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Determinants of Left Ventricular Hypertrophy in Hypertensive Patients seen in a Teaching Hospital in Ghana

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Abstract

Hypertensive heart disease presenting as left ventricular hypertrophy (LVH) is a common and potentially modifiable cardiovascular risk factor often overlooked in most sub-Saharan African countries including Ghana. This cardiac marker occurring in hypertension is very important because it affects the overall cardiovascular disease risk assessment and management. We sought to assess the prevalence and determinants of electrocardiographic left ventricular hypertrophy in patients with hypertension seen at the outpatient clinic of a Teaching Hospital in Ghana. A cross-sectional and prospective study was conducted on three hundred and fifty hypertensive patient at Komfo Anokye Teaching Hospital in Kumasi, Ghana. Following informed consent, a questionnaire was used to gather demographic, anthropometric and clinical details of patients. A standard resting 12-lead resting ECG was performed on all the study participants and Scott's criteria was used to determine LVH. Fischer's exact test for statistical significance at 95% confidence interval was used to evaluate associations between categorical variables. Various independent associations with LVH were also assessed using logistic regression analysis. P value of 0.05 was considered as statistically significant. The mean (± standard deviation) age of the patients was 59.65 ± 13.52 years. The mean systolic and diastolic blood pressures were 141.76 ± 20.26 mmHg, 84.28 ± 10.49 mmHg respectively; and the body mass index was 27.5 ± 6.09 kg/m^2. The prevalence of electrocardiographic left ventricular hypertrophy (ECG-LVH) among the hypertensive patients was 46.6%. When multiple logistic regression analysis was done, male gender (adjusted OR: 2.40, 95% CI 1.53-3.78, P=0.000), cigarette smoking (adjusted OR: 0.34, 95% CI 0.12-0.95, P=0.040) elevated SBP (adjusted OR: 1.79, 95% CI 1.09-2.93, P=0.200) and uncontrolled BP (adjusted OR: 1.86, 95% CI 1.15- 3.01, P=0.011) emerged as independent determinants of left ventricular hypertrophy.

In conclusion, LVH is a common pre-clinical cardiac complication in Ghanaian individuals with hypertension. Male gender, cigarette smoking, elevated systolic blood pressure and uncontrolled BP appear to be the main determinants of this important pre-clinical cardiac damage.

Keywords: Hypertension; Cardiovascular disease; Ghana; Electrocardiographic left ventricular hypertrophy

Introduction

The increasing prevalence of hypertension in sub-Sahara Africa has resulted in an increase in the burden of cardiovascular diseases in this region. With the limited resources, the sub-Saharan African region is already handling the burden of other conditions such as malaria, HIV/AIDs, maternal and infant mortal [1,2]. Hypertension remains a major and important public health challenge associated with considerable morbidity and mortality in most African countries including Ghana [2]. Uncontrolled hypertension usually results in cardiac remodelling leading to a variety of cardiac structural and functional changes, including left ventricular hypertrophy, left ventricular diastolic (initially) and systolic (later) dysfunction. There may be impairment of coronary reserve, arrhythmias, enlargement of left atrial and in extreme cases, overt heart failure [3,4]. Left ventricular hypertrophy in hypertension has been the focus of attention because of its detrimental contribution to cardiovascular disease morbidity and mortality [3].

Studies have shown that left ventricular hypertrophy is a common complication of hypertension, and a major risk factor for cardiovascular disease morbidity and mortality [3,4]. In view of this, management of hypertension has therefore advanced beyond just achieving blood pressure control, but also it involves early detection of hypertension and its associated complications, and appropriate interventions to reduce the associated high morbidity and mortality. It is therefore not surprising that, current guidelines for management of hypertension recommend that evidence of structural or functional hypertensive cardiac damage such as LVH is required in addition to blood pressure level to further define the severity of hypertension [5]. The time is rife to give the needed attention to this strong, important, independent cardiovascular risk factor in our clinical practice. Indeed, the clinical evaluation of LVH has been an important objective in cardiac electrophysiology. The importance of LVH increased in recent years with the discovery that LVH can be regressed or reversed with medical therapy, and that by doing so, heart failure and other cardiovascular diseases can be prevented or delayed [6].

