An Echocardiographic Study Of Right Ventricular Function In Systemic Hypertension

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Abstract

INTRODUCTION: Hypertension remains the commonest cardiovascular disease in Nigeria. Most studies have focused on LV function assessment in HBP with minimal study of RV. The aim of this study is to assess RV function in HBP subjects and to assess the association, if any, of LV dysfunction and RV dysfunction. METHODS: The study was a case control study. A total of 200 subjects were recruited,of which,100 were hypertensive subjects while another 100 were normotensive controls RVSD was defined as Tricuspid annular plane systolic excursion (TAPSE)<1.6cm while RVDD was defined as Tricuspid E/Aratio <0.8 or>2.1 or between 0.8-2.1 with inferior vena cava (IVC)collapsibility of>50%.RESULTS:Amongst the hypertensive subjects, the prevalence of RVSD was 32% while that for RVDD was 64%.Right ventricular hypertrophy (RVH) was present in 79% and RV dilatation in 13%.There was statistically significant association between parameters of LV dysfunction and RV dysfunction in HBP subjects (LVH and RVSD,p=0.003;LVEF and TAPSE,r=0.497,p=<0.001).CONCLUSION: The study found that one-third of patients with HBP had RVSD as defined by reduced TAPSE and over half had RVDD. Also some indices of RV structure like RVWT and RV diameter were abnormal in about one-tenth of these HBP patients. This then is a topic for more research.

KEYWORDS: BP-Blood pressure, Echo-Echocardiography, EF-Ejection fraction, HBP-Hypertension, HHD-Hypertensive heart disease, IVC-Inferior Vena Cava, IVS-Interventricular septum, LVH -Left ventricular hypertrophy, LVEF-Left ventricular ejection fraction, LV -Left ventricle, LVESD-Left ventricular end systolic diameter, LVPW-Left ventricular posterior wall, M-MODE-Motion mode, RIMP-Right ventricular index of myocardial performance, RV-Right ventricle, RVEF-Right ventricular ejection fraction, RVDD-Right ventricular diastolic dysfunction, RVH-Right ventricular hypertrophy, RVSD-Right ventricular Systolic dysfunction, RVWT -Right ventricular wall thickness, TAPSE-Tricuspid annular plane systolic excursion

INTRODUCTION

In 1616, Sir William Harvey was the first to describe the importance of the right ventricle (RV/) in his seminar treatise, when he wrote "Thus the right ventricle may be said to be made for the sake of transmitting blood through the lungs and not for nourishing them".' For many years that followed, emphasis in cardiology was placed on the left ventricle (LV) physiology, overshadowing the study of the right ventricle (RV).'In the first half of the 20th century, the study of the RV and its function was limited to a small group of investigators who were intrigued by the hypothesis that the human circulation could function adequately without the RV. This was to be later disproved. Knowledge about the role of the RV in health and disease historically lagged behind that of the LV. This may be because it was less involved in diseases of epidemic proportions such as Myocardial ischaemia, cardiomyopathy or valvulopathy.2

Another reason for this setback could be due to its structural and functional complexity and its shape being less amenable to geometric simplification for volumetric analysis in echocardiography (Echo).3 Hypertension (HBP) is a common, important and major global public health problem and the overall prevalence of HBP in Nigeria ranges from 8% to 46% depending on the study, target population, type of measurement and the value for defining it.' Elevated blood pressure (BP)is an early indicator of epidemiologic transition and is apparent as the population moves from rural to urban setting. It is projected that 1.5 billionpeople,Le one-thirds of the world's population will be hypertensive by 2025."To date,BP control in our various clinics and health centre is noted to be controlled below 140/90mmhg in less than one-third of the affected individuals.

HBP is responsible for most cases of heart failure (HF) in Nigeria;78.7%6 in Abeokuta heart failure registry, 62.6%inAbuja,and 56.3% in Portharcourt.

The effect of systemic HBP on the LV has been variously studied and recognised, ranging from Left ventricular hypertrophy (LVH) to LV systolic and diastolic dysfunction and eventual cardiac failure." Thus normal values of LV dimensions, mass and function are available unlike for the RV.

In the setting of the effect of HBP on the heart,the RV has generally been considered a mere "by-stander" or a "conduit", a victim of circumstance and indirectly affected by this pathologic process unlike the LV which has a direct relationship with HBP.'Only few works have been done on the effect of HBP on the seemingly "non-stressed" RV. This has hindered the development of normal reference values for RV function using Echo.'Recent evidence has shown that HBP has an effect on the RV, not only in the setting of severe HBP but also in mild hypertensives without evidence of cardiac damage and even untreated pre-hypertensives. This association is mediated through the interventricular septum (IVS) via the term referred to as "VENTRICULAR INTERDEPENDENCE"(VI),"' VI is defined as forces that are transmitted directly from one ventricle to the other through the myocardium and pericardium, independent of neural,humoral or circulatory effects." In order words,HBP causing damage to the LV also affects the RV through VI.

