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Prevalence and Types of Left Ventricular Dysfunction in People Living with both Type 2 Diabetes Mellitus and Hypertension attending University of Port-Harcourt Teaching Hospital, Rivers State, Nigeria

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Abstract

Background: Hypertension and type 2 diabetes mellitus co-occurrence is a common co-morbidity with hypertension being the most common cause of left ventricular dysfunction in our environment. Data on the prevalence, types of left ventricular dysfunction in people living with both type 2DM and hypertension is scarce in our locality. To study prevalence and types of left ventricular dysfunction in people living with type 2 DM and hypertension co-morbidity. **Methodology:** This is a cross-sectional study carried out at the cardiology unit of the department of Internal Medicine, University of Port Harcourt Teaching Hospital. One hundred and sixty adults 18years and above living with hypertension and type 2 diabetes were recruited. 80 adults living with hypertension matched for age and sex served as control. Data on patient's age sex and occupation were obtained. Echocardiography was performed on all participants according to American Echocardiography guidelines and parameters obtained. **Results:** A total of 160 subjects 18 years and above living with type 2 DM and hypertension were recruited as cases while 80 subjects 18 years and above living with hypertension alone were recruited as controls. The mean ages of the cases was 57.51 ±9.1years. The mean ages of the controls was 55.9±12.15 years. There were 60% females and 40% males among the cases. The prevalence of left ventricular dysfunction was significantly higher in the cases compared with the controls (78.8% versus 56.2%, p=0.001) .Slightly more of the cases had diastolic dysfunction than systolic dysfunction (40.6% versus 38.2%.) There was statistically significant higher systolic than diastolic dysfunction among the hypertensive control (42.5% versus 13.8%) Comparing systolic dysfunction among the cases and control did not show any significant difference (p=0.514) while diastolic dysfunction was significantly higher in the cases than the controls (40.6% versus 13.8% P=0-001). Left ventricular Mass Index was comparable in both the cases and controls. **Conclusion:** This study found that the prevalence of left ventricular dysfunction (78%) in hypertensive patients living with type 2 DM is high and diastolic dysfunction being higher than systolic dysfunction in this group of patients. The prevalence of left ventricular hypertrophy was comparable in both hypertensive patients and people living with type 2 DM and hypertension co-morbidity.

Keywords: Prevalence, Types of Left Ventricular Dysfunction, People Living with Type 2 Diabetes Mellitus, Hypertension.

INTRODUCTION

Hypertension is a common co-morbid condition in diabetes mellitus and vice versa. DM and hypertension co-exist in approximately 40-60% of patients with type 2 DM [1]. The concept that DM can precipitate heart failure has been demonstrated even from the Framingham study which estimated that men and women with DM have a two and four- fold respectively increased risk of developing HF compared to non-diabetic subjects [2]. Hypertension increases the risk of heart failure at all ages. Data from the Framingham heart study found that after age 40, the life time risk of developing HF was twice as high in subjects with BP≥160/90mmHg [3]. Therisk of developing HF increases with the degree of BP elevation. However even moderate elevation contributes to risk of heart failure in the long term [3]. Most often, overt heart failure is preceded by asymptomatic and symptomatic left ventricular systolic dysfunction. The prevalence of diastolic dysfunction has been well documented in Caucasians ranging from 46%- 68% of the hypertensive population [4]. In humans left ventricular diastolic dysfunction is considered the earliest manifestation of diabetic cardiomyopathy preceding the development of systolic dysfunction [5]. It remains uncertain whether impaired systolic and diastolic left ventricular function of the diabetic hypertrophied heart is owing only to a larger left ventricular mass induced by the combination of diabetes and hypertension or if diabetes induces further functional changes independent of the presence of the left ventricular hypertrophy. It is however certain that the combined action of diabetes and hypertension can make the heart more vulnerable to structural and functional changes, increased LVH and heart failure [6].

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Knowing the prevalence and types of left ventricular dysfunction in people living with type 2 DM and hypertension co-morbidity would aid tailoring specific management needs of this group of patients.

This study therefore aims at identifying the prevalence and types of left ventricular dysfunction in people living with type 2 DM and hypertension co-morbidity receiving care in University of Portharcourt Teaching hospital in Rivers State Nigeria.

