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Original Article

A Comparative Study of Electrocardiographic and Echocardiographic Evidence of Left ventricular Hypertrophy

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Abstract

This study was carried out in medicine and cardiology indoor of Rajshahi Medical College Hospital from November 2004 to October 2005. 100 cases were selected for this study in random manner. Sensitivity of ECG to diagnose LVH was found to be 87.5%, and specificity was only 50%. ECG is relatively insensitive and can't accurately identify the severity of LVH.

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Introduction

Left ventricular hypertrophy (LVH) is thickening of the wall of the left ventricle resulting in an increase in left ventricular mass. The importance of left ventricular hypertrophy has gained wide recognition. The increased risk associated with left ventricular hypertrophy (LVH) diagnosed echocardiographically (Echo-LVH) or electrocardiographically (ECG-LVH) is well known. LVH by both ECG and echo is a powerful independent risk factor for cardiovascular morbidity and mortality[1].

LVH is associated with coronary events, and there is an association between cerebrovascular disease and increased left ventricular mass (LVM)[2]. The Framingham heart study revealed that LVH by ECG was associated with a five-year mortality of 35% in males and 20% in females. In another study mortality was nearly three times greater for hypertensive patients with LVH compared with those without LVH[3]. The excess risk conferred by LVH is independent of blood pressure (BP) level of a patient. On the other hand, regression of left ventricular hypertrophy is associated with reduction in all cause and cardiovascular mortlity[4].So reversal of LVH is an important goal of antihypertensive therapy[5].

Classically, left ventricular hypertrophy, which represents an increase in LV mass, has been thought to represent a reaction to pressure or volume overload of left ventricle. In the short run, increase in LV mass may be beneficial by allowing the heart to compensate for increased wall stress and potential homodynamic compromise; in the long run, left ventricular hypertrophy is harmful[6]. Hypertension has long been implicated as the most important underlying cause of LV hypertrophy. Other factors implicated in the development of LV hypertrophy include obesity, age, dietary sodium intake, volume load, diabetes, arterial hypertrophy and stiffening, insulin resistance, and neurohumoral factors e.g. adrenergic factors and the renin-angiotensin system.

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ECG is relatively insensitive and can't accurately quantitate the severity of LVH. Also LVH is difficult to diagnose by ECG if left bundle branch block is present. Because of these limitations, other diagnostic modalities have been used for LVH assessment. The most successful and popular of these techniques has been echocardio-graphy[7]. Echocardiography has revolutionized the diagnosis of LVH because echocardiographic evidence of LVH occurs in 30 to 40 percent of hypertensive patients whose ECG and chest X-ray are normal[8].

Aims and Objectives of this study

1. To determine the electrocardiographic and echocardiographic evidence of LVH.
2. To correlate the electrocardiographic and echocardiographic evidence of LVH in patients with various etiologies of LVH.

Materials and Methods

This study has been carried out in medicine and cardiology indoor of Rajshahi Medical College Hospital from November 2004 to October 2005. 100 cases were selected for this study in random manner. Data were collected by preformed questionnaire. The cases were evaluated through proper history taking, thorough clinical examination, and appropriate investigations e.g. ECG, CXR, ECHO, and other relevant investigations. Electrocardiographically, LVH was diagnosed on the basis of increased voltage (LVH alone) and repolarization abnormality (LVH and Strain). Sokolow-Lyon criteria (S in V_1 + R in V_5 or $V_6 \geq 35$ mm) and Cornell voltage criteria (In men: SV_3 + RaVL > 28 mm in women: SV_3 + RaVL > 20 mm) were used to diagnose LVH electrocardiographically. Measurement of left ventricular wall thickness e.g. intenventricular septal thickness (IVST) and left ventricular posterior wall thickness in diastole (PWTd) by M-mode echocardiography was done. LVH was considered to be present if the IVST and PWTd are above their normal limits (> 12 mm in diastole).

