 (±54.82)	141.76 (±39.56)	2.540	0.058
IVR-Time	123.64 (±38.59)	135.42 (±47.00)	130.11 (±42.83)	107.67 (±27.18)	4.258	0.046*

* = Statistically significant, E = E-wave velocity, A = A-wave velocity, DC – Time = Deceleration Time, IVR-Time = Isovolumic relaxation time

Table 4 shows that the E/A ratio was lowest in diabetics and diabetic-hypertensive groups while the deceleration time and the A- wave velocity were highest in diabetic-hypertensive subjects (DM-E/A ratio=1.089, DM+HTN-E/A=1.085, DM+HTNDCtime=154.07and DM+HTN-A-wave=69.02) respectively.

The difference observed in the isovolumic relaxation time among the groups was statistically significant ($p < 0.05$) while for the other parameters of diastolic function such as E/A ratio, E-wave velocity, A-wave velocity and deceleration time there was no statistically significant difference ($p > 0.05$).

Table 5: Demographic, Clinical And Biochemical Correlates Of Left Ventricular Diastolic Function Among The Groups.

Parameters	Correlation coefficient	p-value
Age	0.293	0.048*
Sex	0.096	0.526
WT (kg)	0.239	0.110
BMI($kgm^{2)}$	0.099	0.514
WHR	0.038	0.004*
SBP (mmHg)	0.047	0.258
DBP (mmHg)	0.038	0.022*
PP	-0.058	0.203
Creatinine (umol/L)	0.054	0.723
TC(mmol/L)	0.028	0.852
HDL(mmol/L)	-0.163	0.278
TG(mmol/L)	0.037	0.805
LDL(mmol/L)	0.028	0.854
FBS(mmol/l)	0.290	0.051*
HbA_{IC}(%)	0.415	0.004*
Atherogenic Index	0.236	0.114

* = Statistically significant; PP=pulse pressure.

Table 6 shows correlation of left ventricular diastolic dysfunction with demographic, clinical and biochemical parameters. The following parameters - age, waist-hip ratio, diastolic blood pressure and glycated hemoglobin (HbA_{IC}) were the positive correlates of left ventricular diastolic dysfunction. However, no significant correlation was observed with weight, body mass index, systolic blood pressure, pulse pressure, total cholesterol, high density lipoprotein cholesterol, low density lipoprotien, fasting blood sugar and atherogenic index.

IV. Discussion

The results of this study showed that even though 63.00% of normotensive diabetes had left ventricular diastolic dysfunction (LVDD), higher prevalence of 76.10% was found in diabetic – hypertensive subjects, confirming the independent and combined effects of diabetes mellitus and hypertension on the diastolic function of the heart [22]. The results also showed that the greater percentage of the subjects had impaired relaxation stage of left ventricular diastolic dysfunction (DM = 45.70%, HTN = 52.20%, DM+HTN = 63.20% and control = 13%). Impaired relaxation is the first stage of LVDD characterized by delayed left ventricular early diastolic active relaxation and reduced left ventricular suction force. This is due increased stiffness, abnormal relaxation and increased filling pressure of the heart from left ventricular hypertrophy and increased left ventricular mass index, associated diabetic heart and more in the presence of hypertension [23, 24]. This pattern of left ventricular diastolic dysfunction found in our study may be attributed to the medications the subjects were taking, some of which were used in achieving the reversal of left ventricular remodeling. The duration of the diabetes mellitus and hypertension may have also contributed as some of the subjects were newly diagnosed. Studies have shown that these aforementioned factors can influence the severity and progression of LVDD [23]. The pattern of LVDD in our study is similar to that reported by Akintunde et al in a study carried out among hypertensive patients in Osogbo, Nigeria [25]. The mean age was similar among the four-study groups and the body weight (BWT), body mass index (BMI), waist hip ratio (WHR) did not show any significant difference among the groups. However, the mean of BWT, BMI and WHR were higher in the normotensive diabetic, hypertensive and diabetic-hypertensive subjects than the control, which is in keeping with the role of obesity in diabetes mellitus and hypertension [26]. Lauer showed that body mass index of more than 30 kg/m^2 has been found to be associated with increased prevalence of echocardiographic left ventricular hypertrophy and left ventricular diastolic dysfunction [27]. In this study, the diabetic-hypertensive subjects had a mean body mass index of more than $30kg/m^2$ which may have contributed to the high prevalence of left ventricular diastolic dysfunction in the subjects. There were significant differences in both systolic and diastolic blood pressure among the four groups. The diabetic-hypertensive and non-diabetic hypertensive subjects had higher mean of systolic and diastolic blood pressure than the normotensive-diabetic subjects and controls.