LVH, being a recognized cardiac marker with an ominous prognostic sign, it is very alarming that its prevalence is on the increase, especially in developing countries such as Ghana. The prevalence of LVH has been shown by several studies to be high [7,8]. Even though different studies have demonstrated conflicting results on the prevalence of ECG-LVH hypertensive patients [9-11]. A study in

Eastern Sudan found ECG-LVH prevalence rate of 33.3% among hypertensive patients, while another study in Democratic Republic of Congo detected that 48% of patients with hypertension had ECG-LVH among [9-11]. In Nigeria, Ayodele et al. [12] reported that ECG-LVH was seen in 42.2% of hypertensives they studied. In another study, ECG-LVH was seen in 33.3% of Ghanaian civil servants with hypertension [9]. Owusu [13] also found in a study in Kumasi that 91.5% of patients with hypertensive heart failure had ECG-LVH. In a recent study among hypertensive patients attending a Specialist Clinic in Ghana, a prevalence rate of 39.0% was reported [14-19].

Uncontrolled hypertension is obviously the major stimulus to left ventricular hypertrophy [10-19]. Apart from the severity of the hypertension, other factors play a role in the onset and degree of progression of left ventricular hypertrophy. These factors which include gender, obesity, dyslipidaemia, type 2 diabetes mellitus, cigarette smoking and ageing. These factors have been demonstrated to be positively correlated with the prevalence of LVH in hypertensive patients [14-16]. These risk factors appear to be fundamental to the atherosclerotic process [17,18] and may affect the impact of hypertensive cardiovascular damage and thereby cardiovascular morbidity and mortality [19,20].

There are numerous studies and published guidelines from developed countries on the prevalence and risk factors of LVH in hypertensive patients [7,9,21]. Despite its importance, data on LVH among hypertensive is largely unavailable in Ghana. It is very essential that local data on this major cardiovascular risk factor be readily available so that early interventions could be provided in order to reduce morbidity and mortality of cardiovascular disease in sub-saharan Africa [21,22]. This study was therefore designed to assess and document the prevalence and determinants ECG-LVH in Ghanaian individuals with essential hypertension.

Methodology

Study design and population

This study was a hospital-based cross-sectional prospective carried out from February 2015 to June 2015. The study was done at the hypertension clinic of the Komfo Anokye Teaching Hospital (KATH), Kumasi, Ghana. Written informed consent was obtained from each study participant. Ethical approval was also obtained from the Committee on Human Research Publication and Ethics of School of Medical Sciences, the Kwame Nkrumah University of Science and Technology, Kumasi, Ghana. Three hundred and fifty (350) hypertensive patients were selected using systematic sampling technique. Data were collected with a standardized questionnaire which was prepared specifically for this study. Hypertensive patients aged eighteen years and above were included in the study. Excluded from the study were hypertensive patients with valvular heart disease, chronic kidney disease, left bundle branch block and chest wall abnormalities.

Sample size determination

Sample size was calculated using the formula approved by the world health organization [23].

$N = \frac{Z^2pq}{d^2}$ Where

N=the desired sample size

Z=the standard normal deviation, usually set at 1.96 which corresponds to the 95% confidence level

P=the proportion in the target population estimated to have a LVH

q=1.0- p

d=degree of accuracy desired, set at 0.05.

