Electrocardiographic Left Ventricular Hypertrophy In Patients Seen With Hypertensive Heart Failure

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Citation

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Abstract

This study was done to determine the prevalence of electrocardiographic (ECG) left ventricular hypertrophy (LVH) and other ECG changes in patients seen with hypertensive heart failure at the Komfo Anokye Teaching Hospital (KATH), Kumasi, Ghana.

Patients aged thirteen years and above admitted to the medical wards with the diagnosis of hypertensive heart failure were recruited. Detailed history was obtained. Physical examination, standard 12 lead ECG and chest-X-ray were done.

Seventy-one (71) patients were studied. There were 49.3 % (n=35) males and 50.7% (n=36) females. ECG LVH was seen in 91.5 % (n=65) of the patients. Other ECG findings seen on admission include: left axis deviation 17.0 % (n=44), left atrial enlargement 42.3 % (n=30), and complete bundle branch block 15.5% (n=11).

In conclusion, ECG LVH is highly prevalent in hypertensive heart failure. Early diagnosis and treatment of LVH in patients with hypertension may prevent heart failure.

INTRODUCTION

Hypertension, rheumatic heart disease, and cardiomyopathy are the main causes of heart failure in West Africa [1,2,3,4]. Various risk factors for heart failure have also been described [5]. Clinical factors that are strongly and consistently associated with heart failure include: age, ECG LVH, overweight or obesity and diabetes mellitus [5].

The development of LVH is a relatively early response to hypertension, demonstrable in children and adolescents with borderline elevations in BP. Studies show that patients who have exaggerated transient elevations in BP during mental stress; particularly at work or exercise may be particularly prone to the development of LVH [6, 7].

Ambulatory BP monitoring has suggested that there may be two additional risk factors for LVH. These include: the daytime BP load and nocturnal hypertension; in which the expected nighttime reduction in BP is not seen [7]. Other studies have suggested that the maximal daytime BP or peak exercise BP is most predictive of the level of hypertrophy [8]. These factors may explain why the ambulatory BP values may correlate more closely with the development of LVH than the casual BP taken in the physician's office.

LVH is a common finding in patients with hypertension and can be diagnosed either by ECG or by echocardiography [9]. There is evidence from large population based studies of an increased tendency to LVH in hypertensive blacks, independent of body composition [10]. Hypertension may also result in interstitial fibrosis [11]. Both factors contribute to an increase in left ventricle stiffness, resulting in diastolic dysfunction and an elevation in left ventricular end diastolic pressure.

LVH is not an acute condition. It takes weeks and usually months to years to develop. It has been proposed that a cardiac renin-angiotensin system and angiotensin converting enzyme activity may be an important determinant of the hypertrophic response [12]. There are two predominant types of hypertrophy: concentric; where wall thickness is increased relative to cavity dimensions, and eccentric; where there is left ventricular cavity dilatation, with an increase in muscle mass so that the ratio between wall thickness and ventricular cavity size remains relatively constant.

The ECG is a useful but imperfect tool for detecting LVH. The utility of the ECG relates to its being relatively inexpensive and widely available. The limitations of the

ECG relate to its moderate sensitivity or specificity depending upon which of the many proposed sets of criteria are applied [13]. However, the ECG may be used in poor resource countries where echocardiography is unavailable or too expensive.

ECG LVH is a powerful independent predictor of cardiovascular morbidity and mortality irrespective of aetiology. Patients with ECG LVH from any cause are at increased risk for major cardiovascular complications including heart failure, cardiac arrhythmias, death following myocardial infarction, decreased left ventricular ejection fraction, sudden cardiac death, aortic root dilation, and stroke [14].

Conflicting data exist regarding the prevalence of ECG LVH in patients with heart failure [15]. This study was done to determine the prevalence of ECG LVH and other ECG changes in patients seen with hypertensive heart failure at the KATH, Kumasi, Ghana.

METHODS

The study was a hospital-based prospective descriptive carried out at the Department of Medicine, KATH in Kumasi from October 2004 to March 2005. Informed consent was obtained from each study participant and KATH ethical committee approved the study.

Patients aged thirteen years and above who were admitted to the medical wards with clinical diagnosis of hypertensive heart failure were recruited. Detailed history including duration of hypertension, past medical history, and drug history were obtained from each study participant through a standard questionnaire.