However, the normotensive-diabetic subjects had higher mean of the systolic blood pressure than the controls.These higher values of both systolic and diastolic blood pressure in the subjects may contribute to the higher prevalence and severity of left ventricular diastolic dysfunction (LVDD) found in the subjects as compared to the Controls. Data from studies like the Valsartan in Diastolic Dysfunction (VALIDD) trial showed that reduction in blood pressure (the systolic and diastolic blood pressure) resulted in improvement of diastolic function regardless of the type of anti-hypertensive drugs used [28].

Dyslipidemia has been identified as a major risk factor for cardiovascular disease in diabetes mellitus. The normotensive-diabetic subjects and hypertensive subjects had higher level of total cholesterol and LDL cholesterol than the controls. This abnormal lipid pattern causes accelerated atherosclerosis of the coronary artery, impaired myocardial function, hypertension and eventually left ventricular diastolic dysfunction [22, 29]. All these may have contributed to the high prevalence of left ventricular diastolic dysfunction in the

normotensive-diabetic subjects and diabetic-hypertensive subjects found in this study.The normotensive-diabetic subjects and hypertensive subjects had lower level of HDL-cholesterol and also a higher atherogenic index than the controls. This lipid pattern has been known to be associated with increased risk of atherosclerosis and accelerated hypertension with attendant left ventricular diastolic dysfunction. Studies involving type 2 Diabetes mellitus patients in Kaduna, Northern Nigeria showed high prevalence (71%) of lipid abnormalities in diabetics [30, 31]. Oki et al reported reduced HDL cholesterol as common dyslipidemia in type 2 DM, while Chandaha et al reported raised triglyceride and cholesterol levels in diabetics among Asians in India [32, 33].

Different studies have reported the presence of left ventricular diastolic dysfunction (LVDD) in diabetes mellitus patients (DM), with varying results depending on the patient cohort and tools used. The prevalence of 63.00% in normotensive diabetes mellitus subjects, 76.10% in diabetic-hypertensive subjects, 67.00% in hypertensive subjects and 37.00% in controls are similar to that reported by Cesare Russo et al in Columbia, USA [10] and Danbauchi et al in Zaria, Nigeria [11]; affirming the independent and combined effects of diabetes mellitus and hypertesion on the left ventricular diastolic function. The differences in reported prevalence may be due to use of different instruments, parameters and study design in assessing diastolic function. In this study, left ventricular diastolic dysfunction correlated positively with waist hip ratio, diastolic blood pressure, glycated hemoglobin and age. This corroborates with findings from the study by Dhakshinamurthy et al on the risk stratification and correlates of cardiovascular events in type 2 diabetes ,they reported the following as positive correlates: age, obesity, alcohol, smoking, stress, dyslipidemia and hypertension while Danbauchi et al in Nigeria also reported age, dyslipidemia, hypertension, hyperglycemia and left ventricular hypertrophy as the positive correlates of diastolic dysfunction, which are in keeping with the findings of this study [2, 11] . Weight was positive correlate of LVDD in this study, which may have contributed to the high prevalence rate of 63.00% in normotensive diabetics, with mean waist hip ratio of 0.94, which was higher than the cut off value recommended for both sexes by World Health Organization (WHO) [17]. Age was also a positive correlate of left ventricular diastolic dysfunction (LVDD) in this study. Ageing causes increased atherosclerosis affecting both the arteries and the resistant vessels leading to left ventricular hypertrophy, diastolic stiffness, decreased compliance and concomitant LVDD. This is in keeping with the findings by Masugata et al [34]. Diastolic blood pressure was also a correlate of LVDD in this study, the mean values of diastolic blood pressure was higher in the subjects than the controls. This is in keeping with findings in VALLID (Varlsatan in diastolic dysfunction) trial which demonstrated the linear relationship between LVDD with diastolic and systolic blood pressure [28]. $HbA1_C$ correlated positively with LVDD in this study, and the mean values of HbA1c for the normotensive diabetic and diabetic-hypertensive subjects showed suboptimal control according to International Diabetes Federation (IDF) [35]. The suboptimal $HbA1_C$ control is surrogate marker for chronic hyperglycemia, which leads to myocardial fibrosis, stiffness and impaired compliance with concomitant diastolic dysfunction [36].