Results

In this comparative study on electrocardiographic and echocardiographic evidence of left ventricular hypertrophy (LVH), we have found that, out of 100 patients 64 (64%) were male and 36 (36%) were female with a male female ratio of 1.78**:**1. Mean age of the patients with left ventricular hypertrophy was 51 ± 5.01 years with a range of 20-69 years. Systolic blood pressure was below 140 mmHg in 30 (30%) patients, within 140-159 mmHg is 23 (23%) patients and ≥ 160 mmHg in 47 (47%) patient. Diastolic blood pressure was below 90 mmHg in 41 (41%) patients, within 90-99 mmHg in 15 (15%) patients, within 100-109 mmHg in 11 (11%) patients and ≥ 110 mmHg in 33 (33%) patients. ECG changes were interpreted in all the patients. ECG was normal in 4 (4%) patients, LVH alone was found in 34 (34%) patients, LVH with ST-T change in 46 (46%) patients, only ST-T change in 12 (12%) patients, LBBB in 3 (3%) patients and old MI in 1 (1%) patients. So, overall, LVH was found in 80 (80%) patients. Each patient was studied with a combination of M-mode and 2-Dimensional echocardiography with color-flow Doppler study when needed. 66 (66%) patients were found to have concentric LVH and 24 (24%) patients had eccentric type of LVH. Echocardiography revealed no abnormality in 10 (10%) patients. Of the 80 (80%) patients having ECG-LVH. 70 (87.5%) of them also found to have Echo-LVH but 10 (12.5%) of them had no LVH in echocardiography. On the other hand 20 patients having no LVH in ECG was found to have LVH in echocardiography. When sensitivity and specificity of ECG in comparison to echocardiography in diagnosing LVH was calculated, it was found to be 87.50% and 50% respectively. When a comparison between ECG and Echo findings were made it was found that of the 34 patients having LVH alone in ECG, 18 of them had concentric LVH, 10 of them had eccentric LVH and 6 of them had normal findings in echocardiography. In patients having LVH with ST-T change in ECG, 32 of them had concentric LVH, 10 of them had eccentric LVH and 4 of them had normal finding in echocardiography. ECG changes were correlated with etiology of LVH and hypertension (56%) and aortic stenosis (8%) were the two most important cause of ECG-

LVH. In echocardiography concentric hypertrophy was caused by hypertension in 50 (50%) patients and by aortic stenosis in 10 (10%) patients. On the other hand eccentric hypertrophy was caused hypertension in 8 (8%) patients and by multiple valvular diseases in 4 (4%) patients. All 10 patients (10%) having normal echocardiogram had hypertension as etiology.

Table-1 Showing the ECG changes (N=100)

ECG findings	Number	Percent
Normal	04	04
LVH alone	34	34
LVH with ST-T change	46	46
ST-T change	12	12
LBBB	03	03
Old MI	01	01

Table-2 Showing Echocardiographic findings (N=100)

Echo findings	**Number**	**Percent**
Concentric LVH	66	66
Eccentric LVH	24	24
Normal	10	10

Table-3 Comparison between ECG and Echo findings (N=100)

ECG changes	**Echocardiographic findings**		
	Concentric LVH	**Eccentric LVH**	**Normal**
Normal	04	00	00
LVH alone	18	10	06
LVH with ST-T changes	32	10	04
ST-T changes	08	04	00
LBBB	03	00	00
Old MI	01	00	00
Total	66	24	10

$$\text{Sensitivity} = \frac{TP}{TP + FN} \qquad \text{Specificity} = \frac{TN}{TN + FP}$$

TP = True positive TN = True negative

FP =False positive FN = False negative

ECG Test	LVH	
	Present	Absent
+ ve	TP=70	FP=10
- ve	FN=10	TN=10
Echo	LVH	
	Present	Absent
+ve	TP=80	FP=0
-ve	FN=0	TN=20

Sensitivity of ECG in diagnosing LVH =

$$\frac{70}{70+10} \times 100 = 87.50\%$$

Specificity of ECG in diagnosing LVH=

$$\frac{10}{10+10} \times 100 = 50\%$$

Discussion

ECG is very sensitive (90%) but less specific (20-60%) in diagnosing left ventricular hypertrophy [9].We also found that out of 80 patients having ECG-LVH, echocardiographic evidence of LVH was found in 70 of them. The specificity of ECG criteria in this study was 50%, which is consistent with various other studies.[10,11] In our study, most (66%) patients had concentric LVH and only 24% had eccentric LVH. This is due to the fact that pressure overload (e.g. hypertension, 68%) were more common than volume overload (e.g. multiple valvular heart disease, 4%) in our study and pressure overload usually cause concentric LVH. In our study we have found that, when only LVH is found in ECG, it is less consistent with Echo-LVH than when LVH with strain is found in ECG. This is evidenced by the fact that 82.35% of patient having only LVH in ECG had echocardiographic LVH. On the other hand 91.30% of patients having LVH with strain also had Echo-LVH. This finding is very much consistent with the Copenhagen City Heart Study.[12] Sensitivity of ECG in comparison to Echocardiography was calculated to be 87.50% which is consistent with various other studies.[9]

Conclusion

Left ventricular hypertrophy is a serious condition, strongly associated with the development of coronary artery disease, cerebrovascular disease, cardiac failure, sudden cardiac death, and overall mortality. So, while managing a patient with hypertension the goal should be regression of LVH along with reducing BP to target level. ECG is not as good as Echocardiography to detect LVH, so wider use of Echocardiography is advocated.

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