METHODOLOGY

Study population consisted of hypertensive type 2 diabetic subjects equal or greater than 18years of age seen at the medical out- patient clinic or admitted into the medical ward of the hospital randomly selected. Data was obtained from subjects who were considered hypertensive diabetic on the basis of blood pressure ≥140/90mmHg and existing diagnosis or fasting blood sugar greater than 7.0mmol/l. An arm of control comprising 80 hypertensive patients attending medical outpatient clinic or admitted to the medical ward selected randomly and matched for age and sex were recruited. Data obtained include age, sex, family history of DM and hypertension, history of ischemic heart using a structured questionnaire. The study subjects underwent clinical examination to determine weight, height, waist circumference, body mass index and waist-hip ratio. Pulse was counted for one minute assessing rate, rhythm, volume, character and synchrony. Blood pressure was measured using accoson mecury sphygmomanometer to determine the brachial arterial systolic and diastolic blood pressure using the first and fifth korotkoff sounds respectively [7]. Two blood pressure measurements were taken measured 3 minutes apart and after 5 minutes of rest with the arm at the heart level and the average recorded. Exercise, smoking, and caffeine were avoided at least 30 minutes prior to the blood pressure measurement. Hypertension was deemed present if BP is ≥ 140/90mmHg on at least 3 occasions or current use of antihypertensive agents [7]. A venous blood sample was drawn and analyzed in the hematology, chemical pathology laboratories of UPTH for plasma hemoglobin, fasting plasma glucose. Venopuncture was carried out and 10 ml of fasting blood sample was drawn from each subject and hypertensive control between 7:30am and 8:30am, 2mls was put into fluoride oxalate bottle for FBG estimation.

ECHOCARDIOGRAPHY

Transthoracic M-mode, 2-dimensional and Doppler echocardiography with colour flow was performed on all subjects using Mindray DC – N6 Diagnostic ultra sound system. Calculations were made using internal analysis software of the echocardiography device. The 2-diamensional views were used for real time morphological characteristics and also as a reference for selection of the M-mode beam.

Echocardiographic assessment were done according to the recommendations of American Society of Echocardiography [8]. Subjects were examined in left lateral decubitus position using parasternal long axis and apical short axis view. LV internal diameter, interventricular septal thickness and LV posterior wall thickness were measured at end diastole and end systole. LV systolic performance was assessed using the fractional shortening (FS) and the ejection fraction (EF) of the left ventricle. These were calculated automatically by the machine using the teicholz formula [9]. The left ventricular mass calculated using the estimation of LV mass from LV Linear dimensions [10].

$LVM\ (gm) = 0.8 \times [1.04\ (LVIDd + PWTd + IVSTd)^3 - LVIDd^3] + 0.6g$

Where:

LVIDd = left ventricular internal diameter in diastole.

PWTd = Posterior wall thickness in diastole and

IVSTd = Interventricular septal thickness in diastole.

Left ventricular mass index (LVMI) was calculated by indexing the LVM to the body surface area. Left ventricular hypertrophy (LVH) was defined in absolute terms as LVMI > 115g/m2 in Men and > 95g/m2 in Women [10].

Relative wall thickness (RWT) was calculated using the formula.

$$RWT = (2xPWTd/LVIDd)$$

Where PWTd is posterior wall thickness in diastole.

LVIDd is LV internal diameter in diastolic. The aortic root was measured at end diastole, while, the left atrium was measured at end ventricular systole. Left ventricular diastolic function was evaluated by studying the filling dynamics of the left ventricle, the isovolumetric relaxation time (IVRT), the pulmonary venous flow and tissue Doppler imaging derived myocardial wall velocities [11].

The mitral in flow velocities was measured from apical four chamber views with pulsed Doppler or continuous wave Doppler and with the sample volume position at the tip of mitral valve leaflets.

The inflow characteristics is quantified by measuring the transmitral "E" wave velocity (peak early mitral in flow velocity). The E/A ratio and the deceleration time (DT) (time interval of peak E wave velocity to it's extrapolation to the base line) is calculated [12].

The pulsed Doppler sample was positioned midway between the mitral valve tips and the aortic outflow tract so that isovolumetric relaxation time (IVRT) could be measured between the point of aortic valve closure and mitral valve opening.[12]

The pulmonary venous flow velocities were gotten from an apical 4-chamber view with the pulsed wave Doppler by placing the cursor 1-2cm into the right upper

and minimal optical pulmonary vien close to the atrial septum. Colour flow Doppler was used to identify the pulmonary vien [13].

The peak systolic (S wave), diastolic (D wave) flow velocities and peak atrial reversal (Ar) and Ar duration were recorded and the ratio S/D calculated.

Tissue Doppler echocardiography were obtained using 25cm/s scale gains. In the apical 4-chamber view, a 2mm pulse wave Doppler sample gate will be placed at the medical annulus to obtain the peak early diastolic (E^1), atrial (A^1) and systolic (S^1). These values were used to calculate the E/E^1 ratio [10].

Diastolic filling pattern was classified into normal filling pattern and diastolic dysfunction [14].

