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Prevalence and Correlates of Electrocardiographic Left Ventricular Hypertrophy in Hypertensive Patients at a Specialist Clinic in Techiman, Ghana

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Abstract: *Left ventricular hypertrophy (LVH) measured by either electrocardiography (ECG) or echocardiography in hypertensive patients has been shown to be associated with substantially increased risk of cardiovascular morbidity and mortality. In view of its widespread availability and low cost, the ECG has traditionally been the routine method recommended to recognise this target organ damage in the heart. This was a prospective cross-sectional study designed to determine the prevalence and correlates of ECG LVH in hypertensive patients at a specialist clinic in Ghana. Three hundred and forty two (342) hypertensive patients were selected using simple random sampling. Standardized, pretested, structured questionnaires were used to collect socio-demographic characteristics and clinical data of study participants. The baseline demographic, clinical, chest X-ray and electrocardiographic (ECG) characteristics of the patients were examined. Blood pressure and anthropometric measurements were taken according to recommended standards. LVH was determined with a standard 12-lead resting ECG using Scott's criteria. Associations were determined between the presence of LVH and different variables. Logistic regression analysis was done to assess various independent associations with LVH. P-values of <0.05 was considered as statistically significant. The patients were aged between 23 - 79 years with the mean age (± SD) of 46.9 (± 12.1) years. The prevalence of electrocardiographic LVH was 39.0%. Female gender and systolic blood pressure were significantly associated with LVH on bivariate analysis. In multiple logistic regression analysis, female gender (OR: 2.58, 95% CI 1.42 – 4.69, P= 0.002) and duration of hypertension (OR: 1.39, 95% CI 1.06- 1.82, P = 0.017), remained significantly associated with the presence of LVH. In conclusion, this study showed a high prevalence of ECG LVH and significant association of female gender and duration of hypertension with ECG LVH in hypertensive patients in a semi-urban community in Ghana.*

Keywords: *ECG LVH, left atrial enlargement, hypertension, blood pressure, prevalence, correlates.*

I. Introduction

Cardiovascular diseases are on the increase in most countries in sub-Saharan African including Ghana and hypertension is an important risk factor for cardiovascular diseases. Hypertension in sub-Saharan Africa is now a widespread problem of immense health and economic importance due to its high prevalence in urban areas, and it is further complicated by inadequate diagnosis and the severity of its complications[1–4]. Hypertension leads to stress on several organs (called target organs), including the brain, kidneys, eyes, and heart, causing them to deteriorate over time[5–7]. The complications of hypertension are related either to sustained elevations of blood pressure, with consequent changes in the vasculature and heart, or to atherosclerosis which have multiple causes, with long-standing hypertension playing a variable role. People with untreated or poorly controlled hypertension often have the risk of developing complications such as left ventricular hypertrophy, congestive heart failure, retinopathy, cerebrovascular disease and renal insufficiency[6,8,9].

Left ventricular hypertrophy is the best studied marker of hypertensive heart disease[10]. Echocardiography is considered as the gold standard for left ventricular hypertrophy detection. However, the greater convenience and lower cost of the electrocardiography continue to support its widespread use for the diagnosis of left ventricular hypertrophy (LVH) in clinical practice, as well as in epidemiological studies and clinical trials[11]. LVH detected with standard electrocardiography is a strong and independent predictor of future cardiovascular complications, including myocardial infarction, stroke and sudden cardiac death[11–13]. The detection and assessment of ECG LVH has long been an important objective of clinical electrocardiography. Its importance has increased in recent years with the recognition that LVH can be reversed with therapy, and that by doing so, major adverse cardiovascular events can be prevented or delayed[14,15]. Hence it has become imperative to do routine screening electrocardiographically for LVH in hypertensive patients.

