"CORRELATION BETWEEN THE ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY DIAGNOSIS OF LEFT VENTRICULAR HYPERTROPHY IN PATIENTS WITH ESSENTIAL HYPERTENSION"

By

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LIST OF ABBREVIATIONS USED

ACE -Angiotensin Converting Enzyme

ECG -Electrocardiogram

HTN- Hypertension

JNC -Joint National Committee

LV - Left ventricle

LVH-Left Ventricular Hypertrophy

LVMI -Left Ventricular Mass Index

RAS-- Renin Angiotensin System

RES- Romhilt –Estes score

ABSTRACT

Background: Hypertension is the most important health problem met by general physicians. The development of left ventricular hypertrophy increases with the severity of hypertension and presence of increased left ventricular mass is associated with increased incidence of other target organ damage. Early detection of LVH and appropriate treatment decreases the development of left ventricular hypertrophy and reduces the mortality and morbidity.

Objectives:

1. to correlate electrocardiography and echocardiography findings of left ventricular hypertrophy in patients with essential hypertension.
2. To correlate the duration of hypertension with investigation findings like electrocardiography and echocardiography.

Methods:

50 patients of essential hypertension attending outpatient clinic and inpatients admitted to R.L.Jalappa General Hospital and research Centre, Kolar are included in the study Data is collected and investigation are done between Nov 2008 to Dec 2009.

Results:

mean age of patients is 51.74%,male:female ratio=1.38:1,prevalence of LVH is 80%. More in male compared to female, Prevalence of LVH increased with age -Smoking is significant risk factor for LVH. Retinopathy is more prevalent in patients with LVH as compared to patients without LVH, the sensitivity of ECG –Romhilt-Estes score(RES) was 47.5% and voltage criteria 60%, specificity of both is 100%.

Conclusion:

2D- Echocardiography is more specific and accurate to identify LVH and
Assessing the prognosis and end organ damage in hypertensive patients

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INTRODUCTION

The prevalence of hypertrophy increases with the severity of hypertension and presence of increased left ventricular mass is associated with greater incidence of other target organ damage. Left ventricular hypertrophy is associated with systolic and diastolic function abnormalities, ventricular arrhythmias which are one of the causes of sudden cardiac death among hypertensives and hence left ventricular hypertrophy is an independent predictor of morbidity and mortality. There are many ways of diagnosing left ventricular hypertrophy like ECG, chest x-ray and echocardiography having varied efficiency.

An elevated arterial pressure is probably the most important public health problem in developed and developing countries. It is common asymptomatic, readily detectable, usually easily treatable, and often leads to lethal complications if left untreated. As a result of extensive educational programmes in the late 1960s and 1970s by both private and government agencies, the number of undiagnosed and untreated patients was reduced significantly by the late 1980s to a level of approximately 25% with a concomitant decline in cardiovascular mortality. Unfortunately by mid 1990s, this trend began to wane. The number of undiagnosed patients with hypertension increased to nearly 33%, the decline in cardiovascular mortality flattened, and the number of individuals with chronic diseases with untreated or poorly treated hypertension increased.

The prevalence of hypertension depends on both the racial composition of the population studied and the criteria used to define the condition. In a white sub urban population like that in the Framingham study, nearly one-fifth of individuals have

blood pressure > 160/95 mm Hg, while almost one half have pressures > 140/90 mm Hg. An even higher prevalence has been documented in the non white population[1].

Hypertension in Indian Prospective:

There are no well-coordinated national surveys of prevalence of hypertension available from Indian subcontinent.Several regional small surveys with varying protocols have reported a prevalence which varies widely from 3.80 to 15.63% in men and 2 to 15.38% in women in urban areas and 1.57 to 6.93% in men and 2.38 to 8.81 % in women in rural areas[2].

Hence this study is undertaken to compare the diagnostic efficiency of these methods in diagnosing left ventricular hypertrophy because the best means of decreasing the increased mortality and morbidity is to prevent the development of left ventricular hypertrophy which requires early and continuous antihypertensive therapy even before the hypertrophy becomes clinically manifest.

OBJECTIVES

1. To correlate electrocardiography and echocardiography findings of left ventricular hypertrophy in patients with essential hypertension.
2. To correlate the duration of hypertension with investigation findings like electrocardiography and echocardiography.

REVIEW OF LITERATURE

HISTORICAL REVIEW

The cardiovascular consequences of hypertension have long been appreciated. In1913, the famous clinician Thomas Janeway described congestive heart failure as the manifestation of hypertensive cardiovascular disease[3]. Shortly after, the relationship between hypertension and hypertrophy of the left ventricle was established in observations linking cardiac findings to the then new technique of indirect blood pressure measurement with sphygmomanometry. Although left ventricular hypertrophy (LVH) was quickly associated with congestive heart failure, the relationship of hypertension to cardiovascular morbidity and death is, in fact, complex. Whereas in the past there had been a presumption that hypertension eventuated in end stage cardiomyopathy, characterized by a poorly contractile, dilated left ventricle, this has not been proven in humans. Echocardiographic studies of cardiac anatomy and function in hypertensive subjects have disclosed concentric LVH as the usual cardiac finding[4-7]. Although a small number of patients in such studies may have dilated cardiomyopathy, other sources such as ischemic heart disease or idiopathic cardiomyopathy have been difficult to exclude with certainty.

The m-mode Echocardiography provides an accurate assessment of left ventricular mass that is more sensitive and specific than ECG for detecting left ventricular hypertrophy[8]. Likewise the Echocardiographic left ventricular hypertrophy is more prevalent and more sensitive for ventricular arrhythmias than electrocardiographic ventricular hypertrophy[9].Left ventricular hypertrophy is a major cardiac alteration associated with hypertension accounting for a risk that is independent of the elevated arterial pressure. Diastolic dysfunction is an early manifestation of impaired ventricular function associated with hypertrophy later

causing systolic dysfunction and cardiac failure. The best means of decreasing the increased mortality and morbidity is to prevent the development of left ventricular hypertrophy, which requires early and continuous Antihypertensive therapy even before the hypertrophy, becomes clinically manifest[10].

The classic form of LVH, concentric LVH is defined as thickening of septum and the posterior wall of the left ventricular at the expense of chamber volume, which is the typical ventricular adaptation to an increase in after load that occurs after long standing hypertension. Eccentric LVH is defined as thickening of the chamber wall with concomitant chamber dilatation which occurs in the later phase of hypertensive heart disease and is a precursor of congestive cardiac failure[11].