An average population prevalence of ECG LVH in hypertensive patients, from a community based study conducted in Accra, Ghana of 33.3% [9] was used to obtain sample size of 342 patients.

$$N = \frac{Z^2pq}{d^2}$$

$$N = \frac{1.96^2 \times 0.33 \times 0.667}{0.05^2}$$

N=342

Clinical Evaluation

Baseline clinical and demographic characteristics were obtained from each participant. The study tool captured demographic data, anthropometric data and clinical information of the study patients. Information on lifestyle habits (smoking, physical activity), personal medical history and use of medication were collected from all the participants. Smoking habit was classified into one of two categories-non-smokers: patients who had never smoked; and smokers: those who were currently smoking and had been smoking five cigarettes per day for the previous five years or more, or those who had stopped smoking less than five years ago. All the participants had the following measurements done: blood pressure, heart rate, weight, height, and waist and hip circumferences. Blood pressure was taken in a seated position and it was measured to the nearest 2 mmHg, on the left arm after five minutes' relaxation, using a standard mercury sphygmomanometer. The average of three readings was recorded. Hypertension was defined as the presence of a persistent elevated SBP ≥ 140mmHg and/or diastolic DBP ≥ 90mmHg, and/or the use of anti-hypertensive drugs and/or past medical history of hypertension [21,22]. Uncontrolled hypertension was defined as a blood pressure ≥ 140/90 mmHg or, if the subject had diabetes, ≥ 130/80 mmHg and were on antihypertensive medication [24]. Body weight and height were measured in erect position with participants wearing light clothes and without shoes Body mass index (BMI) was calculated from formula: BMI=weight in kg/ (height in m^2). By the World Health Organization (WHO, 2000) criteria, a BMI <18.5 kg/m^2 is considered underweight, 18.5-24.9 kg/m^2 ideal weight and 25-29.9 kg/m^2 overweight or pre-obese. When BMI was greater than 29.99 kg/m^2, patients were designated as obese. Hip and waist were measured to the nearest 1 cm and waist to-hip ratio (WHR) ≥ 1 defined central obesity.

A standard resting 12-lead resting ECG was performed on all the study participants, with a Standard Philips (PAGER) ECG machine. With the subject relaxed and comfortably lying on supine position, the electrodes were placed and effective skin contact was ensured during the ECG recording. The stylus control was set at 10 mm/mV and paper speed at 25 mm/s. It was regularly checked for any technical faults such as damping and electrical interference. The ECG tracings were analysed and interpreted using callipers. LVH was determined using Scott's criteria for LVH, which is provided below:

R in I added to S in III >25 mm

R in aVL >7.5 mm

R in aVF >20 mm

S in aVR >14 m

S in V1 (or V2) added to R in V5 (or V6) >35 mm

R in V5 or V6 >26 mm

R ± S in any precordial lead >45 mm

LVH was diagnosed if one of the above criteria was present on an ECG tracing [25].

Statistical Design and Analysis

Data from the questionnaire was entered into Microsoft Excel 2007 version. Data was edited to exclude errors, re-organized, and coded. Data was then exported to Strata version 13.0 for statistical analysis. Frequencies and percentages were calculated for categorical variables and a measure of central tendency such as median was used for continuous data. Odds Ratios (ORs) were calculated to measure the strength of associations. Stratified analysis was done to examine the various independent associations with ECG-LVH and effect of confounders. All analyses were done at 95% confidence intervals.

Firstly, univariate analysis was done and information obtained was used to prepared shell tables and graphs. Secondly, a bivariate analysis using Fischer's exact test was performed to examine the relationship between ECG-LVH and age, gender, cigarette smoking, SBP, DBP and uncontrolled BP, and comorbidities. This was followed by stratified analysis done to examine factors found to be statistically significant in the bivariate analysis to control for confounding factors. Stepwise logistic regression analysis was then conducted to determine measures of association of significant variables found in the bivariate analysis. We started with a single variable adding the other variables one by one at the 0.05 level (95% CI) eliminating all non-significant variables until all possible variables were added to identify determinants of ECG-LVH.