The prevalence of LVH among hypertensive patients varies depending on the population, the diagnostic criteria, severity and duration of hypertension, intensity of treatment and patient compliance. The

prevalence rates for ECG LVH among hypertensive subjects ranging between 20% and 70% have been documented in various studies[16 - 21]. In a study that was done in Spain, using ECG, LVH was present in 22.9% of the hypertensive patients[17]. Another study in the same country but only included patients with resistant hypertension, LVH was seen in 57.1% of all study participants[18]. Peer et al in a cross-sectional survey among black South African hypertensive patients found prevalence of ECG LVH to be 35%[19] by using Sokolow-Lyon criteria. In a study involving hypertensive patients in Eastern Sudan, LVH using ECG was detected in 33.3%[20], while in Democratic Republic of Congo, study to determine clinical correlates of LVH in black patients with arterial hypertension revealed that 48% of the patients had LVH using ECG[21]. A study among hypertensive Nigerians reported prevalence rates for ECG LVH of 71.7%, 56.7% and 20% applying Sokolow and Lyon's, Araoye's and Estes' criteria respectively[22]. Another research work by Nkum et al, among hypertensive Gambians reported prevalence rates for ECG LVH of 42.5%, 26.3%, 24% and 57.5%, applying Sokolow and Lyon's, Minnesota, Araoye and Wolff criteria respectively[23]. In Ghana, LVH diagnosed by ECG was observed in 33.3% in a study involving 219 Ghanaian Civil Servants with Hypertension[24]. In Kumasi, Owusu [25] in a hospital-based study of 71 hypertensive heart failure Ghanaians reported a prevalence rate of 91.5% using Scott's criteria for LVH. Conflicting data exist regarding the prevalence of ECG LVH in patients with hypertension[26], but importantly no ECG-LVH based study has ever been conducted in such semi-urban community in Ghana, hence the need for this hospital-based study. This study was therefore designed to determine the prevalence and correlates of ECG LVH in hypertensive patients at a specialist clinic in Techiman, Ghana.

II. Methods And Materials

The study was a hospital-based prospective descriptive study carried out at the hypertension clinic of the Holy Family Hospital, Techiman, Ghana, from November 2014 to April 2015. Informed consent was obtained from each study participant. Patients aged 18 years and above attending the hypertension clinic of the Holy Family Hospital, Techiman, Ghana with clinical diagnosis of hypertension were recruited. Three hundred and forty two (342) hypertensive patients were selected using simple random sampling. Standardized, pretested, structured questionnaires were used to collect socio-demographic characteristics and clinical data of the participants. The baseline demographic, clinical, chest X-ray and electrocardiographic (ECG) characteristics of the patients were examined.

Clinical examination included the pulse (rate, rhythm, volume and the character), the blood pressure (BP), the apex beat, the heart sounds (S1, S2, S3 and S4) and murmurs were also examined. The BP was recorded in left arms, with patients lying supine after a 10-minute rest, using a mercury sphygmomanometer with a cuff size 12cm long and 35cm wide. The cuff was positioned at the heart level and deflated at 2 mm/s and the blood pressure was measured to the nearest 2 mmHg. Systolic blood pressure (SBP) was recorded as appearance of the Korotkoff sounds (phase I) whilst diastolic blood pressure (DBP) was recorded as disappearance of the Korotkoff sounds (phase V). Three readings were taken. A first measurement was used to familiarise the subject with the procedure. The blood pressure was repeated twice at five minutes intervals, during which the subject remained seated. The mean of the later two readings was used in the analysis.

A standard 12-lead ECG was performed on each patient lying supine on a couch in the examination room on the medical ward, by using a portable Cardette Excel 103 ECG machine. With the patient relaxed and comfortably lying on supine position, the electrodes were placed as originally described by Frank[27]. Effective skin contact was ensured during the ECG recording. The stylus control was set at 10mm/mV and paper speed at 25mm/s. It was regularly checked for any technical faults such as damping and electrical interference. The ECG tracings were interpreted by an experienced physician using calipers. The diagnosis of LVH was assessed electrocardiographically using Scott's criteria. The following definitions were used for the study.