Furthermore the echocardiogram provides an extremely sensitive means for the clinical demonstrate on of left ventricular hypertrophy, greater than that provided by the ECG and far greater than that of the chest roentgenogram[12]. Hypertension places increased tension on the left ventricular myocardium that is manifested as stiffness and hypertrophy which accelerates the development of atherosclerosis within the coronary vessels. The combination of increased demand and lessened supply increases the likelihood of myocardial ischemia and thereby leads to a higher incidence of myocardial infarction, sudden death, arrhythmias and congestive failure in hypertensive. In the past left ventricular hypertrophy was recognized on ECG by increased voltage QRS complexes, intrinsicoid deflection over lead V2 or V3 greater than 0.06 seconds and ST segment depression greater than 0.5mm. Increasingly echocardiogram is being used because it is much more sensitive in recognizing early cardiac involvement. By echocardiography, left ventricular mass is shown to progressively increase with increases in blood pressure. Left ventricular

mass is greater in those whose pressure does not fall during sleep because of a more persistent pressure load[13]

The estimation of left ventricular mass by echocardiogram offers prognostic information beyond that provided by the evaluation of traditional cardiovascular risk factors. An increase in left ventricular mass predicts higher incidence of clinical events including death attributable to cardiovascular disease[14]. Accurate assessment and quantification of left ventricular function is of critical importance to clinicians because of the close relationship between left ventricular ejection fraction and long term prognosis.

Despite the emergence of other techniques such as radionuclide imaging and MRI, echocardiography remains the primary non-invasive modality for assessment of regional and global left ventricular function.[15]Nonetheless, the principal cardiac manifestation of isolated hypertension, i.e., increase in left ventricular (LV) mass primarily from increase in LV wall thickness, sufficient to be defined as LVH, is associated with clinically important abnormalities of diastolic, electrophysiologic and systolic function[16-18]. The importance of LVH, in itself, in the morbidity of hypertensive disease has been underscored by the number of electrocardiographic (ECG) and echocardiographic studies that have convincingly demonstrated its importance as an independent predictor of morbidity and mortality[19,20.]

The magnitude of LV mass as a continuous variable is also associated with cardiovascular risk, even with values for LV mass within the "normal range". In the Framingham heart Study, it was shown that for each 50 g/m increase in LV mass(corrected for height) there was a relative risk for mortality of 1.73, even in subjects free of clinically apparent cardiovascular disease[20]. It was also demonstrated that this risk was statistically independent of blood pressure, age, antihypertensive

treatment, and other cardiovascular risk factors. Additionally, the pattern of LVH as well as the magnitude of increase in LV mass is important in predicting the risk of LVH for cardiovascular morbidity. Individuals with a higher relative wall thickness at any value of LV mass have greater risk for cardiovascular events. The relative wall thickness is the proportion wall thickness to ventricular cavity size. A high number indicates "concentric" (thick wall cavity) LVH, as is most commonly found in hypertension, in contrast to "eccentric dilated" (thin wall dilated cavity) hypertrophy that would be represented by a lower relative wall thickness is found in ischemic, alcoholic, and other forms of dilated cardiomyopathy. Importantly, there is an association of LVH with complex ventricular arrhythmia, a possible precursor of sudden death in hypertensive patients even in patients without coronary artery disease as shown on angiography[17].

PATHOPHYSIOLOGY OF LVH

Cardiac hypertrophy is a compensatory change that the heart undergoes when subjected to an increased work load in response to increased pressure resistance (afterload) or volume overload (preload). This definition applies to both atria and ventricles. Cardiac hypertrophy is associated with a significant increase in numbers or size of sarcomeres within each myocardial cell. An increase in preload leads to stretching of cardiac muscle, which invokes the frank-starling relationship, and subsequent increase in force of contractions in order to increase the stroke volume although the systolic arterial pressure is an estimate of after load, the Laplace relationship involving wall stress is a better estimate of afterload[21]. According to Laplace relationship, the tension in the left ventricular wall during systole is equal to the product of mean pressure in the wall of ventricle, and the degree of left ventricle wall stress is directly proportional to intracavity pressure and radius of chamber and inversely proportional to wall thickness. In concentric left ventricular hypertrophy, left ventricular wall stress remains normal despite elevated intracavity pressure because the thickness of wall is increased but the chamber volume is not[22].

The cardiac myocytes are terminally differentiated and therefore undergo hypertrophy than hyperplasia. The type of cardiac overload that is present determines the pattern of hypertrophy. Volume overload produces increased ventricular cavitary volume in proportion to man, called eccentric hypertrophy, whereas pressure overload produces increased left ventricular mass out of proportion to volume, called concentrichypertrophy[23].

Sustained pressure in cardiac chambers leads to changes in gene expression that cause an increase in protein synthesis. The stimulus for hypertrophy appears to be an increase in wall stress of chamber and this stress in pressure overload leads to

replication of sarcomeres, an increase in cell width, ventricular wall thickening and concentric hypertrophy without increasing the number of myocytes. Ultra structural morphometric studies demonstrate that early in pressure overload hypertrophy there is increased ratio of mitochondria to myofibrils where as in stable, compensated hypertrophy this ratio is decreased[24].

Left ventricular hypertrophy occurs as compensation for an increased after load or as a response to an increase in preload. Systolic function is usually preserved in patients with hypertension as demonstrated by a normal relation between ejection fraction and wall stress until late in course of illness[25]. The total oxygen consumption of the heart, already increased by work of expelling blood against a raised pressure is increased further because there is more muscle, therefore any decrease in coronary blood flow has more serious consequences in hypertensive patients that it does in normal individuals, and degrees of coronary narrowing that do not produce symptoms when the size of heart is normal may produce infarction when the heart is enlarged[26].

Although concentric LVH maintains systolic function at a near normal level, left ventricular relaxation is impaired with long standing pressure overload reflecting reduced distensibility of left ventricle and therefore high prevalence of impaired early diastolic filling without systolic dysfunction in hypertensive patients[27,28.] This compensated phase of hypertrophy progresses to decompensation with advanced left ventricular hypertrophy and the transition to systolic dysfunction reflects the severity and duration of hypertension[12].