Left ventricular dysfunction was categorized into systolic and diastolic dysfunction on the bases of Ejection Fraction (EF), and impaired relaxation or pseudo-normalization or restrictive pattern respectively. Cases with EF<55% were considered systolic dysfunction while cases with impaired relaxation/pseudo-normalization/restrictive pattern were considered diastolic dysfunction [15].

RESULTS

A total of 160 subjects 18 years and above living with type 2 DM and hypertension were recruited as cases while 80 subjects 18 years and above living with hypertension alone were recruited as controls. The mean ages of the cases was 57.51 ±9.1 years. The mean ages of the controls was 55.9±12.15 years. There were 60% females and 40% males among the cases. The base line clinical characteristics of the study population are represented in Table 1. The prevalence of left ventricular dysfunction was significantly higher in the cases compared with the controls (78.8% versus 56.2%, p=0.001). Slightly more of the cases had diastolic dysfunction than systolic dysfunction (40.6% versus 38.2%.). There was statistically significant higher systolic than diastolic

dysfunction among the hypertensive control (42.5% versus 13.8%) Comparing systolic dysfunction among the cases and control did not show any significant difference (p=0.514) while diastolic dysfunction was significantly higher in the cases than the controls (40.6% versus 13.8% P=0-001). This is represented in Table 2. Left ventricular Mass Index was comparable in both the cases and controls. Other echocardiographic features of the study population were summarized in Table 3.

Of the 61 cases with systolic dysfunction 22(36.1%) were males while 39(63.9%) were female. This did not show any statistically significant difference (p=0.781). Of the 65 cases with diastolic dysfunction 25(38.5%) were males while 40(61.5%) were females. This was not statistically significant (p=0.712). Among 34 hypertensive controls with systolic dysfunction 17(50%) were males and 17(50%) were females. There was no statistically significant difference in the sex distribution of left ventricular systolic dysfunction in this group (p=0.621). Similarly there was no statistically significant difference in the sex distribution of diastolic dysfunction in this group as 4(36.4%) of males and 7(63.6%) females respectively had diastolic dysfunction (p=0.431). The sex distribution of left ventricular dysfunction across the cases and control was shown in table 4.

Table 1: Clinical characteristics of the study population

VARIABLE	Cases(n=160)	Hypertensive controls (n=80)	P
	Mean ± SD	Mean ± SD	
AGE	57.51±9.18	55.90±12.15	0.323
BMI (kg/m2)	29.46 ± 5.64	26.66± 4.40	0.022*
WC (cm)	100.23±10.54	97.78± 12.10	0.108
WHR	1.0 ± 0.09	0.95± 0.07	0.001*
SBP (mmHg)	137.54±17.14	136.26 ±20.21	0.599
DBP (mmHg)	83.41± 9.44	87.29 11.00	0.000*
PR b/min	83.78 ±11.55	76.90 9.57	0.000*
Pulse pressure (mmHg)	53.59± 13.85	49.00 13.08	0.014*

Key: Cases= Patients with hypertension and diabetes; P= p value for Cases versus Hypertensive controls

Table 2: Prevalence and types of left ventricular dysfunction across cases and hypertensive controls

	Cases n=160(%)	Hypertensive control n=80(%)	TOTAL n=240(%)	P
Left ventricular dysfunction	126(78.8)	45(56.2)	171(71.2)	0.001*
Left ventricular dysfunction type Systolic dysfunction	61(38.1)	34(42.5)	95(39.6)	0.514
Diastolic dysfunction	65(40.6)	11(13.8)	76(31.7)	0.001*

Key: Cases =Patients with Hypertension and Diabetes

Table 3: Echocardiographic features of the study population

VARIABLES	Cases(n=160)	Hypertensive controls(n=80)	P
	Mean ±SD	Mean ±SD	
LVEF (%)	51.40 ±15.38	57.76 15.58	0.021*
LVFS (%)	25.34± 9.74	31.59 ±10.50	0.036*
LVMI (g/m^2)	124.39± 43.44	131.69± 44.10	0.388
RWT	0.74 ±0.57	0.62± 0.28	0.125
LAD (cm)	3.48±0.64	3.47+0.67	0.967
IVSDd (cm)	1.43± 1.25	1.33 ±0.29	0.180
LVIDd (cm)	4.28± 0.98	4.60± 0.90	0.020*
LVPWd (cm)	1.49 ±1.61	1.31± 0.34	0.456
E/A	1.20 ±0.68	1.30 ±0.59	0.001*
IVRT (ms)	101.2 ±16.87	96.88± 17.19	0.138
DT(ms)	208.63± 46.20	203.91± 40.03	0.708
S/D	1.18 ±0.52	1.17 ±0.02	0.616
E/E1	6.05± 2.05	7.38± 2.70	0.001*
GEOMETRY			
Normal	10(6.2%)	6(7.5%)	0.262
Concentric remodeling	27(16.9%)	18(22.5%)	0.293
Concentric hypertrophy	107(66.9%)	44(55%)	0.073
Eccentric hypertrophy	16(10.1%)	12(15.0%)	0.255

