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RESEARCH ARTICLE

PREVALENCE, CLINICAL PRESENTATION AND RELATED RISK FACTORS FOR LEFT VENTRICULAR HYPERTROPHY IN HYPERTENSIVE PATIENTS RECEIVING CARE AT MNAZI MMOJA HOSPITAL HYPERTENSIVE CLINIC IN ZANZIBAR-TANZANIA

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ABSTRACT

Hypertension is a widespread global health problem. It has been the main cause of morbidity and mortality. With the concept of disease transition in Africa, we are moving from an era of communicable diseases to an era of non-communicable diseases such as Hypertension. In sub-Saharan Africa (SSA), patients with hypertension frequently present late to health facility often with complications. Moreover, the fact that hypertension is asymptomatic, patients tend to have poor adherence to medications which results to uncontrolled hypertension. Uncontrolled hypertension will eventually result into progressive damage to target organs such has the heart and kidneys. Chronic elevation of blood pressure (BP) leads to progressive development of left ventricular hypertrophy (LVH) with consequent cardiac remodelling, heart failure (HF) and increased risk of cardiovascular events. The burden of LVH together with the factors associated with its progression has not been well studied. This study aimed to investigate the prevalence, pattern and associated factors for LVH among patients attending MMH Zanzibar. In this current study we found among 389 hypertensive patients 241 (62%) patients had abnormal LV geometry, 55% had concentric hypertrophy. We also found sex, employment, domicile, duration of hypertension, BMI, and urea levels were having the positive value predictors of LVH.

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INTRODUCTION

Hypertension is a widespread, yet preventable, and costly global health problem. Zanzibar, like other developing countries is undergoing rapid demographic and epidemiological transition of disease pattern characterized by the ageing population and the rising trends in major risk factors for chronic, non-communicable diseases. Despite the scarce data on hypertension in Zanzibar, anecdotal evidence indicates an alarming increase in disease burden and associated morbidities, which often results in physical incapacitation or premature deaths. World Health Organization (WHO) define hypertension as a chronic medical condition in which arterial BP is elevated ≥140mmHg systolic and/or 90mmHg on two reading taken apart or a reported diagnosis of hypertension and treatment with recognized anti hypertensive within two weeks before the visit. Chronic elevation of BP causes silent maladaptive abnormalities to the heart that result in changes including geometrical re orientation in different pattern notably concentric remodeling, concentric hypertrophy and eccentric hypertrophy. The characteristic of the pattern is determined by the underlying stressor either pressure overload or volume overload. The Framingham Heart Study pointed out LVH as one of several risk factors for the development of unwanted cardiovascular events (Levy, 1988; Tsao, ?; Krumholz, 1995 and Levy, 1990). Hypertension attributed to the ageing populations together with rising risk factors (Beaglehole, 2011). Hypertension precedes HF in 75% of the cases and the latter accounts for 7% of death in United States (USA) (Manickavasagam, 2009). The current data on hypertension in

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Africa are based on unrepresentative surveys and population disease modeling estimates. However, the available evidence indicates that patients present late to health facilities often with complications or dies outside hospitals (Muna, 2013 and Addo, 2007). In Sub Saharan Africa (SSA) many patients with hypertension are undiagnosed, and those diagnosed are often not on regular medications or have sub-optimal control of BP, and many deaths occur outside hospitals (Edwards, 2000). Not only that but also because of pills burden, prescription drug costs, medication side effects insufficient time for patients education contribute to medication non-adherence. Data on burden of hypertension are scarce in most countries in SSA. However, anecdotal evidence indicates an alarming increase in hypertension and associated morbidities, which in many instances results in physical incapacitation or premature deaths (Muna, 2013). The prevalence of hypertension in SSA reveals more burden in urban than rural areas (Addo, 2007). A survey in Tanzania by Edwards *et al*. demonstrated the prevalence of hypertension to range between 28% and 32% and, of these, less than 20% were aware of their diagnosis, about 10% reported receiving treatment and less than 1% had their BPs controlled (Edwards, 2000). The prevalence of LVH varies across countries and populations partly due to different levels of health care delivery, distribution of associated determinants and risk factors as well as the criteria used. For instance, a review by Cuspid *et al* revealed the prevalence of LVH to range from 36% (conservative criteria) to 41% (less conservative criteria) in a pooled population (Cuspidi, 2012). Moreover, a study by Jaleta GN *et al* reported the prevalence of LVH to be 52% among hypertensive patients in Nigeria (Gari Negeri Jaleta, 2014). Likewise, in Tanzania, a study conducted by Chillo P *et al* among treatment-naïve hypertensive patients showed the prevalence of abnormal LV geometry to be 62% (Chillo, 2012). There are two major categories of systemic hypertension, namely primary or essential or idiopathic hypertension without identifiable cause which accounts for over 95% of patients and secondary hypertension which often results from pre-existing identifiable cause. The common causes for the latter include renal disorders e.g. chronic kidney disease (CKD), renal artery stenosis and a variety of endocrine disorders e.g. pheochromocytoma.