The degeneration of myofibrils and reduction in size of intracellular myofibrils is consistent with decompensated pressure overload. The preservation of most other organelles distinguishes this from necrosis[29].There are also other mechanisms apart from hemodynamic factors which contributes to the left ventricular hypertrophy like[30]

- Raf – 1 Kinase.[31]
- Increased activity of Renin-angiotensin system.[30]
- Increased sympathetic activity by cardiac norepinephrine.[32]
- Age of onset of pressure overload.[30]
- Hormones like vasopressin, catecholamines, insulin like growth factor, platelet derived growth factor, fibroblast growth factor[33.]
- Aldosterone synthase (CY P II B2).[34]
- Impaired vascular endothelin function[35].

A number of studies have supported the view that each component of Renin Angiotensin system (RAS) is synthesized in the ventricle. Cardiac RAS has been documented to participate in the development of left ventricular hypertrophy. There is a strong evidence of intra cardiac generation of angiotensin-II, which is a strong vasoconstrictor and pressor agent in the pathophysiology of hypertensive left ventricular hypertrophy. The stretch of myocyte stimulates angiotensin-II secretion which in turn promotes protein kinase synthesis-protein Oncogene (c-Myc) resulting in cardiac cellular hypertrophy[36.]

Increased angiotensin converting enzyme (ACE) activity has been demonstrated in hypertensive-left ventricular hypertrophy. It is shown that ACE is marked in sites of fibrosis, irrespective of pathological basis and ACE may be responsible for regulating local concentration of angiotensin-II and bradykinin that govern fibroblast-collagen takeover.

Other hormones like aldosterone, endothelin, epinephrine, prostaglandins, bradykinin, growth and thyroid hormones may also be responsible for process of fibrosis and collagen deposition. This increased fibrous content and end-diastolic

volume ratio are further responsible for the increase in myocardial stiffness, resulting in the increase of end diastolic pressure.

It has long been recognized that asymmetric septal thickening may also be an early and frequent abnormality in subjects with systemic hypertension[37]. This is due to the larger bending radius compared with the posterior wall. The septum may tend to develop a greater tension during contraction and therefore hypertrophy could appear earlier and progress faster at septal level than at level of posterior wall for any given level of blood pressure[38]. This asymmetric septal hypertrophy is also accompanied with increased cardiovascular risk.

DIAGNOSIS OF HYPERTENSIVE HEART DISEASE

Hypertensive heart disease is recognized by noninvasive methods which detects the ventricular hypertrophy and the ventricular function[9]. This includes the clinical evaluation, and laboratory methods like chest roentgenogram, electrocardiography and echocardiogram.

Clinical evaluation:

The risk factors for left ventricular hypertrophy are systolic and diastolic blood pressures and the clinical severity of hypertension depends on these systolic and diastolic pressures.

The classification is according to the JNC 7 category.

Category	Systolic (mmHg) Pressure	Diastolic (mmHg) Pressure
Normal	< 120	< 80
Pre hypertension	< 120-139	85-89
Hypertension	> 140	> 90
Stage I	140-159	90—99
Stage II	≥ 160	≥100

The cardiovascular examination may suggest hypertrophy or hyper dynamic circulation.

Hypertrophy: Forceful and sustained apical impulse that has an outward thrust and Lasting two-third or more of the duration of systole.

Hyper dynamic apex: Unsustained apical impulse with a diameter of greater than 2.4cms Left ventricular hypertrophy may be suspected on basis of lateral displacement of apical impulse. Apical impulse> 3 cm in the left lateral decubitus position is more sensitive and specific of left ventricular hypertrophy than location of apex ten cm or more from mid sternal line or lateral to mid clavicular line[12]. Presence of third heart sound or fourth heart sound indicates ventricular decompensation or diminished ventricular compliance. Aortic ejection murmurs may be present due to degenerative changes in aortic valve.

Chest X-Ray:

A useful preliminary investigation in cardiology to look for gross chamber enlargement. Concentric left ventricular hypertrophy results in wall thickening at expense of cavity size producing only a slight alteration of cardiac size. Left ventricle enlarges inferiorly, posteriorly and to left, often increasing cardiothoracic ratio. If cardiothoracic ratio is >0.5, it is suggestive of left ventricular hypertrophy. Although easy to perform and inexpensive, the sensitivity of chest X-ray in diagnosing left ventricular hypertrophy is too low[39].

Electrocardiogram:

Electromotive force generated by cardiac muscle is largely proportional to its mass; electrocardiography based largely on the magnitude of this electromotive force recorded from the chest wall became widely applied in clinical studies of LVH.

It is the only practical method of recording, electrical activity of the heart. ECG and echocardiography are the major methods of detecting LVH: ECG LVH is a strong predictor of cardiovascular morbidity and mortality as compared to systolic blood pressure, diastolic blood pressure, serum lipids and smoking. ECG LVH patients have substantially higher risk of coronary heart disease, angina, myocardial ischemia, congestive cardiac failure and sudden death. Risk of sudden death in ECG LVH patients is six fold elevated in men and three folds in women. ECG LVH is sensitive diagnostically and a powerful prognostic predictor[40].

The electrocardiographic changes are as follows.

i) Increased voltage of QRS deflection[41]:

In the production of the normal left ventricular surface pattern in leads V5and V6, the R deflection results from the stimulus traveling through the left ventricular wall towards the electrode. Similarly, in the recording of the normal right ventricular surface pattern in lead V1, the S wave is also produced by the activation wave in the left ventricular wall traveling away from the electrode. In the presence of ventricular hypertrophy with increased electrical forces from the hypertrophied left chamber, leads over the left precordium. V5 and V6 shows high amplitude 'R' waves and the right precordial lead V1shows a deep S wave. In the presence of left ventricular hypertrophy the disproportion between the left and right ventricular forces generated during simultaneous activation of both ventricles is exaggerated. The increased magnitude of the left ventricular forces without appreciable change in the right ventricular forces reacts in an increased magnitude of the mean QRS special vector and rotation of the vector towards the hypertrophied left ventricle. The increased QRS vector is therefore oriented more posteriorly, superiorly, and to the left than normally. The limb leads usually show left axis deviation. The transition zone is displaced to the

right (V2) or if left ventricular hypertrophy is marked and the heart position is strongly horizontal, the transition zone is displaced to the left and superiorly (V5or V6) clockwise rotation on the longitudinal axis. Some of the theories that have been postulated to account for the increased magnitude of the left ventricular forces.

ii) (QRS) voltages are as follows:

a. The resultant vector is magnified by the increased preponderance of the left ventricular wall; in other words, left ventricular activation continues unopposed by right ventricular activation for a greater than normal period.

b. The hypertrophied fibre has diminished internal resistance as a result of its increased cross sectional area. This tends to increase the magnitude of the potential.

c. The enlarged ventricle exposes a greater portion of the heart to the exploring electrode.