Hypertension was defined as the presence of a persistent elevated SBP $\geq$ 140mmHg and/or diastolic DBP $\geq$ 90mmHg, and/or the use of anti-hypertensive drugs and/or past medical history of hypertension[28].

Diabetes mellitus type 2 was defined as previous diagnosis of diabetes, at least two random blood glucose readings of > 11.1 mmol/L or a fasting blood glucose reading of > 7 mmol/L or taking oral hypoglycemic agent or insulin and did not have previous episodes of ketosis in the past[29].

Dyslipidaemia was defined as serum total cholesterol of > 5.2 mmol/L or low density lipoprotein cholesterol LDL-C > 3.2 mmol/l or high density lipoprotein cholesterol HDL-C < 1.03 mmol/L for men and < 1.29 mmol/l for female or pretreatment with a cholesterol lowering agent. Hypertriglyceridaemia was defined as a serum triglyceride > 1.7 mmol/l according to AHA/NHLBI criteria [30]

Serum uric acid was classified as elevated if levels are > 420 umol/l in men and > 360 umol/l in women, taking into consideration patients on thiazide diuretics[31].

Obesity was defined as a raised body mass index >29.9 kg/m2 in ambulant patients or waist circumference of > 80 cm in females and >94 cm in males. Then the body mass index (BMI) will be calculated from formula: BMI=weight in kg/ (height in m) ^{2}. By the World Health Organization (WHO, 2000) criteria, a BMI <18.5kg/m^2 is considered underweight, 18.5–24.9 kg/m^2 ideal weight and 25–29.9kg/m^2 overweight or pre-

obese. When BMI was greater than 29.99 kg/m^2, patients were designated as obese. Waist circumference was measured by a tape measure horizontally placed at the level of the superior crest and ensuring the tape measure was snug and did not compress the skin. Measurement was made at the end of normal expiration[32].

A resting 12 lead ECG was obtained from each hypertensive patient according to standard procedure, and evaluated by the author. Electrocardiographic LVH criteria used in this study was Scott's Criteria[33] and LVH was diagnosed on the basis of fulfillment of at least one of the following criteria on ECG:

- 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

Chest X-rays were obtained from each patient and examined for increased cardiac size as judged by a cardiothoracic ratio more than 0.5.

Inclusion criteria:

Adults aged 18 years and above with documented diagnosis of hypertension attending hypertension clinic, who met the criteria were included in the study.

Exclusion criteria:

The following patients were excluded from the study: Patients admitted with suspected hypertension but could not meet the diagnostic criteria.

Ethical considerations

All procedures were carried out according to a study protocol approved by the Committee on Human Research Publication and Ethics of School of Medical Sciences, the Kwame Nkrumah University of Science and Technology, Kumasi, Ghana. Informed consent was obtained from all subjects. The objectives and nature of the study were explained to all subjects. The information about participant's identity was not included with the other data and only the principal investigator had access to this information.

Statistical Design and Analysis

Data was collected and edited to exclude errors, re-organized, coded and manipulated with appropriate software for efficient analysis. Data were entered into Filemakerpro11.0 version and then exported to Microsoft Excel 2007 version for cleaning. Data was then transferred to Strata SE version 11.1 for statistical analysis. Data was analyzed for frequency of distribution, proportions, percentages and mean ± SD. For all categorical valuables, bivariate analysis were done using Fishers exact test for statistical significance. Multiple logistic regression analysis was employed to determine the independent effect of variables on LVH.

III. Results

A. The sample comprised 342 hypertensive patients. The mean age of the participants was 46.94 (±12.11) years with a range of 23 to 79 years. There were more females (62.28%; n=213) than males (37.72.78%; n=129).