The current demographic and epidemiological transition in the pattern of disease characterized with ageing population and rising trends in major risk factors is the most important explanation for increasing burden of hypertension and other chronic non-communicable diseases in SSA (Beaglehole, 2011; WHO 2013). Besides age, other major risk factors for developing hypertension includes excessive dietary intake of high-fat, high-calories, low-fibre food stuff and obesity, sedentary lifestyle and lack of physical exercise, excessive consumption of alcohol together with smoking and tobacco use (Beaglehole, 2011; WHO, 2013) . In developed countries, poor people are at increased risk of developing hypertension and its complication attributed to lack of access to, and affordability of healthcare services and increased exposure to risk factors (Artham, 2009). Noteworthy, in the African context, economically transitioning high income earners are equally affected by hypertension due to increased exposure to risk factors due to increased access and affordability of sedentary lifestyle and dietary products. In addition, unexplored genetic risk factors could be contributing to the emerging hypertension in SSA. Comparative studies in the USA indicate that African Americans have an increased risk of developing hypertension, often at a younger age and usually suffer more severe forms of complications including stroke (Addo, 2007; Bloch, 2009; Tocci, 2008). In SSA, patients with stroke are relatively non-obese, typically with a history of undiagnosed or poorly controlled hypertension; with similar observation in Tanzania (Walker, 2000; Walker, 1995). Paradoxically, in certain part of SSA, obesity is perceived as a status symbol of affluence, happiness, beauty and good health rather than a problem. Tsioufis, C. *et al*. found LVH to be present in 38.3% of patients whereas normal geometry (LV-NG), concentric remodelling (LV-CR), concentric hypertrophy (LV-CH) and eccentric hypertrophy (LV-EH) represented 34.5, 27.1, 25.7 and 12.7%, respectively (Tsioufis, 2009). A study in Tanzania by Silangei LK *et al* showed the distribution of LV geometrical patterns to be 19.8%, 28.2% and 22% for CR, CH and EH respectively (Lairumbe Korduni Silangei, 2012).Study done by Chillo, P.*et al* found that 62.1 % of the patients had abnormal LV geometry, predominately LV hypertrophy(24.2% eccentric and 28.6% concentric LV hypertrophy) (Cohn, 2000).

Cohn J *et al* define cardiac remodelling as structural alteration and function in the heart in the response of hemodynamic load and/cardiac injury in association with neurohormonal activation. Remodelling may be described as physiological or pathological, alternatively may be classified as adaptive or maladaptive (Cohn, 2000). Adaptive remodelling is a compensatory change in dimensions and function of the heart in response to physiologic stimuli (Addo, 2007). Pathological remodelling may occur with pressure overload (aortic stenosis, hypertension), volume overload (valvular regurgitation) or as the result of cardiac injury (myocardial infarction, myocarditis).In each of these settings remodelling may transition from an apparently compensatory process to a maladaptive one (Cohn, 2000). Concentric hypertrophy (CH) is geometrical pattern characterized by thickening of the individual cardiomyocytes with parallel increase in sarcomere. CHis the most common type of LVH in hypertensive patients, and an independent determinant or risk factor for cardiovascular disease (Dobrowolski, 2014). A study by Cuspid *et al* revealed a higher BP levels to be associated with CH compared with other types of cardiac geometry among hypertensive patients (Cuspidi, 2012). Eccentric hypertrophy (EH) is the form of LVH in which both ventricular wall mass and chamber volume are increased together with serial arrays of sarcomeres. EH poses a great risk of coronary heart disease (Zabalgoitia, 1998).

Chronic uncontrolled systemic hypertension imposes greater total peripheral resistance on the vasculature. This increased pressure overload leads to progressive alterations of cardiac structural geometry (often concentric cardiac remodelling) characterized by LVH and septal hypertrophy. These pathological changes are the nidus for progressive myocardial fibrosis, impaired diastolic filling without systolic dysfunction (Gradman, 2009). LVH is currently considered as a marker for target organ damage in patients with systemic hypertension (Cuspidi, ? and Cuspidi, 2012). This is particularly common in SSA where patients with hypertension often present to health facilities at late stages of disease. For instance, a study by Chillo, P *et al*. revealed a prevalence of 62.1% of abnormal LV geometry in untreated hypertensive patients in Tanzania (Chillo, 2012) Cardiac remodelling results in altered geometry and ventricular functions (Zabalgoitia, 1998; Balci, 2002 and Takasaki, 2012). The magnetic resonance imaging (MRI) is more accurate and robust modality for evaluation of cardiac