Clinical examination included general assessment to look for dyspnoea at rest, pedal oedema or generalized swelling, cyanosis. The pulse rate, rhythm, volume and the character were noted. Jugular venous pressure, the BP, the apex beat, the heart sounds (S_1, S_2, S_3 and S_4) and murmurs were also examined. The chest was auscultated for crackles, and the presence of hepatomegaly and ascites were also noted.

The BP was recorded in both 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) [16].

Hypertension was defined as the presence of a persistent elevated SBP ≥ 140mmHg and/or diastolic DBP ≥ 90mmHg [16], and/or the use of anti-hypertensive drugs and/or past medical history of hypertension.

A resting 12 lead ECG was obtained from each patient on admission according to standard procedure, and evaluated by the author. ECG LVH was diagnosed using Scott's criteria for LVH [17]:

Figure 1

Limb leads:	R in 1 + S in 3:	more than 25 mm
	R in aVL:	more than 7.5 mm
Chest leads:	S in V_1 or V_2 + R in V_5 or V_6:	more than 35 mm
	R in V_5 or V_6:	more than 26 mm
	R + S in any V lead:	more than 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, and the presence of pulmonary upper lobe blood diversion and/or alveolar oedema and/or pleural effusion.

Diagnosis of heart failure was confirmed, using the following modified Framingham criteria for the diagnosis of heart failure [1, 18]:

1. Major criteria: Paroxysmal nocturnal dyspnoea, raised jugular venous pressure, clinical cardiomegaly, basal crepitations, S3 gallop, clinical acute pulmonary oedema, pulmonary upper lobe blood diversion on chest X-ray (or pulmonary oedema on chest X-ray).
2. Minor criteria: tachycardia, orthopnoea, exertional dyspnoea, nocturnal cough, hepatomegaly, pleural effusion, diuretic use.

Heart failure was diagnosed if the patient had two major and one minor or one major and two minor criteria.

INCLUSION CRITERIA

Patients aged thirteen years and above admitted to the medical wards for the first time, with the diagnosis of hypertensive heart failure who met the modified

Framingham criteria for the diagnosis of heart failure, were included in the study.

EXCLUSION CRITERIA

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

STATISTICS

Data from the standard questionnaire were entered into a Microsoft Excel (2000) sheet and then exported into Stata Version 8.0 statistical software for analysis. Measure of central tendency using means and median, measure of spread using standard deviation and range were calculated.

RESULTS

Seventy-one (71) patients were studied. There were 49.3 % (n=35) males and 50.7% (n=36) females. They were aged between 28-95 years with a mean age of 59 ± 15 years (95 % confidence interval for mean age of 55.5-62.5 years), and a median age of 60 years.

Table 1 shows the duration of hypertension as at the time of admission. Thirty-one percent (31 %) of the patients were not aware that they were hypertensive, and therefore they were not receiving any anti-hypertensive treatment.

Figure 2

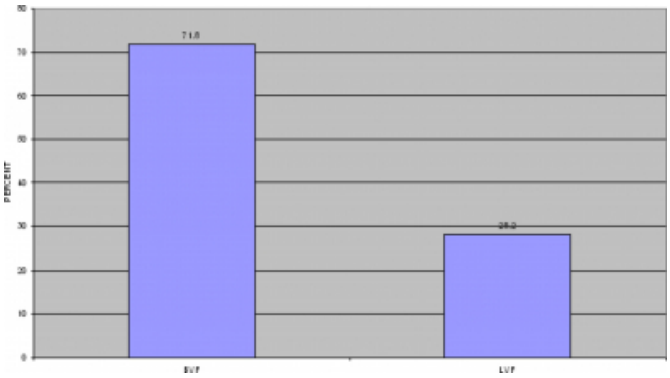
Table 1: Duration Of Hypertension

DURATION OF HYPERTENSION (YEARS)	N	%
Unknown	23	31.0
≤ 5	5	7.4
6-10	15	22.0
11-15	14	20.6
≥16	13	19.1

Figure 1 shows the nature of heart failure on admission. Majority of the patients (71.8 %, n=51) were admitted with biventricular failure. Mean SBP and mean DBP of the patients were 175 (±15) mmHg, and 112 (±24) mmHg respectively.

Figure 3

Figure 1: Nature Of Heart Failure On Admission



ECG LVH was seen in 91.5 % (n=65) of the patients. Other ECG findings seen on admission include: left axis deviation 17.0 % (n=44), left atrial enlargement 42.3 % (n=30), sinus tachycardia 19.7 % (n=14) and complete bundle branch block 15.5% (n=11). Table 2 shows the resting 12 lead ECG findings at the time of admission.