V. Conclusions

In conclusion therefore, the prevalence of LVDD is 63.0% and 67.4% in normotensive diabetic and hypertensive patients respectively, with a higher prevalence of 76.1% in hypertensive – diabetic patients. Moreso, older age, higher waist-hip ratio, diastolic blood pressure, fasting blood sugar and glycated hemoglobin were positive correlates of LVDD.

VI. Limitations

This study is not without some limitations as it is a hospital based study which may not be a true representative of findings in the general population and stress echocardiography and ECG could have revealed more subtle cases of left ventricular function impairment.

References

[1]. WHO Library Cataloguing-in-Publication Data:Global Status report on Non-communicable disease 2011. www.who.i
[2]. V.A.Dhakshinamurthy , L. Eric, H.David et al, Risk stratification and predictors of cardiovascular events in type 2 diabetes mellitus, European Heart Journal, 27, 2006, 713- 721.
[3]. K.G.Albert and P.Z.Zimmet, Definition, diagnosis and classification of diabetes mellitus and its complications part I. Diagnosis and classification of diabetes mellitus, Provisional Report of WHO Consultation, Debate, Medicine, 15, 1997, 539.
[4]. American Diabetes Association: Diabetes Care, 2(1), 2000, 37.
[5]. A.G.Powers, Diabetes mellitus, classification, Harrison's Principles of Internal Medicine 17th edition ,McGraw Hill Michael Publishing Division, 33, 2001, 2109-2137.
[6]. Report of the expert committee on the diagnosis and classification of diabetes mellitus, Diabetes Care, 20, 1997, 1183-1186
[7]. National Diabetes Group, Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance, Diabetes, 28, 1979, 1039-1057.
[8]. International Diabetes Federation, Global burden of diabetes estimates for 2011 and projection for 2030, IDF Diabetes Atlas, 5th edition, 106, 2011, 23-25. www.idf.org/diabtesatlas.
[9]. W.B.Kannel , M.Hjortland and W.P.Casteli, Role of diabetes in congestive heart, the Framingham study, American Journal of Cardiology, 34, 1974, 29 – 34.