Resting 12-lead ECG findings at the time of study

The ECG findings recorded in the study patients are shown in Table 1. ECG-LVH was seen in 39.0 % (133) of the study patients. Other ECG findings seen includes: left atrial enlargement 14.7% (50), right atrial enlargement 9.4 % (32), left axis deviation 1.8 % (6), sinus tachycardia 10 % (34), sinus bradycardia 8.6 % (29) and ST-T-wave abnormalities 102(30.1%). Two patients had atrial fibrillation.

Table 1: The resting 12-lead ECG findings at the time of study

ECG Findings	n (%)
Left ventricular hypertrophy	133 (39)
Left axis deviation	6 (1.76)
Left atrial enlargement	50 (14.71)
Right atrial enlargement	32 (9.41)
Atrial fibrillation	2 (0.59)
Ventricular premature beats	6 (1.76)

ST-T waves abnormalities	102 (30.12)
Sinus bradycardia	29 (8.55)
Sinus tachycardia	34 (10.03)

B. *Correlates of Left Ventricular Hypertrophy*

LVH in relation to Age:

Of the 39% (133) hypertensive patients with LVH, 75.2% were within the range of 30-59 years. A bivariate analysis showed that age was not significantly associated with the development of LVH. (P=0.742, Table 2).

LVH in relation to Gender

The prevalence of LVH in female was found to be 51.1% (68) versus 48.9% (65) in male (P=0.001, Table 2). There was a significant difference in the prevalence between female and male genders. Bivariate analysis showed that females were more likely to develop LVH as compared to their male counterparts. After controlling for other potential risk factors in a multiple logistic regression model, this gender effect remained significant (adjusted OR=2.58, CI 1.42- 4.69, P=0.002, Table 4).

LVH in relation to BMI

The mean BMI was 27.7 ± 6.99 Kg/m^2. Eighty-nine (28.7%) of the study patients were found to be obese and 97 patients (31.4%) were classified as overweight according to the standard classification of overweight as BMI ≥ 25 Kg/m^2 and obesity as BMI ≥30 kg/m^2. Overall, 186 (60%) of the cases had BMI ≥ 25 Kg/m^2. %). A higher proportion of females were obese 73 (38.0%) compared to males 16 (13.6%) of the study patients. However, there was no significant difference in the proportion of LVH between individuals with normal or high BMI. (P=0.401; Table 2)

LVH in relation to Duration of Hypertension

The bivariate analysis of duration of hypertension was not significantly associated with LVH (Table 3). However with the multivariate analysis, when other factors came into play, longer duration of hypertension was significantly associated with LVH. Thus after adjusting for all potential risk factors, patients with longstanding hypertension were 1.39 times more likely to develop LVH (adjusted OR: 1.51, 95% CI 1.06- 1.82, P=0.017; Table 4).

LVH in relation to Blood Pressure Control

Elevated systolic blood pressure (SBP) was found to be significantly associated with LVH on bivariate analysis (Tables 3). However, a significant association between SBP and LVH was not seen in the multivariate analysis (95% CI 0.97 - 1.06, p=0.727, Tables 4).