Results

Demographic and baseline characteristics of study participants

Table 1 shows the demographic and clinical characteristic of the hypertensive patients. A total of three hundred and seventy (370) hypertensive patients were selected for the study. Twenty (20) of the patients were dropped from the final analysis because of incomplete data. Data from three hundred and fifty (350) hypertensive patients were analysed (94.6%). There were one hundred and seventy five (175) men, constituting 50.0% of the patients studied. The mean (± standard deviation) age of the patients was 59.7 (± 13.5) years with a range of 30 to 89 years. Two hundred seventy-three (75.8%) of the patients were ≥ 50 years. The mean duration of hypertension among the study patients was 7 years (range 1 month-40 years). The mean systolic (SBP) and diastolic (DBP) blood pressures were 141.7 ± 620.26 mmHg, 84.28 ± 10.49 mmHg respectively; and the body mass index was 27.5 ± 66.09 kg/m^2. Regarding CVD risk factors; family history of hypertension was seen in 59.9% (195) of the patients, diabetes mellitus was seen 27.3% (95) of the patients, and obesity was seen in 27.3% (92) of the patients. Obesity was seen more in female patients than male patient and the difference was statistically significant. Male patients were more significantly prone to develop diabetes mellitus than the female patients, and the men were more likely to have a positive history of cigarette smoking. Sex and age distribution of the patients is shown in Figures 1 and 2.

Characteristic	Male, N=175, Mean ± SD	Female, N=175 Mean ± SD	P-value	Total, N=350 Mean ± SD
Age (Years)	61.68 ± 14.16	57.62 ± 12.57	0.002*	59.65 ± 13.52
SBP (mmHg)	141.93 ± 18.19	141.59 ± 22.18	0.439	141.76 ± 20.26
DBP (mmHg)	84.71 ± 10.27	83.84 ± 10.73	0.000*	84.28 ±10.49
Weight (kg)	72.17 ± 14.69	70.60 ± 16.00	0.179*	71.38 ± 16.00
Height (m)	1.65 ± 0.08	1.57 ± 0.09	0.000*	1.610 ± 0.09
BMI (kg/m^2)	26.71 ± 5.57	28.43 ± 6.48	0.005	27.56 ± 6.09
Level of Education				
Tertiary, n (%)	32 (18.71)	13 (7.47)	0.003*	45 (13.04)
Secondary, n (%)	70 (40.94)	62 (35.63)		132 (38.26)
Primary, n (%)	45 (26.32)	65 (37.36)		110 (31.88)
No Formal, n (%)	24 (14.04)	34 (19.54)		58 (16.81)
CVD Risk Factors				
Diabetes n (%)	62 (35.63)	33 (18.97)	0.000*	95 (27.30)
Dyslipidaemia, n (%)	21 (12.26)	20 (11.56)	0.599	41 (11.92)
Obesity, n (%)	36 (21.18)	56 (33.53)	0.037	92 (27.30)
FHH, n (%)	103 (59.54)	92 (54.12)	0.261	195 (56.85)
Cigarette Smoking, n (%)	19 (12.93)	1 (0.60)	0.000*	20 (6.39)

Table 1: Demographic and baseline characteristics of the hypertensive patients.

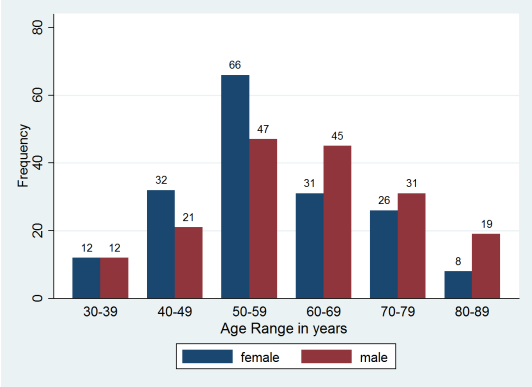

Figure 1: A graph showing the age and gender distribution of the study subjects.