These are two possible explanations for the inverted wave in leads over the left precordium.

a. The T wave changes may be primary, due to myocardial ischemia resulting from the relative disproportion between the increased ventricular mass and the available blood supply.

b. The changes may be secondary i.e. if the ventricular gradient remains the same, the T wave area must decrease as the ST area increases.

iii) Delayed onset of intrinsicoid deflection over the left ventricle (Delayed ventricular activation time)[41]:

The onset of intrinsicoid deflection is measured from the onset of QRS to the peak of the R wave and represents the time interval for passage of the impulse through the ventricles to the epicardium underlying the exploring electrode. An increased mass of myocardium, as in the left ventricular hypertrophy, prolongs the

time interval for the passage of stimulus to the epicardial surface. The onset of intrinsicoid deflection in left ventricular hypertrophy thus occurs later than it would normally in the left precordial leads V5 and V6. The onset of the intrinsicoid deflection over the right ventricle remains early.

iv) Increased duration of QRS complex:[41]

As a consequence of the increased muscle mass the activation must travel a longer than normal course, and the QRS complex is widened. The normal duration of the QRS measured in the standard limb leads is considered to be 0.10 seconds or less. In left ventricular hypertrophy the QRS interval in the standard limb leads may be prolonged to 0.11 or even 0.12 seconds.

v) Left axis deviation:[41]

In left ventricular hypertrophy the mean manifest QRS axis may have a leftward orientation. Several explanations have been offered:

a. The left ventricular mass is greatly increased.

b. There may be an anatomical change in the position of the heart.

c. Some degree of left bundle branch block may be present.

d. Peripheral or partial block may be present.

Based on the changes described above various criteria had been proposed for the diagnosis of left ventricular hypertrophy. The important criteria used are:

1. Sokolov-Lyon voltage criteria:[41]

In 1949 Sokolov and Lyon pointed out that the presence of left ventricular hypertrophy in adult is suggested when the sum of S wave in V1 and R wave in V5 or V6 potentials are more than 35mm. The other voltage criteria based by them are – Rin V5 or V6 more than 25 mm. R more than 11mm in AVL. R1+ SIII more than 25 mm etc., out of these SV1+RV5 or v6 is the most commonly used criteria. Although the

voltage criteria are easy and quick, various studies have shown that it has a sensitivityof 20% and specificity of > 90%.

2. Romhilt and Estes point score system:[42]

Voltage criteria alone have low sensitivity and specificity for diagnosis of left ventricular hypertrophy. To increase the sensitivity of an ECG for diagnosis of left ventricular hypertrophy Romhilt and Estes in 1968 developed a point scoring system.

The system is as follows:

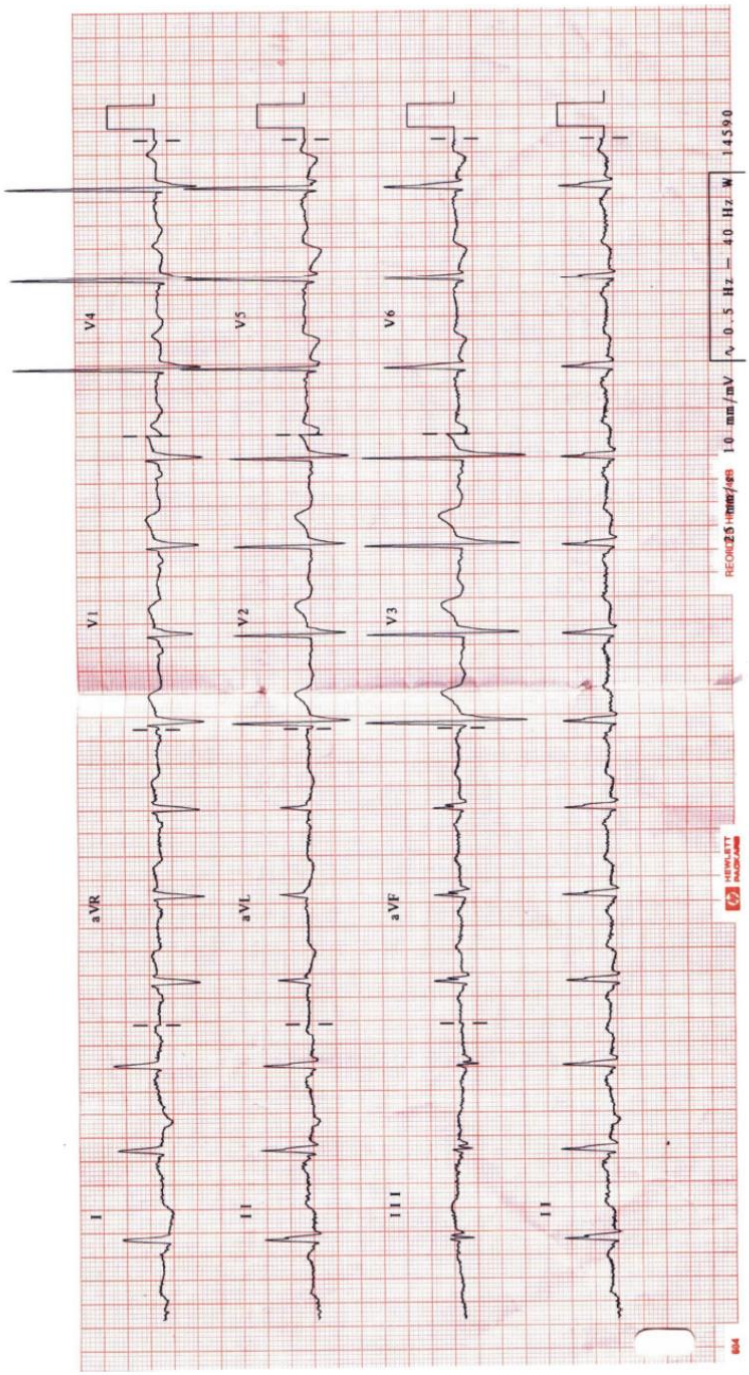
1.	R or S in limb leads S in V_1, V_2 or V_3 R in V_4, V_5or V_6	20 mm or more 25 mm or more 25 mm or more	3 points
2	Any ST –T changes		3 points
3	A 'P' wave of left atrial Enlargement in V_1 (P-terminal force> 0.04 sec)		3 points
4	Left axis deviation of -15^0 or more		2 points
5	Intrinsicoid deflection in V_5, V_6 o.04 sec or more		1 point
	Total		**12 points**