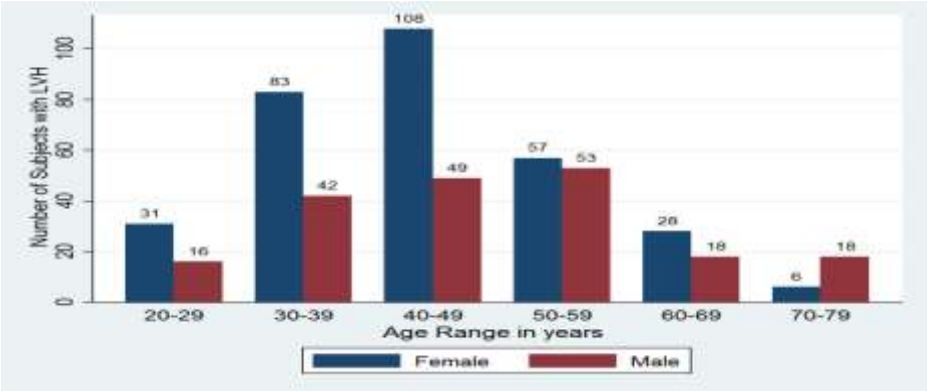

Figure 1: LVH in relation to age and gender distribution of study patients

LVH in relation to Diabetes Mellitus

The prevalence of diabetes in the study patients at the time of the study was 24.6 % (25.6 % in males and 23.9% in females). (Table 2) Seventy-five (90%) of the diabetic patients were on insulin or oral glucose lowering agents. Diabetes was not found to be significantly associated with LVH in this study. (p= 0.492, Table 2)

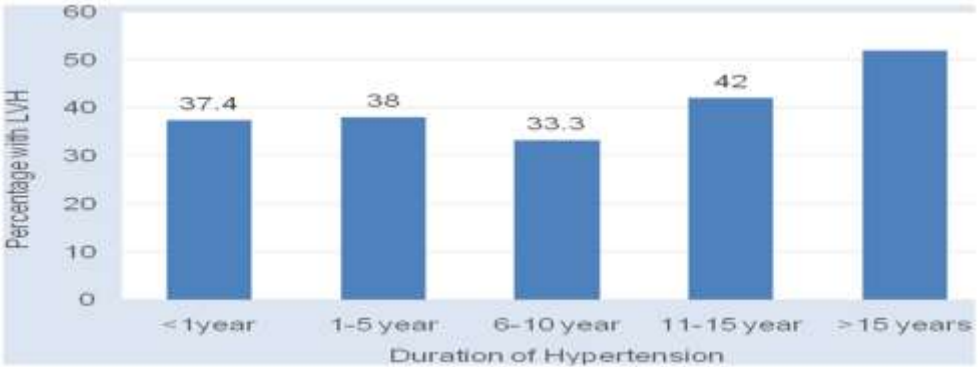

Figure 2 shows LVH in relation the duration of hypertension at the time of the study

LVH in relation to Dyslipidaemia

The prevalence of dyslipidaemia among the study patients was 49.1% (51.6 % in males and 47.6% in females) at the time of the study (Table 2). Dyslipidaemia was not found to be significantly associated with LVH in this study. (P-value = 0.627, Table 2)

LVH in relation to Cigarette Smoking

The prevalence of significant smoking among the study patients at the time of the study was 6.2% (15.8% in males and 0.4% in females) Table 2. Cigarette smoking was not found to be significantly associated with LVH in this study.(P-value = 0.712, Table 2)

LVH in relation to serum uric acid level

The prevalence of high serum uric acid (SUA) was 21.4% (25.8% in males and 18.8.0% in females), (Table 2). SUA was not found to be significantly associated with LVH in this study. (P-value = 0.119, Table 2)

Table 2: Bivariate analysis of LVH in relation to CVD risk factors in the study patients

Predictor variables		**LVH=133**		
		Yes, n (%)	**No, n (%)**	**P-value**
Sex	Female	68 (51.1)	145 (69.4)	0.001*
	Male	65 (48.9)	64 (30.6)	
Age categories	20-29	13 (9.8)	19 (9.1)	
	30-39	38 (28.6)	46 (22.0)	0.742
	40-49	38 (28.6)	68 (32.5)	
	50-59	24 (18.1)	47 (22.5)	
	60-69	13 (9.8)	19 (9.1)	
	70-79	7 (5.3)	10 (4.8)	
Body mass index	Underweight	0 (0.0)	5 (3.4)	0.401
	Normal	58 (19.3)	140 (70.7)	
	Overweight	1 (10.0)	43 (29.3)	
	Obese	1 (10.0)	21 (14.3)	
Diabetes mellitus	Yes	103 (77.4)	155 (74.2)	0.492
	No	30 (22.6)	54 (25.8)	
Dyslipidaemia	Yes	64 (49.3)	106 (52.0)	0.627
	No	66 (50.8)	98 (48.0)	
Microalbuminuria	Present	9 (7.2)	9 (4.6)	0.309
	Absent	116 (92.8)	186 (95.4)	
Serum uric acid	Normal	99 (75.0)	169 (80.8)	0.199
	High	33 (25.0)	40 (19.1)	
Alcohol	Yes	45 (33.8)	68 (32.7	0.027
	No	88 (66.2)	140 (67.3)	
Cigarette smoking	Yes	9 (6.8)	12 (5.8)	0.712
	No	123 (93.2)	194 (94.2)	