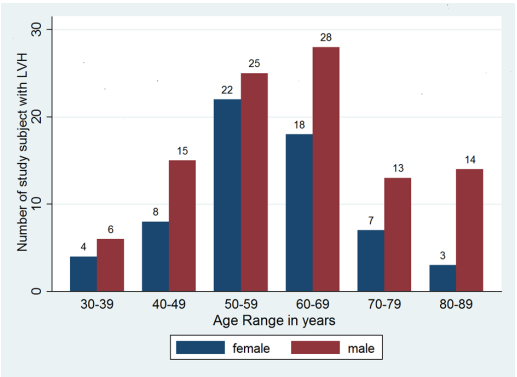

Figure 2: A graph showing the age and gender distribution of subjects with LVH.

Electrocardiographic findings in the study

Electrocardiographic abnormalities seen among the hypertensive patients are shown in Table 2. The prevalence of LVH using the Scott`s criteria was 46.6% (35.4% for women and 57.7% for men). More severe ECG abnormalities were seen in male patients than female patients.

Specific abnormalities	Male, n (%)	Female, n (%)	p-value	Total
Left ventricular hypertrophy	101 (57.71)	62 (35.43)	0.000*	163 (46.57)
Left axis deviation	15 (8.67)	2 (1.15)	0.19	17 (4.90)
Left atrial enlargement	30 (17.71)	23 (13.14)	0.297	53 (15.14)
Right atrial enlargement	23 (13.14)	27 (15.52)	0.527	50 (14.33)
Atrial fibrillation	2 (0.59)	0(0)	0.474	2 (0.01)
Ventricular premature beats	6 (1.76)	0(0)	0.082	6 (0.02)
ST-T waves abnormalities	49 (28.00)	36 (20.57)	0.105	85 (24.29)
Sinus bradycardia	16 (76.19)	5 (23.81)	0.429	21 (0.06)
Sinus tachycardia	19 (19.4)	33 (8.26)	0.807	52 (0.15)

Table 2: A summary of the electrocardiographic abnormalities seen in the study.

Clinical determinants of left ventricular hypertrophy

The associations between the various risk factors for developing LVH are shown in Table 3. There were no significant difference between ECG-LVH and the following CVD risk factors; age, diabetes mellitus, obesity, family history of hypertension and dyslipidaemia. However there was a significant difference between ECG-LVH and male gender, cigarette smoking, SBP and uncontrolled BP on bivariate analysis. In multiple logistic regression analysis, Male gender (adjusted OR: 2.40, 95% CI 1.53-3.78, 12 P=0.000) and cigarette smoking (adjusted OR: 0.34, 95% CI 0.12- 0.95, P=0.040) were significantly associated with LVH.

Risk factor		LVH=133 Yes, n (%)	No, n (%)	P-value	Total n (%)
Sex	Male	101 (57.71)	74 (42.29)	0.000*	175 (100)
	Female	62 (35.43)	113 (64.57)		175 (100)
Age	30-39	10 (41.67)	14 (58.33)	0.157	24
	40-49	23 (43.40)	30 (56.60)		53
	50-59	47 (41.59)	66 (58.41)		113
	60-69	46 (60.53)	30 (39.47)		76
	70-79	20 (35.09)	37 (64.91)		57
	80-89	17 (62.96)	10 (37.04)		27
Body mass index	Normal	64 (48.48)	68 (51.52)	0.806	132 (100)
	Overweigh t	55 (48.67)	58 (51.33)		113 (100)
	Obese	41 (44.57.0)	51 (55.47)		92 (100)
Diabetes mellitus	Yes	24 (55.81)	19 (44.19)	0.428	
	No	138 (45.25)	167 (54.75)		
Dyslipidaemia	Yes	18 (43.90)	23 (56.10)	0.531	41
	No	142 (46.86)	161 (53.14)		303
Cigarette smoking	Yes	7 (35)	13 (65)	0.027*	20 (100)
	No	137 (46.8)	156 (53.2)		293 (100)

Table 3: Bivariate analysis of risk factors for left ventricular hypertrophy.