The maximum number of points an ECG can score is 12. Five or more points are diagnostic of left ventricular hypertrophy. Unfortunately, these criteria, offer limited sensitivity for the detection of LVH when compared with autopsy findings.In reality the increased magnitude of the QRS deflection is probably due to a combination of these three factors rather than to any single one.

vi) ST Segment and T wave changes over the left ventricle: [41]

The ST segment is depressed and T wave is inverted in leads V5 and V6 or other leads over the left ventricle. In the right precordial leads (V1, V2) where theQRS is predominantly negative, the T wave is upright and the ST segment may be slightly elevated. ST segment and T wave changes of this type are secondary and presumably result from the altered ventricular depolarization process in the presence of left ventricular hypertrophy. The overall balance of the T forces in the hypertrophied ventricle causes the 'T' vectors to be oriented away from the QRS vectors. Thus a wide QRS –T angle results.

The ST segment is normally isoelectric for the following reason: repolarization begins shortly before ventricular activation is completed, however the potential of early repolarization does not reach sufficient magnitude at the completion of ventricular activation to be recordable. When the repolarization forces reach sufficient potential to be registered, the 'T' wave is inscribed. In left ventricular hypertrophy, left ventricular activation is prolonged, hence by the time ventricular activation is completed, the potential of repolarization is of sufficient magnitude to produce a deviation of the ST segment of LVH, which range from 20% to approximately 50% depend on how criteria are adjusted to optimize the inverse relationship between sensitivity and specificity. Recently, large data basis using echocardiographically measured LVH have been utilized to improve ECG criteria for estimation of LV mass[43]. Despite the limitations of ECG in the detection of LVH, theca had identified LVH as a risk factor for cardiovascular morbidity and mortality in hypertension. Moreover, ECG studies have, detected regression of LVH with drug therapy.



ECG showing left ventricular hypertrophy with strain pattern

Echocardiographic evaluation of left ventricular hypertrophy:

The diagnostic noninvasive tool in cardiovascular diagnosis which is safe and informative is echocardiography. The assessment of left ventricular function is an essential component of evaluation of hypertensive patient. Today it is possible to obtain a fairly extensive and comprehensive evaluation of left ventricle[44].

Echocardiography is an extremely sensitive diagnostic tool for detecting LVH. It detects its presence in very early stage and also identifies various forms of LVH.

Concentric LVH is an increase in LV mass and increase in relative wall thickness (RWT), Eccentric LVH is an increase in LV mass and normal RWT, Concentric remodeling is normal LV mass and increase RWT and normal geometry is normal LV mass with a normal RWT[45].

Impaired LV diastolic dysfunction detected by Doppler echocardiogram identifies hypertensive patients at increased cardiovascular risk[46.]

In echocardiography ultrasound waves are passed through the heart and the returning echoes details the position and movement of the cardiac acoustic interfaces. Cardiac ultrasound presently consists of three inter-related modalities: M-mode echocardiography, two-dimensional echocardiography and Doppler echocardiography.

Most modern ultrasound systems, intergrade M-mode and two-dimensional imaging in a convenient, easy to use mobile unit. These units can display the two-dimensional image alone or simultaneously with an M-mode image, since one or two cursors are usually available to select from the two-dimensional sector, the acoustic scan lines to be displayed in M-mode.

Echocardiography now provides a simple non-invasive, reproducible means of assessing LVH For this reason recent studies evaluating ECG criteria for left ventricular hypertrophy have used echocardiographic left ventricular hypertrophy as standard[38]

The American society of echocardiography has accepted to standardize the common M-mode measurements and recommended that they use the “leading edge” method. That is, measurements from one structure to another should be made from the

leading edge of last echo produced by the anterior structure to the leading edge of the first echo produced by the posterior structure. It is also recommended that all measurements should be obtained by averaging five cardiac cycles recorded at end expiration, since inspiration causes an increase in right ventricular and a decrease in left ventricular volume.

For a diagnosis of left ventricular hypertrophy, left ventricular wall thickness and left ventricular mass are utilized. To obtain them the following recordings are required[47].

1. Inter ventricular Septal Wall Thickness (ST):

It is measured from the leading edge of the right septal echo to the leading edge of the left septal echo, at end diastole.

2. Posterior wall Thickness (PWT):

It is measured as the distance between the anterior endocardial echo and the anterior surface of the epicardial echo, at end diastole.

3. Left Ventricular Internal Dimension (LVIdD):

Is the distance between the left side of the interventricular septum and the posterior left ventricular endocardium at the level of chordae tendinae. End diastole is defined as the onset of the QRS.

4. Left Ventricular Mass:

The echocardiographic left ventricular mass can be obtained by the following formula: PENN'S CONVENTION FORMULA; (LVM = Leftventricular mass; LVIdD = Left ventricular internal diameter at end diastole; PWT =Posterior wall thickness; IVST = Interventricular septal thickness)

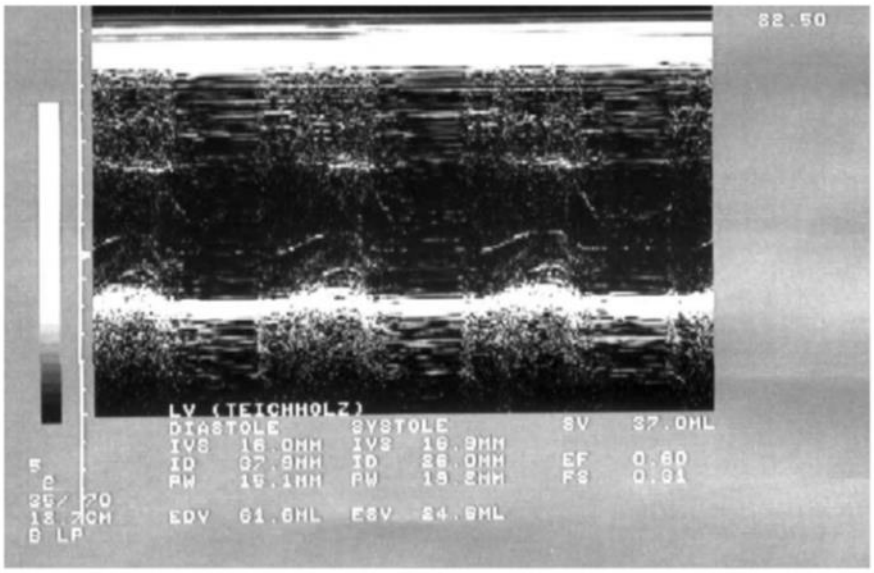
$$LVM = 1.04\ (LVIdD + PWT + IVST]^3 - LVIdD^3) - 14\ \text{gram.}$$