Not much has also been done on the RV in hypertensive heart failure (HHF). RV systolic function represents a strong and independent predictor of mortality in left heart failure(HF). It has also been shown to be associated with poor exercise capacity in HF patients.7,3,9,10

As advances in Echo is occurring,there is the need to examine the RV as part of a complete cardiac Echo and also because of its role in the morbidity and mortality of patients presenting with signs and symptoms of cardiopulmonary disease.In recognition of this fact, the American society of Echocardiography (ASE) developed a set of guidelines in 2010 for the assessment of the RV."Due to the paucity of data on the RV inHBP in Nigeria and indeed sub-Saharan Africa, this study therefore seeks to provide data on RV function in our hypertensive patients.

AIM AND OBJECTIVES OF THE STUDY

AIM

- To determine RV function in hypertensive subjects,

SPECIFIC OBJECTIVES

- To determine the prevalence of RV dysfunction (systolic and diastolic) in hypertensive patients.
- To determine the type of RV dysfunction in hypertensive patients.
- To determine the relationship between RV function and the duration of hypertension
- To determine the association between LVH and RV function

MATERIALS AND METHODS

STUDY SUBJECTS

A total of 200 subjects who gave consent for the study was enrolled.

They were divided into 2 groups:

- Hypertensive subjects
- Age and sex matched controls

SAMPLING PROCEDURE

Participants who met the inclusion criteria were interviewed via a structured questionnaire on their socio-demographic details such as age and sex. Physical examination, BP, weight, height, Electrocardiography and Echo were carried out for each participant.

INCLUSION CRITERIA

- The subject must be at least 18 years
- Diagnosis of HBP was made by a physician and verified by the researcher with a blood pressure value of >140/90 mmHg or was on antihypertensive medications. A set of 3 BP readings done 5minutes apart. The average of the last 2 taken as the clinic BP
- Consent to participate in this study

EXCLUSION CRITERIA:

- Hypertensive patients with Atrial fibrillation,ventricular fibrillation, ventricular tachycardia and Acute coronary syndrome
- Hypertensive patients with known valvular lesions or congenital heart diseases
- Documented or suspected cardiomyopathy
- Documented or suspected COPD and Corpulmonale
- Documented primary pulmonary Hypertension
- Documented Pulmonary embolism
- Chronic kidney disease
- Diabetes mellitus
- Obese subjects

CONTROL SELECTION

Apparently healthy individuals and staff of the hospital and University above the age of 18 years with BP <140/90mmHg and not on any antihypertensive medication, was used. These were matched with the cases by age and sex.

Left ventricular mass was calculated using an autopsy-validated formula by Devereux.'

LV hypertrophy was considered present when LV mass index is>104g/m^2∈women and>116g/㎡ in men. RV wall thickness measured by M-mode or 2-D Echo via subcostal or parasternal window, values >5 mm indicated RVH.RV dimensions was taken at end diastole from a RV focused Apical 4 chamber view. RV basal diameter which is the maximal short axis diameter in the base of the RV, at the level of the tricuspid annulus.

Tricuspid annular plane systolic excursion ((TAPSE)was measured from the tricuspid lateral annulus in M-mode. It measures the amount of longitudinal motion of the annulus at peak systole. Values <16 mm indicated right ventricular systolic dysfunction.

Using pulse doppler, right ventricular diastolic dysfunction;Where E/A ratio of <0.8 suggests impaired relaxation and>2.1 suggests a restrictive physiology.Pseudonormal diastolic function is suggested by a normal E/A ratio between 0.8-2.1 but with an IVC collapsibility <50% on inspiration, this implies an elevated right atrial pressure.

DATA ANALYSIS

- Data obtained was entered into IBM SPSS version 21 software for analysis
- Categorical data was described using frequencies,percentages and proportions.
- Normally distributed continuous data was described using means and standard deviations
- Ordinal data and non-normally distributed continuous data was described using medians and interquartile ranges
- Chi square test was used to asses for associations between categorical variables
- Difference of means between groups was analysed using students t-test or ANOVA as appropriate
- Odds ratio was used to analyse risk factors for RV dysfunction in hypertensives

- Correlation between quantitative variables was also performed
- A P-value of less than 0.05 was considered statistically significant

RESULTS

There were a total of 96 females (48%) and 104 males (52%).The sex ratio of participants was 1.1:1 in favour of males. The mean age was 57.8±13.1 years. The mean age of hypertensive participants was 57.8±13.3 years while that of controls was 57.7±12.8 years.This difference in the mean ages of hypertensives and controls was not statistically significant(t= 0.05,p=0.96).There was also no statistical difference between the mean ages of both sexes (57.8±13.8 vs 57.7±12.3,t=0.05,p=0.96). The mean systolic BP was 131.70+17.60 mmHg while diastolic was 82.30±10. 31mmHg.The mean systolic BP was higher amongst hypertensive participants than controls (139.1±20.0mmHg vs 126.4±10.6mmHg).