geometry and ventricular hypertrophy (Subramaniam, 2009). However, ECG and echocardiography with 2D, M-mode and Doppler Imaging (DI) are routinely used in clinical practice with good results. The ventricular hypertrophy, but not geometry, was reported to be an important predictor for LA size (Tsioufis, 2009). A study by Baltabaeva, A. *et al*. reported that the differential regional geometrical remodelling occurs early in hypertension and can be predicted by sensitive markers e.g. longitudinal end-systolic strain (ESS) and peak systolic strain rate (SSR) (Baltabaeva, 2008). The use of DI in the assessment of LV longitudinal function plays an important role in identifying diastolic dysfunction and asymptomatic LV systolic dysfunction in hypertensive patients. For instance, based on peak systolic velocity <6.1 cm/s as a cut-off value for abnormal velocity, Nishikage, T. *et al* found 10% of asymptomatic hypertensive patients and 53% of diastolic HF to have impaired LV longitudinal systolic velocity. In these patients, systolic annular velocities were independently predicted by early and late peak diastolic annular velocities, female gender and deceleration time of the E wave velocity (Nishikage, 2008).

In hypertensive patients, the alterations in the LA structure and function are early predictors for development of LVDD and LVSD respectively in pre-clinical stages (Miyoshi, 2013). This progressive LVH leads to increased ventricular wall mass and ventricular wall stiffness thereby resulting in alterations of myocardial structure and perturbed LV functions. The resultant myocardial hypertrophy leads to progressive maladaptive compensatory mechanisms characterized by increased myocardial oxygen consumption and mismatch epicardial coronary perfusion. Similarly, it is speculated that alterations in nitric oxide (NO)-related endothelial dysfunction which occurs early in hypertension contributes to the development of ischaemic sub-endomyocardial fibrosis occurring secondary to cardiac hypertrophy (Lapu-Bula, 2007). The resulting tissue ischemia and cellular hypoxia triggers a range of cellular mechanisms characterized by apoptotic cell death, aberrant healing with consequent myocardial fibrosis and remodelling, abnormalities in long-axis function and torsion. The admixture of these cardiac maladaptive changes results in the abnormalities, which accounts for diastolic and systolic HF. The two clinical syndromes are considered to represent alternative patterns of same clinical disease of varying degree in chronic myocardial injury and ventricular remodelling (Yip, 2009). However, it is very evident that about 50% of patients with systemic hypertension develop left ventricular diastolic dysfunction (LVDD); alias heart failure with preserved systolic function or normal ejection fraction (HFPEF) or diastolic HF (Manickavasagam, 2009; Gradman, 2009; Wilson, 2009; Verma, 2009; Susic, 2008). LVDD is often classified as secondary diastolic HF in contrast to primary diastolic HF which occur as a result of infiltrative or restrictive cardiomyopathy (Desai, 2008). The observed sympathetic overdrive in diastolic dysfunction is believed to contribute to poor outcome (Grassi, 2009). Though BP is a continuous variable, the Eighth Report of the Joint National Committee (JNC8) classifies elevated BP and hypertension into operational and risk stratification categories (Chobanian, 2003). The goal of treatment of hypertension is achieving optimal BP; setting target BP as ≤140/90 mmHg for uncomplicated hypertension and generally ≤130/80 mmHg for patients with compelling indications (Chobanian, 2008). Therefore, early identification of hypertensive patients with asymptomatic LVD is a prudent strategy for prevention of HF.

Cardiovascular diseases (CVD) are widespread, yet preventable, and costly global health problem. Zanzibar-Tanzania, like other developing countries is undergoing rapid demographic and epidemiological transition of disease pattern characterized by the ageing population and the rising trends in major risk factors for non-communicable diseases. Development of LVH is a major maladaptive response and important risk factor in patients with hypertension. The risk of cardiovascular morbidity and mortality is two to four fold compared to patients with normal left ventricular mass, mainly due to complications of LVH inclusive of atrial fibrillation, systolic and diastolic heart failure, angina pectoris and sudden death. Despite hypertension being the commonest etiology for CVD; the magnitude, clinical presentation and related risk factors for LVH are not well characterized in Zanzibar-Tanzania. LVH is a marker of hypertensive related-target end organ damage, among hypertensive patients, LVH is clinically relevant because it represents an integrated marker of cardiovascular (CV) risk and reflects the long-term effects on the heart of hemodynamic and non-hemodynamic factors operating in hypertension. CV risks increases with increasing left ventricular mass and decreases with regression of LVH in response to antihypertensive therapy. Thus, detection of LVH and geometrical patterns in hypertensive patients who would be classified as at low or moderate CV risk, if assessed by routine procedures, is crucial for stratifying total CV risk and for grading the therapeutic approach according to absolute risk (Cuspidi, 2012). This study was aimed to assess the magnitude of LVH and its associated factors in hypertensive patients attended at MMH. The results of this study hope to not only raise awareness on the practitioners attending such patients but also raise the index of suspicion for early diagnosis of LVH in hypertensive patients in resource limited areas.