LVH in relation to blood pressure classification

There was no statistically significant difference between ECG-LVH and duration of hypertension (p=0.771) and pulse pressure (p=0.474). Elevated SBP, DBP and BP were significantly associated with LVH on bivariate analysis (Table 4). However, in the multivariate analysis, elevated SBP (adjusted OR: 1.79, 95% CI 1.09-2.93, P=0.002) and uncontrolled BP (adjusted OR: 1.86, 95% CI 1.15-3.01, P=0.011) remained independent determinant of LVH (Table 5).

Blood pressure		LVH=133 Yes, n (%)	No, n (%)	P-value
Systolic blood pressure	≥ 140 mmHg	109 (52.91)	97 (47.09)	
	<140	54 (37.50)	90 (62.50)	0.004*
	≥ 90	80 (52.63)	72 (47.37)	0.046*
Diastolic blood pressure	<90	88 (66.17)	133 (63.64)	
Family history of hypertension	Yes	59 (44.36)	107 (40.52)	0.013*
	No	74 (55.64)	102 (48.80)	

Duration of hypertension	<1	35 (44.87)	43 (55.13)	0.771
	02-05	55 (47.82)	68 (55.28)	
	06-10	40 (46.51)	46 (53.49)	
	11-15	12 (46.15)	14 (53.85)	
	>15	21 (56.76)	14 (43.24)	
Blood pressure ≥ 140/90	Yes	121 (36.52)	73 (63.48)	0.008
	No	42 (46.06)	200 (58.48)	
Pulse pressure	High	129 (79.14)	142 (75.94)	0.474
	Normal	34 (20.86)	45 (24.60)	

Table 4: LVH in relation with of blood pressure classification.

Risk Factors	Adjusted Odds	Odds Ratio	[95% Confidence Interval]		P-value
Age	1	1.01	0.99	1.02	0.576
Diabetes mellitus	1.22	1.27	0.77	1.93	0.392
Dyslipidaemia	0.97	0.91	0.5	1.89	0.929
Cigarette smoking	0.34	0.33	0.12	0.95	0.040*
Duration of hypertension	1.02	1.02	0.98	1.05	0.347
Diastolic blood pressure	1.42	1.22	0.74	1.96	0.458
Systolic blood pressure	1.79	1.75	1.09	2.93	0.200*
Male gender	2.4	2.48	1.53	3.78	0.000*
Blood pressure ≥ 140/90 mmHg	1.86	1.92	1.15	3.01	0.011*
Pulse pressure	1.02	1.02	0.98	1.05	0.323
FHH	1.59	1.48	0.86	2.52	0.154
Obese	0.99	1	0.52	1.61	0.754

Table 5: Multiple logistic regression analysis of LVH after adjusting for other independent variables.

Discussion

Left ventricular hypertrophy is a cardinal manifestation of hypertensive cardiac damage. This target organ damage is strongly associated with increased cardiac events. In this study, the prevalence of LVH was found to be high. LVH is a marker of severity of hypertension. It is an important cardiac risk factor and it has a substantial clinical significance on the course of cardiovascular events in terms of morbidity and mortality [26-32]. Our study demonstrated a prevalence of ECG-LVH of 46.6% among hypertensive patients attending a Teaching Hospital in Ghana. This finding shows that nearly half of all the hypertensive patients we studied had ECG-LVH.

The high prevalence rate of ECG-LVH observed in this study might perhaps be a reflection of the burden of uncontrolled hypertension in the patients we studied; it might also be as a result of delayed diagnosis of the hypertension. Evidence from available studies has shown that arterial hypertension and LVH are associated with increased incidence of heart failure, coronary artery disease, stroke, cardiac arrhythmias and sudden cardiac death [3-6,33].