This was statistically significant (t=5.71, p=<0.001).The difference in the mean diastolic BP of both hypertensives and control was also statistically significant (85.7±11.3mmHg vs 79.8±8.0 mmHg ,=4.20, p=<0.001). Amongst the hypertensive participants, RVDD (which was suggested by trans tricuspid E/A ratios, deceleration time and IVC diameter and compressibility) was present in 64% (64) and 15% of controls, this difference was statistically significant(p=<0.001). RVSD was present in 32% (32)of the hypertensive participants and 2% of controls (p=<0.001).The greater portion of the RVSD(56.25%)was produced by the participants that met the Framingham criteria for heart failure. LVH was present in 50% of hypertensive participants and 10%of controls. This association was statistically significant(p=<0.001).RVH was present in 79% of hypertensives and 77% Of controls(p=0.321). RIMP was abnormal in 64% of cases and 18% of controls. This was also statistically significant (p=<0.001).RVSD was present in 23 participants (46%) of this subset with LVH as compared to 9 (18%)of hypertensive participants without LVH. This was statistically significant(X^2=901,p=0003)RVDDwapresent in 35 participants (70%) with LVH as compared to 29 participants (58%) without LVH. This association was not statistically significant(x^2=156,p=0.211).

TAPSE had a positive correlation with LVEF via Pearson's correlation and this association was statistically significant(r=0.5,p=<0.001).The value of R2was 0.247 which is a 24.7% predictability of TAPSE based on LVEF.

DISCUSSION

RVDD was present in 64% of hypertensive participants. RVDD in hypertension could be as a result of LVH and its bulge into the RV increasing RV end diastolic pressure resulting in RVDD. A similar value was obtained in the study done by Karaye et al13where the RVDD was present in 61.72% of the hypertensive participants. This difference may be due to the fact that the study by Karaye was only with patients that had been diagnosed with HHD on echo unlike this study where participants were recruited prior to echo. The study by Akintunde et al 14 had RVDD present 47.5% of participants. This dissimilarity between this study and that of Akintunde et al may be due to the modality used in assessing RVDD as

only trans-tricuspid doppler flow was used as against the American society of Echocardiography recommendation of trans-tricuspid doppler and/or IVC diameter and hepatic vein flow as was used in this study. RVDD amongst controls was 15% and this association was statistically significant(p= <0.001).This resulted from the increase in pulmonary pressure with age and also age related ventricular stiffening which causes elevated RV end diastolic pressure and eventually RVDD.15.16

RVSD was present in 32% of the hypertensive participants in this study. This value was similar to that done by Karaye etal^13which was 32.03%. RVSD was present in 2% of controls and this association was significant(p= <0.001). The RV is linked to the LV in several ways: by a shared septum, epicardial fibres and a pericardial space. This isthe concept of V.I, where about 20-40% of the RV systolic output results from LV contraction.5 This interdependence can be affected by the deleterious effect of HBP on the LV (LVH for example)and thence affectation of the RV directly. LVH affects the LV contractility and thence the RV looses its 20-40%support from the LV leading to RVSD. Also LVH increases LV end diastolic pressure and eventually elevated pulmonary pressures that can affect the RV.RVDD also results from pressure of the LVH on the non-compliant pericardium and the increased bulge of the hypertrophied IVS increasing both LV and RV end diastolic pressures leading to RVDD.17,18,19

LVH was present in 50% of cases.A study done on hypertensives in Ibadan by Salako et al2° showed LVH to be present in 46.5% of participants on echo. This value was similar to that in this study. Ten percent (10%) of controls in this study had LVH and this difference was statistically significant (p= <0.001). The LVH in the controls may have resulted from undiagnosed hypertension especially in the elderly. RVH was present in 79% of cases and a close value of 77% was present in normotensives. This difference however was not statistically significant(p=0.733).A rationale for the high prevalence of RVH in the controls could be due to the fact that myocardial walls are thicker in Africans than Caucasians21 and the value of >5mm thickness for RVH was derived from pooled studies on Caucasians. There is paucity of data on RVH in hypertensive Nigerians but an analysis of pooled data of 13 studies in Italy by Cuspidi etl^22resulted in a range of prevalence of RVH as 17-80%.Another study also by Cuspidi et al got a prevalence of RVH of 33.6%.