MATERIALS AND METHODS

This is cross-sectional observational prospective hospital based analytical study was conducted between June 2018 to Dec 2018 a National Hospital at (MMH), Zanzibar, Tanzania.MMH is the largest commercial hub in Zanzibar with the current population of about 1.5 million people projected to reach 2.12 million by 2020 (PHC, 2012). Adult patients aged 18 years and above with systemic arterial hypertension receiving care at MMH and who have fulfilled the inclusion criteria were recruited into the study. The actual sample size for the study was determined using formula for single population proportion by assuming 5% marginal error (ε), 95% confidence interval. In estimation of the sample size I utilize the findings from a similar study conducted by Jaleta GN *et al* which revealed 52% prevalence rate of LVH in hypertensive patients in Nigeria (Gari Negeri Jaleta, 2014). Data was obtained by a structured questionnaire, which will include demographic data pertaining the current age, gender, level of education and occupation and history of alcohol intake and cigarette smoking together with other hypertensive variables including duration of the illness, drugs and compliance to the medication. Detailed clinical physical examination was done by investigator including anthropometric measurements (weight and height). The weight was measured using a SECCA weighing scale after the subject had removed his/her coat, jacket and left with light clothing. Weight was recorded to the nearest 0.5kg. Height was measured after the individual had removed his shoes and or cap and recorded to nearest0.5 centimeters. Body mass Index (BMI) was then calculated for each individual by using the formula BMI=(kg)/(height in

m²).The BMI was classified according to World Health Organization into: Underweight: BMI< 18.5; Normal weight: BMI 18.5-24.9; Overweight: BMI 25.0-29.9; Obesity: BMI>30,Blood Pressure (BP).

After a 5-minutes rest, the patient's pulse rate and BP were measured with a standard manual mercury sphygmomanometer while in the sitting position. The systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements were determined by the 1st and 5th Korotokoffs sounds respectively. Two such readings were taken from the top of the meniscus and expressed to the nearest 2mmHg. The mean of the two readings of blood pressure was used for the final record. Hypertension was defined as systolic blood pressure (SBP) of more or equal to 140mmHg and/or diastolic blood pressure (DBP) of more or equal to 90mmHg or known hypertensive on treatment. A venepuncture at the antecubital area was performed to draw about 10mls of blood for biochemical analysis including random blood sugar, urea and creatinine. Blood was kept in appropriate vacutainer bottles and transported within 60 minutes to the laboratory, where all samples were processed. Standard operating procedures (SOP's) for MMH-Laboratory were observed at all levels from sample collection to sample analysis. Patients with investigations carried at MMH-Laboratory within 7 days prior recruitment were not re-investigated; instead their results were extracted from their record files and regarded as valid. A standard 12 lead ECG was done by a technician at MMH. The investigator interpreted all ECGs and the findings were verified by a Cardiologist. The assessment of LVH was based on Voltage criteria, which consist of S wave in V1 plus an R wave in V5 or V6 of greater than 3.5mV or an R wave in V5 or V6 of greater than 2.6mV or R wave in V6 > V5,R wave on AVL >1.3mV.

All patients had an echocardiograms performed by a Cardiologist. Patients were positioned in left lateral decubitus using SIEMENS ACUSON P300 machine with a 3.5MHz transducer using standardized echocardiographic protocols. Structural and functional evaluation using 2D; M mode echocardiography and Doppler studies was recorded .An average of 3 values/readings were taken as per the American Society of Echocardiography (ASE) recommendations. Left ventricular mass were evaluated using the formula below: Left Ventricular Mass=0.8(1.04[LVIDd+LVPWd+IVSd)3 – (LVIDd)3] +0.6.Where by:-LVPWd; posterior wall thickness at end of diastole, IVSd; septal wall thickness at end of diastole, and LVIDd; dimension of left ventricles at end of diastole. Gender-specific and indexation of LVM was used to diagnose LVH using the following defining criteria: >115g/m^2 and >95g/m^2 for men and women respectively when LVM was indexed to body surface area. Data were entered on pre-programmed excel spread sheet followed by importing into SPSS statistical package version 18.0 for Windows (SPSS Inc, Chicago, Illinois, USA) for analysis. The appropriate frequencies and tables were created for calculations of appropriate means and proportions. The Chi-square or Fisher's exact tests were used to detect differences in the frequencies of categorical characteristics between the groups. The continuous variables were analysed by Student t-test. The effect of specific variables on the study endpoints was assessed by using a logistic regression model with outcome variable LVH as Yes or No. A p value of < 5% indicated statistical significance.
Ethical clearance and approval was sought from ZANZIBAR Institutional Review Medical Board. Further approval and permissions to conduct the study was sought from MMH. No biological samples were collected. Confidentiality was ensured. All data and research tools including datasets were anonymous. The data were stored in electronic format in a computer with secured access. The study protocol was conducted in accordance to the Helsinki Declaration Research Ethics Guidelines.