The high prevalence rate of LVH observed in this study compares well with the prevalence of 48% seen in a study in Democratic Republic of Congo. However, previous studies in Kenya, Nigeria, South Africa and Ghana showed lower prevalence rate of 27.5%, 31.0%, 35% and 33.3% respectively [9,12,21,26,27]. The prevalence of LVH in this study was comparably higher than the prevalence rate of 39.0 % obtained in a previous Ghanaian study [28]. This finding has clearly shown the high preponderance of LVH in black hypertensive patients, and highlighting the need for determining the predictors of cardiac damage in these patients.

It has been shown by previous studies that the gender effect on LVH varies from population to population, irrespective of whether ECG or echocardiography was used for its assessment [11,29]. This study found out that being male was an independent determinant of ECG-LVH in the hypertensive patients with adjusted OR of 2.40. This is consistent with some studies from Asian and European countries, but different from what Ajayi et al. [32] have reported from Nigeria where they found more females with ECG-LVH than males [16,30-32]. Another Ghanaian study also found out that women were 2.55 times more likely to develop LVH than men even after adjusting for other factors [32]. Even though the reason for this gender difference is not immediately obvious, these differences might be due to factors such as the ECG voltage criteria used for the assessment, and/or the population studied. There is therefore the need for further epidemiological studies to adequately understand and characterize this effect.

Various studies have reported significant association between ECG-LVH and age, duration of hypertension, diabetes mellitus, obesity and dyslipidaemia [11,15,33-38]. This study found out that increasing age, overweight or obesity, dyslipidaemia, and type 2 diabetes mellitus were not significantly associated with ECG-LVH. Probably the sample size for this study was not large enough to be empowered to detect the significant association between ECG-LVH and these risks factors.

Our study demonstrated a significant association between LVH and increasing systolic blood pressure or uncontrolled blood. There was no correlation between diastolic blood pressure and LVH in this study. An association between elevated systolic blood pressure and/or uncontrolled blood pressure; and hypertensive cardiac damage has already been reported [26-40]. The development of LVH is highly correlated with systolic hypertension [34-48]. In the Framingham Heart Study, even borderline isolated systolic hypertension at an elderly age was associated with increased left ventricular wall thickness and impaired diastolic filling. It is well established that uncontrolled systolic blood pressure is a more powerful independent predictor of cardiovascular disease and is an even stronger risk factor than diastolic blood pressure [41-44].

Epidemiologic studies establishing and describing the relationship between cigarette smoking and cardiovascular diseases are extensive and well documented [38,45,46]. This study showed significant association between ECG-LVH and cigarette smoking. This finding is consistent with results from previous studies that showed that hypertensive patients who are current smokers have higher prevalence of LVH compared to non-smoker hypertensive [26,40]. In agreement with our observation, patients with ECG-LVH in the LIFE study were more likely to be current smokers [38]. Several studies has demonstrated the evidence of cigarette smoking as an important and independent CVD risk factor; a major cause of atherosclerosis and

adverse cardiovascular events [47,48]. Smoking acts synergistically with other risk factors, substantially increasing the risk of cardiovascular events [49]. Cigarette smoking is considered to be the single most important avoidable cause of premature morbidity and mortality in the world [50]. Indeed the rising consumption of cigarette in the developing countries warrants urgent, robust and effective public health measures to reverse this unhealthy lifestyle in order to forestall these impending epidemics of cardiovascular events.

Conclusion

This study highlighted ECG-LVH as an important health challenge in the hypertensive patients who were involved in the study. Male gender, increasing systolic blood pressure, uncontrolled blood pressure and cigarette smoking were found to be the main determinants of LVH in these patients. LVH and its related CVD risks factors are clearly poised to add to the cardiovascular disease burden in Ghana. Our findings have shown the need for extensive assessment of LVH, an established, major emerging CVD risk factor among Ghanaians with essential hypertension.

Conflict of Interest

The authors confirm that this article content has no conflict of interest.

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