Table 3: Bivariate analysis of LVH with classification of blood pressure

Predictor variables		**LVH=133**		
		Yes, n(%)	**No, n(%)**	**P-value**
Systolic blood pressure	>=140 mmHg	84(63.16)	105(50.24)	
	<140	49(36.84)	104(49.76)	0.019*
Diastolic blood pressure	>=90	45(33.83)	76(36.36)	0.633
	<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	<1year	23(17.29)	39(18.66)	
	1-5years	70(52.63)	113(54.07)	
	6-10years	19(14.29)	38(18.18)	0.221
	11-15years	8(6.02)	11(5.26)	
	>15	13(9.77)	8(3.83)	
Blood pressure>=140/90	Yes	46(34.85)	85(40.67)	0.282
	No	76(46.06)	200(58.48)	
Pulse pressure	High	25(18.80)	51(24.40)	0.224
	Normal	108(81.20)	158(75.60)	

Table 4: Multiple logistic regression analysis of LVH in cases after adjusting for other independent variables.

Risk Factors	Adjusted Odds	Odds Ratio	[95% Confidence Interval]		P-value
Diabetes mellitus	0.54	0.56	0.30	1.07	0.081
Dyslipidaemia	0.97	0.91	0.54	1.55	0.74
Cigarette smoking	0.44	0.42	0.13	1.36	0.147
Duration of hypertension	1.51	1.39	1.06	1.82	0.017*
Microalbuminuria	1.42	1.43	0.47	4.38	0.532
Systolic blood pressure	0.99	1.00	0.97	1.02	0.727
Female gender	3.42	2.58	1.42	4.69	0.002*
Serum uric acid	1.00	1.00	1.00	1.00	0.706
Pulse pressure	1.02	1.02	0.98	1.05	0.323
FHH	1.59	1.48	0.86	2.52	0.154
Obese	1.54	1.77	0.41	7.62	0.444

FHH= Family history of hypertension

IV. Discussion

Left ventricular hypertrophy is associated with a substantially increased risk of cardiovascular morbidity and mortality, so its detection is of major importance, especially for individuals with hypertension[34,35]. This study has revealed a significantly high prevalence of ECG-LVH among the hypertensive patients examined. The prevalence of LVH in hypertensive patients in most literatures worldwide varies from 20 to 70% based on the population studied and the criteria used [16=21, 24, 36–38]. The prevalence of LVH of 39% among patients in this study is within this range. However, the prevalence in this study is lower than the prevalence of 48% seen in a study in Democratic Republic of Congo; but higher than the findings of 27.5%, 31.0%, 35% and 33.3% in Kenyatta Hospital, Nigeria, South Africa and Ghana respectively[19,21,24,39,40]. A significant proportion of the hypertensive patients with LVH had ST- T waves abnormalities on ECG, which has been reported to be associated with a worse prognosis, as ECG strain pattern itself is also a marker of increased cardiovascular risk in hypertensive patients independent of baseline severity of ECG LVH [41–44].