RESULTS

Among 389 hypertensive patients attending the MMH outpatient clinic of these, 219 (56.3%) were females and males constituted 43.7% of the study participants. The mean age was 58.8 and over 60% of participants were aged above 55 years. Majority of participants (60%) were married and 44.2% had an education level of primary school. Nearly 21% of participants were jobless, 24.7% were retired, and 54.5% had a current regular income generating activity. Over 88% of the participants resided in Zanzibar town and about 12% came from upcountry. Almost 60% of participants were physically inactive and 78% of participants were either overweight or obese. About 18% of participants had a positive smoking history while and 50% were regular alcohol users. 58.6% of participants had hypertension for over 2 years and 84% were on current use of anti-hypertensive medications. We performed multivariate logistic regression analyses to determine factors associated with LVH. On bivariate analysis of 15 potential sociodemographic and clinical characteristics, 9 characteristics including; sex, employment, domicile, physical activity, duration of hypertension, treatment of hypertension, BMI, urea and creatinine levels proved to be significantly associated with LVH, Table 2.

Table 1. Socio-demographic characteristics of Participants N=389

	Variable	Observations (n)	Percentage (%)
Sex	Male	170	43.7
	female	219	56.3
Age group	<=45	65	16.7
	46-55	87	22.4
	56-65	113	29.0
	66+	124	31.9
Marital status	single	22	5.6
	married	234	60.2
	separated	133	34.2
Education level	Primary and below	172	44.2
	secondary	146	37.5
	Post-secondary	71	18.3
Employment	Not employed	81	20.8
	Employed	76	19.5
	Self employed	136	35.0
	Retired	96	24.7
Domicile	Urban	343	88.2
	Rural	46	11.8

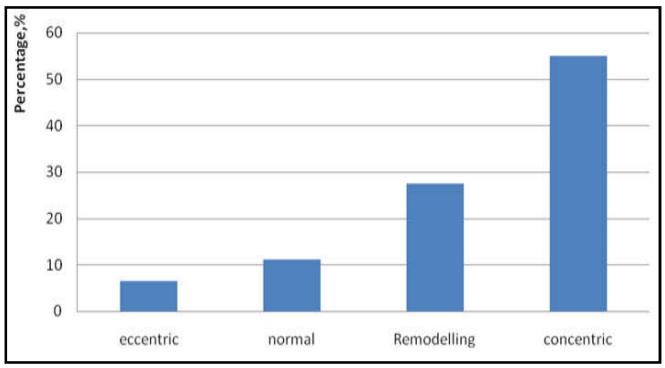

Figure 2. Chart showing the pattern of LVH among hypertensive patients attending clinic at MMH

Table 2. Factors associated with LVH among hypertensive patients receiving care at JKCIN=389, multivariate logistic regression

Factors	N	LVH patients n (%)	OR(95%CI)	P-value	AOR(95%CI)	p-value
Sex						
Male	170	94(55.3)	ref			
Female	219	147(67.1)	1.7(1.1-2.5)	0.02	1.7(1.1-2.7)	0.02
Age group						
<=45	65	38(58.5)	ref			
46-55	87	58(66.7)	1.4(0.7-2.8)	0.30		
56-65	113	62(54.9)	0.9(0.5-1.6)	0.64		
66+	124	83(66.9)	1.4(0.8-2.7)	0.25		
Marital status						
Single	22	17(77.3)	2.0(0.7-5.9)	0.18		
Married	234	141(60.3)	0.9(0.6-1.4)	0.68		
Separated	133	83(62.4)	ref			
Education level						
Primary and below	172	110(64.0)	ref			
Secondary	146	89(60.9)	0.9(0.6-1.4)	0.58		
Post-secondary	71	42(59.2)	0.8(0.5-1.4)	0.48		
Employment status						
Employed	76	33(43.4)	ref			
Not Employed	81	58(71.6)	3.3(1.7-6.4)	0.00	3.2(1.6-6.4)	<0.001
Self employed	136	91(66.9)	2.6(1.5-4.7)	0.001	2.7(1.5-4.9)	0.02
Retired	96	56(61.5)	2.1(1.1-3.8)	0.02	2.2(1.2-4.2)	0.01
Domicile						
Urban	343	204(59.5)	ref			
Rural	46	37(80.4)	2.8(1.3-5.9)	0.01	3.1(1.4-6.8)	<0.001
Smoking						
Yes	69	46(66.7)	1.3(0.7-2.2)	0.37		
No	320	195(60.9)	ref			
Alcohol						
No	193	114(59.1)				
Yes	196	127(64.8)	1.3(0.8-1.9)	0.24		
Exercise						
Yes	156	55(54.5)	ref			
No	233	156(66.9)	1.7(1.1-2.6)	0.01	1.5(1.0-2.4)	0.07
HTN medication						
Yes	327	213(65.1)	2.3(1.3-3.9)	0.00	1.8(1.0-3.3)	0.06
No	62	28(45.2)	ref			
HTN duration						
>2 years	228	153(67.11))	1.7(1.1-2.6)	0.01	1.6(1.0-2.5)	0.04
<=2 years	161	88(54.7)	ref			
Glucose level						
Normal	309	188(60.80	ref			
Pre diabetic	33	23(69.7)	1.5(0.7-3.2)	0.32		
Diabetic	47	30(63.8)	1.1(0.6-2.1)	0.69		
BMI						
Normal	86	40(46.5)	ref			
Over weight	119	77(64.7)	2.1(1.2-3.7)	0.01	1.8(1.0-3.3)	0.05
Obese	184	124(67.4)	2.4(1.4-4.0)	0.00	2.0(1.1-3.4)	0.02
Urea						
Normal	333	193(57.9)	ref			
Abnormal	56	48(85.7)	4.4(2.0-9.5)	0.00	4.3(1.9-9.5)	<0.001
Creatinine						
Normal	302	177(58.6)				
Abnormal	87	64(73.6)	1.9(1.2-3.3)	0.01	1.2(0.8-2.2)	0.11