Figure 4

Table 2: Resting 12 Lead Ecg Findings Of Patients At The Time Of Admission

ECG FINDINGS	N	%
Left Ventricular Hypertrophy	65	91.5
Left Axis Deviation	44	17.0
Left Atrial Enlargement	30	42.3
Sinus Tachycardia	14	19.7
Complete Bundle Branch Block	11	15.5
Left Bundle Branch Block	7	9.9
Right Bundle Branch Block	4	5.6
Right Atrial Enlargement	8	11.3
Biatrial Atrial Enlargement	5	7.0
Atrial Fibrillation	5	7.0
Ventricular Premature Beats	4	5.6
Sinus Bradycardia	2	2.8

DISCUSSION

Despite its limitations, the ECG is a useful tool for detecting LVH, especially in poor resource countries where echocardiography is unavailable or too expensive. Hypertension is a major problem in black patients who are much more prone to the associated cardiovascular complications [10].

Hypertension awareness, treatment, and control rates as low

as 20%, 10%, and 1%, respectively have been found in West Africa [19]. In this study, 31 % of the patients were unaware that they were hypertensives. Undiagnosed, untreatment and inadequately treated hypertension can result in an enormous burden of cardiovascular disease in blacks. Therefore, the importance of treating high BP to achieve the appropriate BP goal cannot be overemphasized. Early detection and treatment of hypertension, is important to avoid major cardiovascular events in blacks.

The mean SBP (175 ±15 mmHg) and mean DBP (112 ±24 mmHg) of the patients were high. Isezuo et al. [20] also reported high mean SBP and mean DBP of 180.4 (± 28.2) mmHg and 117 (± 12.9) mmHg respectively, among Gambians and Nigerians with hypertensive heart failure. Studies have demonstrated that as SBP and DBP increase, the risk of cardiovascular events increases continuously [21]. A positive relationship between diastolic dysfunction and the level of the BP has also been established [22], with the degree of the diastolic dysfunction proportionate to increasing level of blood pressure. High BP pressure leads to concentric hypertrophy, which finally results in left ventricular diastolic dysfunction. Concentric hypertrophy in actual fact results in reduced left ventricular relaxation and increased left ventricular end-diastolic pressure. Pulmonary eodema eventually occurs if left ventricular diastolic dysfunction is not detected early and treated.

ECG LVH is a common finding in heart failure. Two studies of 172 and 229 consecutively hospitalized patients with heart failure demonstrated a prevalence of ECG LVH of 51.0 % and 49.0 % respectively [23, 24]. There is evidence that hypertensive black patients have an increased tendency to LVH, therefore it is not surprising that ECG LVH was seen in 91.5 % of the patients in this study[10, 19, 22].

LVH, which results from cardiac remodeling, has been shown to increase the risk of heart failure [5, 22, 25]. In the Framingham heart study [25]it was found out that ECG LVH was associated with a 15-fold increase in the incidence of heart failure. The prevention or regression of LVH should be an important therapeutic goal. LVH should be detected early in patients with hypertension, and the ECG can be a useful tool in detecting it. When present, aggressive treatment of LVH may prevent or delay the onset of heart failure in these patients.

Studies have shown that both hypertension and LVH are associated with increased risk for cardiac arrhythmias [14], and this study has also confirmed that. Seven percent of the patients had atrial fibrillation. Complete bundle branch block, ventricular premature beats, and sinus bradycardia were also seen in 15.5 %, 5.6 %, and 2.8 % of the patients respectively.

Hypertension and LVH may result in interstitial fibrosis [11]. LVH also causes several electrophysiological changes or electrical remodeling leading to prolongation of action potential and altered repolarization. This leads to an increased in cardiac arrhythmias and sudden death. Although cardiac asynchrony was not evaluated in these patients, those with complete bundle branch block were likely to develop cardiac asynchrony requiring cardiac resynchronizing therapy, which is not available in Ghana at the moment. In view of this, efforts should be made by physicians to control hypertension adequately to prevent LVH and heart failure in patients with hypertension.

In conclusion, ECG LVH is highly prevalent in patients with hypertensive heart failure. Cardiac arrhythmias are also common in these patients. Early detection and treatment of hypertension and LVH may prevent heart failure.

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