[10]. R. Cesare , J. Zhezhen , H. Shunichi, M. Tatjana Runde's , S.V. Elkind et al,The effect of diabetes mellitus and hypertension on left ventricular diastolic function in a high risk population without evidence of heart disease, European Journal of Heart Failure ,12, 2010, 454-461.
[11]. S.S. Danbauchi , F.E. Anumah , M.A. Alhassan , S.O. David and G.C. Onyemelukwu , Left ventricular function in type 2 diabetes patients without cardiac symptoms in Zaria, Nigeria, Ethnic and Disease ,15, 2005, 635 – 640.
[12]. O. Ogah O, A. Adebayo, A. Falase , D. Ojji, W. Parsonage , M. Dooris et al, Left ventricular dysfunction in normotensive type 2 diabetic subjects, Journal of National Medical Association, 100, 2008, 1066 – 1072.
[13]. B.B. Harry, Echocardiographic assessment of left ventricular function in adult Nigerians with newly diagnosed type 2 diabetes mellitus. A dissertation submitted to the National Post Graduate Medical College of Nigeria, 123, 2005.
[14]. Joint National Committee VII National High Blood Pressure education program, The seventh report of the Joint national committee on prevention, detection, evaluation and treatment of high blood pressure. Hypertension, 42, 2003, 1206-1252.
[15]. D. Perloff , C. Grim, J. Flack, E.D. Frohlick, M. Hill M, M. Mc Donald et al, Human blood pressure determination by sphygmomanometry, Circulation , 88, 1993, 2460-2470.
[16]. International Journal of Obesity (2004), 28, 2004, 226 – 231. Doi: 10.1038/sj.ij. 0802663. BMI Categories (WHO 1997).
[17]. Canadian guidelines for body weight classification in adults; waist circumference measurement, 2003, Appendix 7.7.
[18]. Kligfield , S.L. Gettes, J.J. Bailey, R.Childers, B.J.Deal, W. Hancock et al ,Recommedations for the standardization and interpretation of the echocardiogram, American Journal of Cardiology, 49(10), 2007, 1109-1127.
[19]. M.A. Araoye, Electocardiogram (ECG) diagnosis of left ventricular hypertrophy in blacks, 1996 www.medlinenigeria.com.
[20]. R.M. Lang, M. Bierig, R.B. Devereux, Chamber quantification writing group, American society of echocardiography's guidelines and standards committee , Journal of American Society of Echocardiography, 18, 2005, 1440.
[21]. R. B. Devereux, D. R. Alonso and E.M. Lutas, Echocardiographic assessment of left ventricular hypertrophy: comparison to autopsy findings, American Journal of Cardiology, 57, 1986, 450 – 458.
[22]. The Hypertension in Diabetes Study group. Hypertension in diabetes study (HDS): Prevalence of hypertension in newly presenting type2 diabetic patients and the association with risk factors for cardiovascular and diabetic complications, Journal of Hypertension 11, 1993, 309.
[23]. D.S. Schulman, A.R. Flores, J. Tugoen, S. Dianzumba and N. Reichek, Antihypertensive treatment in hypertensive patients with normal left ventricular mass is associated with normal left ventricular remodeling improved diastolic function, American Journal of Cardiology, 78, 1996, 56–60.
[24]. S. Zieman and D. Kass, Advanced glycation end product cross-linking:Pathophysiologic roles and therapeutic target in cardiovascular disease, Congestive Heart Failure, 10, 2004, 144-149.
[25]. A.A. Akintude, Echocardiographic indices for diastolic dysfunction in hypertensive patients, The International Journal of cardiology, ISSN, 1578 – 1534.
[26]. A.G. Bertoni, W.G. Hundley, M.W. Massing et al, Heart failure prevalence, incidence, and mortality in the elderly with diabetes, Diabetes Care, 27, 2004, 699-703.
[27]. M.S. Lauer, K.M. Anderson, W.B. Kannel et al, The impact of obesity on left ventricular mass and geometry, The Framingham Heart Study, Journal of the American Medical Association, 266(2), 1991, 231-6.
[28]. S.D. Solomon, Effect of angiotension receptor blockade and anti-hypertensive drugs on diastolic function of the heart, A randomised trial: Lancet, 369, 2007, 2029 – 2087.
[29]. P. J. Watkins, Cardiovascular disease, hypertension and lipids, British Medical Journal, 326, 2003, 874-876.
[30]. Milan study on atherosclerosis and diabetes (MISAD) group. Prevalence of unrecognised silent myocardial ischaemia and its association with atherosclerotic risk factors in non-insulin dependent diabetes mellitus, American Journal of Cardiology, 79, 1997, 134-139.
[31]. A. Shehadeh, T.J. Regan, Cardiac consequences of diabetes mellitus, Clinical Cardiology, 18, 1995, 301-305.
[32]. J.C. Oki, Dyslipidemias in patients with diabetes mellitus: classification and risks and benefits of therapy, Pharmacotherapy, 15(3), 1995, 317-337.
[33]. H.B. Chandalia, A. Jayshree, B. Jayshree B, P. S. Lamba and B.D. Punekar, Lipid abnormalities in diabetes mellitus, International Journal of Diabetes in Developing Countries, 19, 1999, 1-6.
[34]. H. Masugata , S. Senda, F. Goda et al, Left ventricular diastolic dysfunction in normotensive diabetic patients in various age strata Diabetes Research and Clinical Practice, 79(1), 2008, 91-96.
[35]. International Diabetes Federation, Type 2 diabetes clinical practice guideline for Sub-Saharan Africa, International Diabetes Federation, 17, 2006.
[36]. Z.Y. Fang, J.B. Prins and T.H. Marwick , Diabetic cardiomyopathy: evidence, mechanisms, and therapeutic implications, Endocrine Reviews, 25(4), 2004, 543-567.

*Vincent Maduka Uhegbu. "The Predictors of Left Ventricular Dysfunction Among Hypertensive And Diabetic - Hypertensive Patients in A Tertiary Hospital in Nigeria." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 16.9 (2017): 64-70