Six of the nine characteristics (i.e. sex, employment, domicile, duration of hypertension, BMI, and urea levels) remained significant associated factors even after multivariate analyses, Table 2. Female sex showed a 70% increased risk for LVH compared to males (AOR 1.7, 95% CI 1.1-2.7, P = 0.02). Regarding employment status, unemployed participants displayed the highest risk for LVH (i.e. 3-fold) compared to their employed counterparts (AOR 3.2, 95% CI 1.6-6.4, P<0.001). Furthermore, rural inhabitants displayed a 3 times odds for LVH compared to those who resided in urban areas (AOR 3.1, 95% CI 1.4-6.8, P<0.001). Longer duration of hypertension was associated with a 60% increased risk of hypertension (AOR 1.6, 95% CI 1.0-2.5, P=0.04). Increasing BMI was also associated with increased risk for LVH with obesity having the highest risk (AOR 2.0, 95% CI 1.1-3.4, P=0.02). High urea levels was found to be associated with a 4 times risk for LVH (AOR 4.3, 95% CI 1.9-9.5, P<0.001).The results are summarized in the tables and figures.

DISCUSSION

In this present study we found the prevalence of abnormal LV geometry to be 62%. These findings are in consonance with a number of previous studies. For instance a study done by Silangei *et al* (Lairumbe Korduni Silangei, 2012) in the same setting in 2012 found a prevalence of 70%. Similar findings were also echoed by studies by Aje *et al* (Aje, 2006) and Akintunde *et al* (Akintunde, 2010), which both observed prevalence of 72%. However, some studies have shown lower prevalence. In a study by Li *et al* (Li, 2014), the prevalence of LVH among untreated hypertensive patients was 20.2%. Furthermore a systematic review (Karaye *et al*) of 10 articles involving a total of 1722 hypertensive patients revealed an LVH prevalence of 36.4%. This variability in the prevalence of abnormal LV geometry could be attributed by the different thresholds used to define abnormality among studies. With regards to the pattern of LV geometry, concentric hypertrophy

(55%) followed by concentric remodelling (27.5%) were the commonest. This pattern is similar to Silangei study (Lairumbe Korduni Silangei, 2012), however the prevalence were lower, i.e. 28.2% concentric hypertrophy and 19.8% concentric remodelling. The pattern in Aje *et al* (2006), study is also similar with concentric hypertrophy (28%) and concentric remodelling (26%) as the commonest LV abnormalities. Several other studies have displayed a different pattern to ours. For instance in the Akintunde *et al* [43] study, concentric hypertrophy was the commonest pattern (60.1%). Similarly, in the Li *et al* (2014), study, concentric remodelling was seen in 34.9% followed by concentric hypertrophy in about 11% of participants. Furthermore, in the systematic review by Karaye *et al* (KARAYE, 2013), concentric remodelling predominated (28.3%) followed by concentric hypertrophy (18.4%). The pattern differences among studies could largely be a result of variations in participants characteristics mostly ethnicity and duration of hypertension and medications used. Several associated factors for abnormal LV geometry have been documented in the literature. In our study, female sex, unemployed status, rural residence, longer duration (>2 years) of hypertension, obesity, and elevated urea levels were found to be significant predictors after multivariate logistic regression analysis. Silangei (2012) and Akintunde (2010) studies also found duration of hypertension to be a strong associated factor for abnormal LV geometry. Similar to our findings, the Li study (Li h, 2014), also found that female sex and increased BMI were associated with LV geometry abnormality.