Left ventricular mass index (LVMI) = the normal left ventricular mass derived with this formula is divided by the body surface area. For the Indian population normal LVMI for males is 121gm/m^2 and 110gm/m^2 in females. Any value above this is suggestive of left ventricular hypertrophy[48].Various studies have shown that echocardiographic left ventricular mass correlates fairly well with the left ventricular weight. The posterior wall thickness correlated better with the left ventricular hypertrophy than the septal thickness. Many studies which had used the post mortem left ventricular weight as the standard have shown that the echocardiographic left ventricular mass has a sensitivity of 83% and specificity of 95% in diagnosing left ventricular hypertrophy[38]. These studies have shown that there is poor relative sensitivity of ECG criteria for left ventricular hypertrophy.

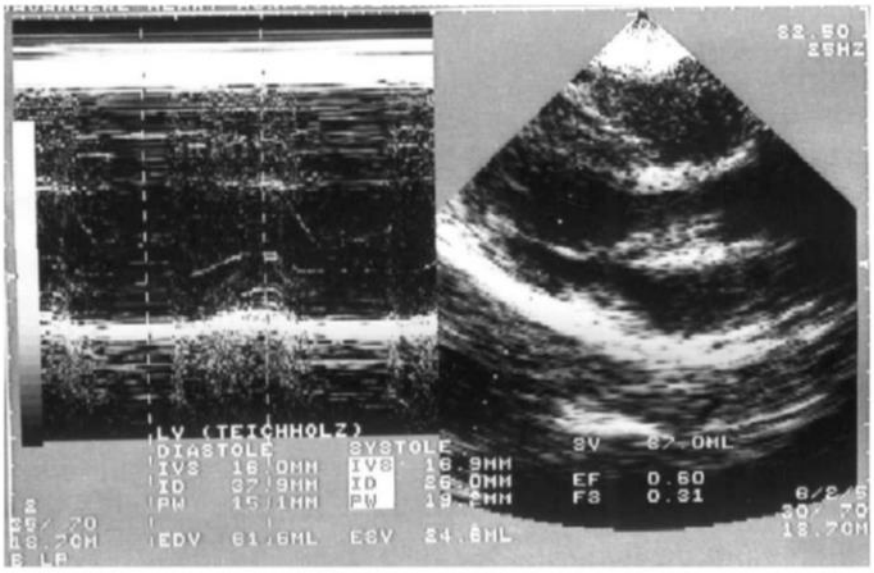
There have been over 150 studies using echocardiography to measure regression f LV mass with antihypertensive drug therapy[49] and echocardiography has found wide application in population study of hypertensive patients to define LVH prevalence and effects of hypertension on cardiac function and structure. As echocardiography has been a useful tool in clinical research, its place in clinical management of patient with hypertension is less certain.

Other techniques for measuring LV mass have included rapid cardiac computed tomographic (CT) scanning and magnetic resonance imaging (MRI). Although these techniques offer the potential of more reproducible measurement of LV mass, less dependent on technical and subject variability, they are not readily available at most institutions and expensive[50].

LVH measured by echocardiography independently predicts adverse cardiovascular outcomes in patients being treated for moderate hypertension[51]



M-Mode MV tip



M-Mode MV tip

The purpose of risk assessment is to assist clinicians in determining the appropriate treatment of individual patients to best protect against the various complications associated with hypertension.

Cardiovascular disease:

Abnormalities in the left ventricular function develop even before hypertrophy develops, in the form of diastolic dysfunction. With increasing hemodynamic load either systolic or diastolic dysfunction may evolve and progress to congestive cardiac failure.

Most episodes of congestive cardiac failure in hypertensive patients are associated with systolic dysfunction as reflected in a reduced ejection fraction. However about 40% of episodes of congestive heart failures are associated with diastolic dysfunction and preserved left ventricular systolic function[52].

Coronary heart disease:

Hypertension is the major risk factor for myocardial infarction and ischemia, mainly due to imbalance between myocardial oxygen supply and demand[53].

Mechanisms of coronary disease may be abnormal high resistance of coronary microvasculature in presence of left ventricular hypertrophy; limited coronary reserve reduces the expected increase in coronary blood flow in response to stimuli.

Acceleration of atherosclerotic narrowing of larger coronary arteries[54].Sudden death in patients with left ventricular hypertrophy is due to ventricular ectopic activity myocardial ischemia leading to ventricular dysarrhythmias like ventricular tachycardia or ventricular fibrillation, myocardial fibrosis and cardiac dysfunction[53].

The Framingham study has shown that age adjusted death rate in men with echo left ventricular hypertrophy was four times more than in men without left ventricular hypertrophy[55.]

Cerebrovascular disease:

Hypertension may accelerate cognitive decline with age. Hypertension particularly systolic is a major risk factor for both ischemic stroke and intra cerebral hemorrhage. Cerebral white matters lesions are a common finding by Brain MRI seen in 41 percent of asymptomatic middle aged hypertensive patients[52].Mechanisms may be due to arteriosclerosis and lipohyalinoses in small diameter penetrating cerebral end arteries Aggravating atherosclerosis in aortic arch and cervico cerebral arteries.

Promoting heart disease like left ventricular hypertrophy complicating stroke LVH is strongly associated with ischemic stroke in all age, sex and ethnic groups.Increased LV relative wall thickness imparts an increased stroke risk after adjustment for mass and is of additional value in stroke risk prediction[56].

Retinopathy:

Vascular changes in fundus reflect both hypertensive and arteriosclerotic retinopathy. The two process first induce narrowing of the arteriolar lumen (grade I) and then sclerosis of adventitia or thickening of arteriolar wall, visible as arteriovenous nicking (grade II) progressive hypertension induces rupture of small vessels, seen as hemorrhage and exudate (grade III) and eventually papilledema (grade IV)[52.] This is based on Keith and co-workers classification. Hypertensive patients with left ventricular hypertrophy were found to have higher incidence of grade III and grade IV retinopathy as compared to without left ventricular hypertrophy.