The mechanism of RVH in HBP has not been fully elucidated but a few factors have been proposed viz:transmission of tension experienced by the LV to the RV due to common structures like IVS and pericardium, also neurohormonal factors and inflammatory mediators which mediate LVH in response to pressure overload are thought to affect the RV leading to RVH and fibrosis.17,18.19

RIMP was abnormal in 64% of hypertensives and 18% of controls. This difference was statistically significant (p=

<0,001). RIMP is a non-specific test which is a pointer to either RV systolic or diastolicdysfunction or both There also is paucity of data on RIMP in hypertensive Nigerians.

TAPSE had a strong positive correlation wwith LVEF which was statistically significant(r=0.5,p=<0.001).This was almost identical with the finding in the Karaye et al study〖^18〗(r=0.462,p=<0.001). The relationship of TAPSE to LVEF is linear, thus as LVEF decreases,TAPSE also decreases. This is also through the concept of V.I ®'5 where if the RV looses its contractile support from the LV, its systolic function declines.Through the concept of V.I, the RV gets 20-40% of its contractile force from the LV via the IVS. This therefore implies that if the LV has poor systolic function,its assistance of RV contractility will also be poor,hence the positive linear correlation. The correlation is not perfectly linear because TAPSE is an indirect measure of RVEF. A better correlation may have been obtained if LVEF was compared with RVEF. TAPSE is said to be a partial representation of global RV function." In the study be Karaye et al", the best correlate for TAPSE was its Mitral counterpart,Mitral annular plane systolic excursion (MAPSE).

RVDD and RVSD together was present in 45.31% of hypertensive participants while RVSD was present in only 8.33% without RVDD, and this relationship was statistically significant(p= <0.001). The RV wall is thinner than the LV wall and with reduced pressures',thus a change in pressure due to RVDD can eventually affect the RV systolic function as its pressure adaptation is poor and vice versa. Both constitute part of the effect of hypertensive heart disease. This is also seen on the left where LVDD and LVSD coexist in subjects with HBP.23 From literature,there is no perfect explanation why RVSD and RVDD coexist in the setting of HBP but it is thought to be part of the spectrum of hypertensive heart disease.

Diastolic and systolic dysfunction are inter-related in HHD.A study by Akintunde et al discovered that most of the hypertensive participants with systolic dysfunction often have associated diastolic dysfunction.24 Achieving good BP control causes improvement in systolic dysfunction but lesser effect on diastolic dysfunction.

CONCLUSION:

The RV is now no longer seen as a bystander in the setting of HBP. The changes of HBP on the LV to an extent are mirrored on the RV

The technicalities in assessing the RV via Echo was a limitation to this study as not every participant had a clear Echo window for the RV

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TABLES AND FIGURES

TABLE 1: Indices on Echo

	Cases	Controls Meant SD	t-test	p-value
LVEDD	5.64±5.46	4.75±0.50	1.60	0.113
LVESD	3.58±2.34	2.98±0.47	2.48	0.015
LVPW	1.17±0.28	1.00±0.18	4.91	<0.001
LVMI	106.31±40.74	77.27±18.61	6.19	<0.001
EF	61.64±16.60	66.25±8.01	-2.62	0.010
IVS	1.24±0.27	1.00±0.18	7.38	<0.001
RVWT	0.53±0.09	0.51±0.09	1.66	0.090
TAPSE	2.00±0.53	2.27±0.46	-3.83	<0.001
RIMP	0.43±0.12	0.36±0.06	1.25	0.214
RV BASAL DIAMETER	2.54±0.22	2.43±0.15	5.11	<0.001

KEY:

LVEDD:Left Ventricular Diastolic Diameter

LVESD: Left Ventricular End Systolic Diameter

LVPW: Left Ventricular Posterior Wall

LVMI: Left Ventricular Mass Index

RVWT: Right Ventricular Wall Thickness

RIMP: Right Ventricular Index of Myocardial Performance

IVS: Intraventricular Septum

RV: Right Ventricle

TAPSE: Tricuspid Annular Plane Systolic Excursion

EF: Ejection Fraction

TABLE 2: Abnormalities on Echo

Variables	Cases n=100(%)	Controls n=100(%)	x	p
Left Ventricular Hypertrophy	50	10	38.10	<0.001
Right Ventricular Hypertrophy	79	77	0.12	0.733
Right Ventricular Dilatation	13	0	13.90	0.001
Right Ventricular Systolic Dysfunction	32	2	31.89	<0.001
Tei's Index	64	18	41.08	<0.001
Right Ventricular Diastolic Dysfunction	64	15	50.24	<0.001
Left Ventricular Diastolic Dysfunction	71	39	20.69	<0.001
Left Ventricular systolic dysfunction	19	1	18	<0.001