Conclusion

The prevalence of LV geometry abnormality was high in this study which was 62%. Female sex, unemployment, rural inhabitants, and hypertension duration of >2 years, obesity and elevated urea level in hypertensive patients were associated with increases risk for LVH. Since not all patients with hypertension develop left ventricular hypertrophy, there are clinical findings that should be kept in mind that may alert the physician to the presence of left ventricular hypertrophy these including, palpitation, chest pain/angina equivalent and fatigue

REFERENCES

Addo, J., L. Smeeth, and D.A. Leon, Hypertension in sub-saharan Africa: a systematic review. Hypertension, 2007. 50(6): p. 1012-8.

Aje A, Adebiyi AA, Oladapo OO, Dada A, Ogah OS, Ojji DB, *et al*: Left ventricular geometric patterns in newly presenting Nigerian hypertensives: an echocardiographic study. *BMC Cardiovasc Disord.* 2006, 6: 4-10.1186/1471-2261-6-4.

Akintunde A, Akinwusi O, Opadijo G: Left ventricular hypertrophy, geometric patterns and clinical correlates among treated hypertensive Nigerians. *Pan Afr Med J.* 2010, 4: 8

Artham, S.M., *et al*., Obesity and hypertension, heart failure, and coronary heart disease-risk factor, paradox, and recommendations for weight loss. *Ochsner J,* 2009. 9(3): p. 124-32.

Balci, B. and O. Yilmaz, Influence of left ventricular geometry on regional systolic and diastolic function in patients with essential hypertension. *Scand Cardiovasc J*, 2002. 36(5): p. 292-6.

Baltabaeva, A., *et al*., Regional left ventricular deformation and geometry analysis provides insights in myocardial remodelling in mild to moderate hypertension. *Eur J Echocardiogr,* 2008. 9(4): p. 501-8.

Beaglehole R, Priority actions for the non-communicable disease crisis. Lancet 2011. Lancet, 2011.

Bloch, M.J. and J.N. Basile, Heart failure in people younger than 50 years is more common in black than white Americans and is closely associated with poorly treated and controlled hypertension: the CARDIA study. *J Clin Hypertens (Greenwich),* 2009. 11(7): p. 388-90.

Chillo, P., *et al*., Prevalence and covariates of abnormal left ventricular geometry in never-treated hypertensive patients in Tanzania. *Blood Press.* 23(1): p. 31-8.

Chobanian, A.V., *et al*., The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA, 2003. 289(19): p. 2560-72.

Cohn JN, F.R., Sharpe N., Cardiac remodeling--concepts and clinical implications: a consensus paper from an international forum on cardiac remodeling. Behalf of an International Forum on Cardiac Remodeling. *J Am Coll Cardiol,* 2000. 35(569).

Cuspidi C, S.C., Negri F, Mencia G, Morganti A., Prevalence of left ventricular hypertrophy in hypertensive patients: an updated review of echocardiographic studies. *J Hum Hypertens,* 2012. 26(6): p. 343 - 9.

Cuspidi, C., *et al*., Left ventricular geometry, ambulatory blood pressure and extra-cardiac organ damage in untreated essential hypertension. *Blood Press Monit.* 15(3): p. 124-31.

Cuspidi, C., M. Tadic, and C. Sala, A New Electrocardiographic Marker of Hypertensive Cardiac Damage. J Clin Hypertens (Greenwich). 17(6): p. 450-2.

Desai, A. and J.C. Fang, Heart failure with preserved ejection fraction: hypertension, diabetes, obesity/sleep apnea, and hypertrophic and infiltrative cardiomyopathy. *Heart Fail Clin,* 2008. 4(1): p. 87-97.

Dobrowolski, P., *et al*., Independent association of obstructive sleep apnea with left ventricular geometry and systolic function in resistant hypertension: the RESIST-POL study. Sleep Med. 15(11): p. 1302-8.

Edwards, R., *et al*., Hypertension prevalence and care in an urban and rural area of Tanzania. *J Hypertens,* 2000. 18(2): p. 145-52.

Gari Negeri Jaleta, E.K.G., Wondim Getinet, Left ventricular hypertrophy among black hypertensive patients focusing on the efficacy of angiotension converting enzyme inhibitors. BMC Res Notes, 2014.

Gradman, A.H. and J.T. Wilson, Hypertension and diastolic heart failure. *Curr Cardiol Rep*, 2009. 11(6): p. 422-9.

Grassi, G., *et al*., Sympathetic and baroreflex cardiovascular control in hypertension-related left ventricular dysfunction. Hypertension, 2009. 53(2): p. 205-9.