LVEF = left ventricular Ejection fraction, LVFS = left ventricular fractional shortening, LVMI = Left ventricular mass index, RWS = Relative Wall Thickness, LAD = left atrial diameter, IVSD = Interventricular septal thickness in diastable LVIDd= left ventricular internal diameter in diastole, LVPWD = left ventricular posterior wall thickness in diastole. E/A = Mitral Early inflow and late Atrial inflow velocity ratio, S/D= Pulmonary Vein Systolic/Diastolic flow ratio, E/E = Mitral valve Early Velocity/ Mitral Annular Tissue Early Velocity, DM=Diabetes, P1=For Cases versus Hypertensive controls, P2=Cases versus Normotensives

Table 4: Sex distribution of left ventricular dysfunction types

	CASES n=126		TOTAL	HYPERTENSIVE CONTROLS n=45		TOTAL
	Male n=47(%)	Female n=79 (%)		Male n=21(%)	Female n=21(%)	
Types of left ventricular dysfunction						
Systolic Dysfunction	22 (36.1) p =0.781	39 (63.9)	61 (100)	17(50) p = 0.621	17(50)	34(100)
Diastolic Dysfunction	25(38.5) P=0.712	40 (61.5)	65 (100)	4(36.4) p =0.431	7(63.6)	11(100)

Key: Cases=Patients with hypertension and diabetes; Hypertensive control= Patients with hypertension

DISCUSSION

The prevalence of left ventricular dysfunction in hypertensive patients living with type 2DM diagnosed primarily by echocardiography in this study is high (78.8%). The proportion of this group of patients with diastolic and systolic dysfunction were 38.2% and 40.6% respectively. Systolic dysfunction has been found to be caused by advanced diabetic cardiomyopathy by inducing impaired contractile function [16]. Myocardial cell death and necrosis are increased and may be mediated by augumented angiotensin converting enzyme expression in hyperglycemic state, resulting in poor systolic function [17]. Sustained hyperglycemia increases glycation of interstitial protein such as collagens resulting in myocardial stiffness hence diastolic dysfunction. Cessaro *et al* documented 14.7% of left ventricular diastolic dysfunction in their study which is far lower than that found in this study [18]. This difference can be explained by the fact that they excluded symptomatic patients from their study. Similar to this study, Amusa *et al* documented a prevalence of 34% of diastolic dysfunction in their study [19]. Anderson *et al* reported even higher prevalence of 60% of diastolic dysfunction in their study [20].

Grade 1 diastolic dysfunction was found more in the cases than the controls (50.6% versus 28.8%). This is not surprising considering the additive effect of type 2 DM and hypertension on the cardiac myocytes. The greater proportion of the hypertensive controls had grade 2 diastolic dysfunction compared to the cases 37.5% versus 23.1%. The explanation for this is not readily discernible from this study. Restrictive pattern was slightly higher in the cases than controls. The finding in this study is in agreement with that documented by Anderson et al.20Similiar finding was documented by Vinereanu *et al* [21].

In the present study, the prevalence of both systolic and diastolic dysfunction were slightly higher in females than males (40.6% versus 34.4%) and (41.7% versus 39.1%) respectively. Danbauchi *et al* [22] in Jos Nigeria found similar pattern of sex distribution among hypertensive diabetic patients with left ventricular dysfunction.

Left ventricular mass index was comparable across the cases and hypertensive controls similar to findings by Hilderbrandt *et al* [23] but contrary to that documented by Anderson etal20who documented significant difference in LVMI across the cases and hypertensive controls in their study population. This difference may likely be due to small sample size in their study (70 cases, 35 controls). The finding of impaired systolic function despite comparable LVMI in the present study suggests that additional impairment in left ventricular function is associated with DM independent of the left ventricular hypertrophy.

CONCLUSION

This study found that the prevalence of left ventricular dysfunction (78%) in hypertensive patients living with type 2 DM is high and diastolic dysfunction being higher than systolic dysfunction in this group of patients. The prevalence of left ventricular hypertrophy was comparable in hypertensive patients and people living with type 2 DM and hypertension co-morbidity. The combination of hypertension and type 2 DM increases the risk of left ventricular dysfunction. Appropriate measures should be put in place to address this comorbidity beyond blood pressure and glycemic control.

Conflicts of Interest

There is no conflict of interest in this study.

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