Age was not associated with LVH in this study, however age was found to be an independent risk factor for LVH in most studies[36,44,45]. Age has been reported to contribute strongly to the clustering of cardiovascular risk factors. Aging is a powerful and independent predictor of cardiovascular morbidity and mortality [46, 47]. It is possible that the sample size for this study was not large enough to be empowered to detect the association between age and LVH. Another possible reason may be due to attenuation of QRS voltage with advancing age in this studied population[48].

Female gender was found to be an independent risk factor for LVH in hypertensive patients in our study with adjusted OR of 2.55. Different studies from around the world have shown that the gender effect on prevalence of LVH varies from population to population[45,49–51]; whether ECG or echocardiography is used for its assessment and criteria used[36,38]. A study in Pakistan, for instance, reported that women were 11.35 times more likely to develop LVH than the male counterparts even after adjusting for other factors[49]. Similarly, studies in Nigeria, China and the LIFE (losartan intervention for endpoint reduction in hypertension) study showed a trend towards a higher prevalence in women[50–52]. However, some studies from European countries have shown a higher prevalence in men [45]. One possible factor that may have contributed to the observed gender difference is the lack of different thresholds for men and women for ECG changes.

Few studies have specifically addressed the differential prognostic value of LVH in men and women. Recent literature suggests that increased LV mass is a stronger risk factor in women than in men. This is particularly true of echocardiographic LVH. Liao et al described an unfavourable prognosis in terms of total death and cardiac-related deaths in black women with echocardiographic LVH compared to black men with echocardiographic LVH [53]. Data from the Glasgow blood pressure clinic also support this observation of survival disadvantage conferred by LVH in women[54]. In the presence of ECG LVH, women had a substantially higher risk of dying from cardiovascular causes. The Framingham Heart Study highlighted an increased risk of stroke and CVD mortality among women with ECG LVH[55]. The difference in survival between genders

conferred by ECG LVH has no clear underlying mechanism but a higher prevalence of the concentric geometric abnormality in women than men is a possible explanation. The latter carries the highest risk and eccentric hypertrophy an intermediate risk[56].

Duration of hypertension was found to be significantly associated with LVH in this study, which is consistent with most studies from around the world [57,58]. The duration of hypertension, a marker of process of aging, was associated with a greater risk for the development of LVH. The impact of longer duration of hypertension to structural remodelling of the heart is well established. The remodelling process in long-standing hypertension[59,60] consists of hypertrophy, fibrosis and impaired microvascular circulation. Furthermore, longer duration of hypertension appears as the main clinical determinant of large artery stiffness, independent of other risk factors[60]. Longer duration of hypertension with arterial stiffness is accompanied by higher SBP and pulse pressure, which are well-known cardiovascular risk factors[60]. The longer the duration of hypertension the greater the incidence of LVH.

No association was found between blood pressure control and the occurrence of LVH in this study. Although the design of this study could not allow for the assessment blood pressure control over the long-term, it has been shown in other studies by Peer et al in South Africa and Shirfkan et al in Iran, that patients with uncontrolled blood pressure have high prevalence of LVH compared to those with controlled blood pressure[19,61]. These studies concluded that, tight control of blood pressure would reduce the prevalence of LVH among hypertensive patients.

Diabetes mellitus was not associated with LVH in the present study, however diabetes mellitus has been observed to be positively associated with ECHO or ECG-determined LVH[62–64]. In a previous study, LVH was present in 31% of diabetics, and systolic blood pressure was of no value in identifying those diabetics who had LVH, possibly because insulin resistance itself stimulates LVM growth [65,66]. Verdecchia P et al proposed that in addition to circulating insulin, insulin growth factor-1 is also an independent determinant of LVM and geometry in essential hypertension[67]. In a relatively healthy, population-based sample of hypertensive adults, type 2 diabetes was associated with higher LVM, more concentric LV geometry, and lower myocardial function, independently of age, sex, body size, and arterial blood pressure[64].