KARAYE K and HABIB AG. Left ventricular geometric patterns in hypertensive Nigerians: a systematic review. International Cardiovascular Forum Journal, [S.l.], v. 1, n. 2, p. 84-87, dec. 2013.

Krumholz, H.M., M. Larson, and D. Levy, Prognosis of left ventricular geometric patterns in the Framingham Heart Study. *J Am Coll Cardiol,* 1995. 25(4): p. 879-84.

Lairumbe Korduni Silangei, V.P.M., Helmut Diefenthal, Gibson Kapanda, Mathew Dewhurst, Hery Mwandolela and Ben Hamel, Assessment of left ventricular geometrical

patterns and function among hypertensive patients at a tertiary hospital, Northern Tanzania BMC Cardiovasc Disord, 2012. 12(109).

Lapu-Bula, R. and E. Ofili, From hypertension to heart failure: role of nitric oxide-mediated endothelial dysfunction and emerging insights from myocardial contrast echocardiography. *Am J Cardiol,* 2007. 99(6B): p. 7D-14D.

Levy, D., *et al*., Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med,* 1990. 322(22): p. 1561-6.

Levy, D., Left ventricular hypertrophy. Epidemiological insights from the Framingham Heart Study. Drugs, 1988. 35 Suppl 5: p. 1-5.

Li h, pei f, Shao l, *et al*. prevalence and risk factors of abnormal left ventricular geometrical patterns in untreated hypertensive patients.bmc cardiovasc disord. 2014 oct 4;14:136.

Manickavasagam, S., *et al*., Management of hypertension in chronic heart failure. Expert Rev Cardiovasc Ther, 2009. 7(4): p. 423-33.

Miyoshi, H., *et al*., Early predictors of alterations in left atrial structure and function related to left ventricular dysfunction in asymptomatic patients with hypertension. J Am Soc Hypertens. 7(3): p. 206-15.

Muna, W.F., Comprehensive strategies for the prevention and control of diabetes and cardiovascular diseases in Africa: future directions. Prog Cardiovasc Dis. 56(3): p. 363-6.

Nishikage, T., *et al*., Subclinical left ventricular longitudinal systolic dysfunction in hypertension with no evidence of heart failure. Circ J, 2008. 72(2): p. 189-94.

PHC, Basic Demographic and Socio-Economic Profile: Dar es Salaam Region. 2012.

Subramaniam, V. and G.Y. Lip, Hypertension to heart failure: a pathophysiological spectrum relating blood pressure, drug treatments and stroke. Expert Rev Cardiovasc Ther, 2009. 7(6): p. 703-13.

Susic, D. and E.D. Frohlich, Optimal treatment of hypertension with diastolic heart failure. Heart Fail Clin, 2008. 4(1): p. 117-24.

Takasaki, K., *et al*., Left ventricular dysfunction assessed by cardiac time interval analysis among different geometric patterns in untreated hypertension. Circ J. 76(6): p. 1409-14.

Tocci, G., S. Sciarretta, and M. Volpe, Development of heart failure in recent hypertension trials. J Hypertens, 2008. 26(7): p. 1477-86.

Tsao, C.W., *et al*., Left Ventricular Structure and Risk of Cardiovascular Events: A Framingham Heart Study Cardiac Magnetic Resonance Study. J Am Heart Assoc. 4(9): p. e002188.

Tsioufis, C., *et al*., Left ventricular mass but not geometry determines left atrial size in the early stages of hypertension. J Hum Hypertens, 2009. 23(10): p. 674-9.

Verma, A. and S.D. Solomon, Diastolic dysfunction as a link between hypertension and heart failure. Med Clin North Am, 2009. 93(3): p. 647-64.

Walker, R.W. and D.G. McLarty, Hypertension and stroke in developing countries. Lancet, 1995. 346(8977): p. 778.

Walker, R.W., *et al*., Stroke mortality in urban and rural Tanzania. Adult Morbidity and Mortality Project. Lancet, 2000. 355(9216): p. 1684-7.

WHO, WHO Non-communicable Diseases and Mental Health; NCD Action Plan 2013-2020.

Wilson, R.M., *et al*., Effects of fixed-dose isosorbide dinitrate/hydralazine on diastolic function and exercise capacity in hypertension-induced diastolic heart failure. Hypertension, 2009. 54(3): p. 583-90.

Yip, G.W., *et al*., Hypertension and heart failure: a dysfunction of systole, diastole or both? J Hum Hypertens, 2009. 23(5): p. 295-306.

Zabalgoitia, M., *et al*., Comparison in systemic hypertension of left ventricular mass and geometry with systolic and diastolic function in patients <65 to > or = 65 years of age. *Am J Cardiol,* 1998. 82(5): p. 604-8.

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