Nephropathy:

Hypertensive patients with LVH have greater incidence of renal involvement than those without LVH.

Hyalinization and sclerosis of walls of afferent arterioles referred as arteriolar nephrosclerosis which may range from microalbuminuria to end stage renal disease[57].

MANAGEMENT

To accomplish the broad goal of eliminating all blood pressure related disease in community the detection and treatment of hypertension must be complimented by prevention strategies.

This includes a population strategy to achieve a slight downward shift in the entire distribution of blood pressure in the community and a more intensive targeted strategy to reduce blood pressure in those who are at greater risk of hypertension. The latter includes persons with a high normal blood pressure, family history of hypertension, overweight, over consumption of sodium, physical inactivity and alcohol consumption[12].

Because most people in the general population are candidates for primary prevention interventions, small changes in blood pressure are likely to yield substantial health benefits in reducing the prevalence of hypertension as well as associated risk of cardiovascular disease.

It is estimated that a population wide reduction in diastolic blood pressure of 2mm hg would result in a 17% reduction in prevalence of hypertension as well as 15% reduction in stroke and TIA and 6% reduction in reduction of coronary heart disease[12].

The primary strategies for prevention of hypertension requires life style modifications like

a. Dietary sodium reduction
b. Alcohol moderation
c. Physical activity (Aerobic exercises) and weight loss.[58]
d. Potassium supplementation.

Pharmacologic treatment:

The occurrence of left ventricular hypertrophy in patients with hypertension reflects a structural adaptation of the heart to pressure overloading. The presence of left ventricular hypertrophy predicts cardiovascular risks independent of pressure, but it remains to be shown whether reducing left ventricular mass / hypertrophy reverses that risk and whether reversal of left ventricular hypertrophy should be a therapeutic goal. Not all antihypertensive agents in the same class of drugs have the same effects on cardiac mass. Generally speaking, all antihypertensive agents, when used for a long time, will reduce left ventricular mass but only certain drugs will reduce left ventricular mass in a period of weeks.[43,59]

In patients with essential hypertension and baseline ECG LVH, lower left ventricular mass during antihypertensive treatment is associated with lower rates of clinical end points additional to effects of blood pressure lowering and treatment modality[60].

Thus, short term therapy with diuretics and direct acting smooth muscle vasodilators (such as hydralazine) will not reduce left ventricular mass, where as short term therapy (4-8wks) with adrenergic receptor blockers, centrally acting adrenolytic drugs (methyldopa), ACE inhibitors and calcium channel blockers will reduce left ventricular mass rapidly.

These show that reversal of left ventricular hypertrophy in weeks may result from non hemodynamic mechanisms, where as reduction in left ventricular mass with prolonged therapy involves hemodynamic changes.

Left ventricular hypertrophy reversal with short term therapy may be due to certain mechanisms like inhibition of adrenergic or renin-angiotensin system, changes in intracellular calcium, and induction or inhibition of growth factors or proto-

oncogenes[49].If reversal of left ventricular hypertrophy is eventually proved to reduce risk, then the improvement in outcome is independent of associated reduction in blood pressure, improved coronary flow, or antiarrhythmic effects of antihypertensive drugs[23].The best means of reducing the increased morbidity and mortality is to prevent the development of LVH. This requires early and continued antihypertensive therapy even before the hypertrophy becomes clinically manifest. Prevalence of LVH is markedly increased both in subjects with an untreated hypertension and in treated hypertension compared with normotensive subjects[61].

It is tempting in clinical practice to use LV mass as an index of the efficacy of antihypertensive treatment and of the presence of elevated LV mass as an indication for therapy in patients with intermittent or border line hypertension, in whom therapy might otherwise be deferred.

METHODOLOGY

This study was conducted in the R.L.JALAPPA. Hospital and research Centre attached to Sri devaraj urs medical college during the years 2008-2009.

The study was done on 50 hypertensive patients attending outpatient clinic and those admitted in the medical wards.

The study group consisted of patients above the age of 18 years. All freshly detected and old cases of essential hypertension, irrespective of duration of hypertension and type of treatment receiving were taken into the study. The exclusion criteria were all cases of secondary hypertension, congenital heart disease and patients with valvular heart disease.

History, physical examination chest X-ray, standard 12 lead ECG and two dimensional echocardiography were done for all patients. The following clinical information was obtained, apart from investigations.

1. Duration of hypertension
2. Number and type of antihypertensive drugs.
3. Standard cuff blood pressure in supine and standing.
4. Body surface area calculated by using standard table.
5. Cardiovascular examination:

· Site and character of apical impulse

· Character of heart sounds

· Presence of abnormal heart sounds and murmurs.

6. H/O stroke or recurrent transient cerebral ischemia.
7. Ophthalmic examination for any evidence of hypertensive retinopathy.

8. Investigations:

· Fasting Blood Sugar

· B. Urea

· S. Creatinine

· Fasting lipid profile

· Urine

- Alb

- Sug

9. Chest X-ray: PA view- to measure cardiothoracic ratio.

Electrocardiogram:

Standard 12 lead ECG was obtained in all patients with BPL –Cardart 108 electrocardiograph.

The ECG criteria used in this study are

1. Sokolov –Lyon index:

S in V_1+R in V_5/V_6 >35mm.

2. Romhilt –Estes point Score system:

1.	R or S in limb leads	≥ 20 mm	3 points
2.	ST – T Changes		3 points
3.	'P' terminal force in V_1 more than 0.04 sec		3 points
4.	Left axis deviation of -15^0 or more		2 points
5.	Intrinsicoid deflection in V_5, $V_6 \geq 0.04$		1 point
	Total		**12 points**

Left ventricular hypertrophy is considered to be present if total score is five or more.

Echocardiographic Studies:

Combined m-mode and 2-dimensional echocardiographic studies were performed using an advanced technology laboratories ultramark 6 ultrasound. All patients were positioned in a 30^0 left decubitus position with slight elevation of the head.Comprehensive 2-D tomographic planes were employed with multiple parasternal views of left ventricle in long and short axis, apical four chamber and long axis view and subcostal four chamber and short axis views. After positioning of the cursor through intraventricular septum and posterior wall, at the level of chordae tendinae, simultaneous M-mode and two dimensional recording were obtained from the parasternal transducer position in both long and short axis views of the left ventricle.