Obesity has been shown to be associated independently with increased anatomical LVH among hypertensive subjects and in a general population[206,207,208]. However, obesity was not associated with LVH in the present study. The sensitivity of ECG-LVH to recognise ECHO- LVH among obese subjects, especially among obese women, has been confirmed to be low[69,70]. This decreased sensitivity in obese subjects may be because of accumulation of the subcutaneous adipose tissue of the chest wall, although in women the effect of breast tissue appears to have a surprisingly small effect on ECG voltages[69–71].

About 60% of the study patients had obesity or were overweight in this semi-urban community in Ghana. Considering the rising global epidemic of obesity, it is likely that adverse health consequences of excess adiposity will escalate in the near future[72,73]. LVH is one of the cardiac complications of obesity and ECG or ECHO determined LVH is a powerful independent predictor of cardiovascular morbidity and mortality[11,74,75]. More recently epidemiological research has indicated that the pattern of obesity is important, with centralized or abdominal obesity being particularly hazardous[76]. This android variety of obesity has been linked to occurrence of cardiovascular disease, hypertension, dyslipidemia and insulin resistance[77,78].

In this study, Serum Uric Acid (SUA) did not show significant association with LVH. However several studies have shown that subjects with higher SUA levels more frequently have LVH[79–81]. For example, in an analysis of essentially healthy male individuals, Mitsuhashi H et al showed that individuals with SUA values of 6.6–11.0 mg/dL had an increased prevalence of LVH, which was independent of age, body mass index, serum creatinine, hypertension, diabetes and hyperlipidaemia[79]. In addition, Viazzi F et al demonstrated that the association between SUA and cardiac hypertrophy remained significant after adjustment for body mass index, age, creatinine clearance and high-density lipoprotein cholesterol in middle-aged untreated female patients with essential hypertension[82].

Few studies have looked at the association of cigarette smoking and dyslipidaemia with ECG LVH. In the LIFE Study patients with ECG-LVH were more likely to be current smokers and have a lower serum cholesterol level than patients without LVH[63]. In the present study, there was no association of smoking and dyslipidaemia with the presence of LVH. Inclusion of individuals who have smoked little or stopped for many years ago may have under-estimated the strength between ECG-LVH and smoking. Besides absence of association between LVH and cigarette smoking and dyslipidaemia in this study could also be explained by the small sample size.

With the fact that ECG has high specificity in detecting LVH and tends to detect more severe forms of LVH, this greater prevalence means significant number of the study population could be at high risk for cardiovascular diseases. Data on prognostic implication of LVH in the Framingham study indicated that LVH has emerged as a powerful indicator of rapidly evolving lethal atherosclerotic disease whether determined by ECG or ECHO[83]. Presence of LVH has important clinical implications in hypertensive patients as it is

associated with increase in the incidence of heart failure, ventricular arrhythmias, death following myocardial infarction, decreased left ventricular ejection fraction, sudden cardiac death, and a cerebrovascular event. The increase in cardiovascular risk has been directly related to the degree of increase in left ventricular mass[84].

The observed higher prevalence of LVH in the Ghanaian hypertensive population studied, might contribute to the understanding of the development of an increase in high risk of cardiovascular events in hypertension. The findings strongly suggest a substantial future burden of both morbidity and mortality from LVH in this semi-urban community and Ghana at large. This represents a situation that demands keen attention if its potential morbidity and mortality are to be reduced or better still avoided. Early detection of hypertension and strict blood pressure control with antihypertensives, which includes angiotensin II receptor blocker or an angiotensin-converting enzyme inhibitor, will help to reduce LVH in the hypertensive population studied.

V. Conclusion

In conclusion, this study has revealed a significantly high prevalence of ECG-LVH and significant association of female gender and duration of hypertension with ECG-LVH in hypertensive patients in a semi-urban community in Ghana. The high prevalence of ECG LVH found in this study should further prompt clinicians to actively screen for this acknowledged potential CVD risk factor and implement aggressive secondary prevention therapy when present.

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