Measurement: The left ventricular posterior wall and septum were measured at the time of atrial depolarization before the onset of notch. The left ventricle internal dimension was measured at the level of chordae tendinae, as the distance between the left side of interventricular septum and the posterior left ventricle. M-mode measurements were taken by the leading edge to leading edge technique as recommended by the American society of echocardiography. All measurements were averaged to the closest 1 mm from three good quality cardiac cycles.

The average sum of septal wall thickness and posterior wall thickness of 1.1cm was taken as normal. Any value above this was taken as evidence of left ventricular hypertrophy.

The left ventricular mass index was calculated by using: Penn's convention formula:

$$LVM = 1.04\ (LVIdD+\ PWT\ +IVST]^3\ -\ LVIdD^3)\text{-}14\ \text{gram.}$$

LVMI = LVM/BSA.

[LVM = left ventricular mass; LVIDd = left ventricle internal dimension in end diastole; PWT = posterior wall thickness; IVST = interventricular septal thickness; LVMI= left ventricular mass index; BSA=body surface area].

The normal left ventricular mass index for the Indian population [48] is:

1. Males-121g/m^2

2. Females - 110g/m^2

Any value more than this was considered as left ventricular hypertrophy.

Statistical analysis

Mean, standard deviation, sensitivity, specificity, probability value are obtained.

"P" value <0.05 is considered significant by using SPSS software.

PROFORMA

Name IP No

Age/sex DOA

Height DOD

Weight BSA

Address

1. Breathlessness: present/absent

Onset duration severity

PND /orthopnea

2. Palpitation : present/absent

Onset duration

Aggravating factor

Relieving factor

3. Chest pain : onset duration

Site radiation

Aggravating factors Relieving factors

4. Headache : present/absent

Site type

5. Visual disturbance: present/absent

6. Visual disturbance: present/absent

7. Syncope : onset/duration/frequency

7. Edema: onset duration

Site pitting/non pitting

Past history

Duration of hypertension

D/o of DM/HTN/TB

Family history

H/o HTN/DM

Treatment history

Hypertension diagnosed old new

duration

Current antihypertensive therapy

Personal history

Alcohol smoking

General physical examination:

Built nourishment

Pallor icterus clubbing

Cyanosis edema

JVP raised/not raised

Height weight BMI surface area

Vital signs

Pulse B.P

R.R temperature

Cardiovascular system

1. Peripheral vascular system

Pulse rate rhythm volume

character condition of vessel

Peripheral pulses

Radio femoral delay

2. Parasternal pulsation epigastric pulsation others

3. Apical impulse position character

4. Auscultation heart sounds added sounds

5. Other systems

6. Fundus examination RE LE

Investigation

1. cbc

2. RBS urea creatinine

3. Lipid profile

4. Urine alb sugar micro

5. Others

6. ECG

Rate rhythm axis

LVH voltage criteria +/_ Romhilt- Estes score

Echo cardiography

LVIDd

LVIDs

LVIPWd

LVPWs

IVSd

EF

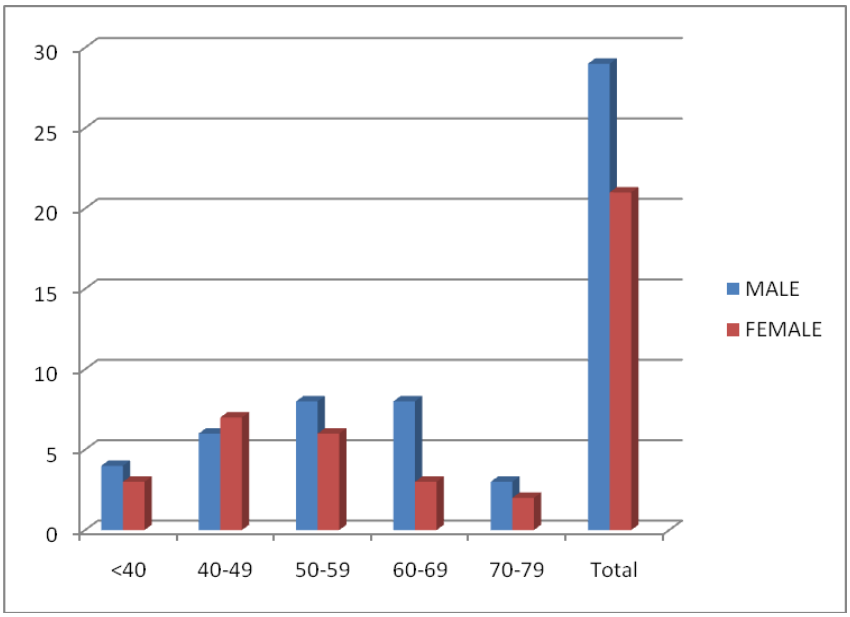
LVM=0.8(1.04(IVSDd+LVIDd+LVPWd) 3-LVIDd3)+0.6g

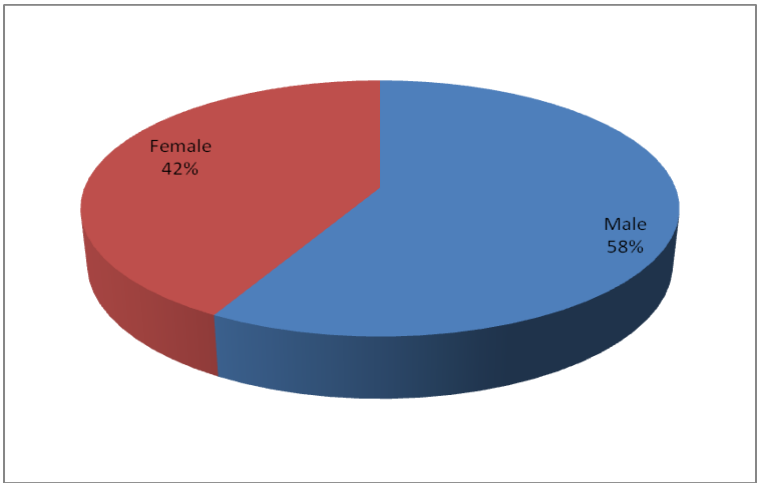
LVMI=LVM/BSA.

OBSERVATION AND RESULTS

Age and sex distribution of patients

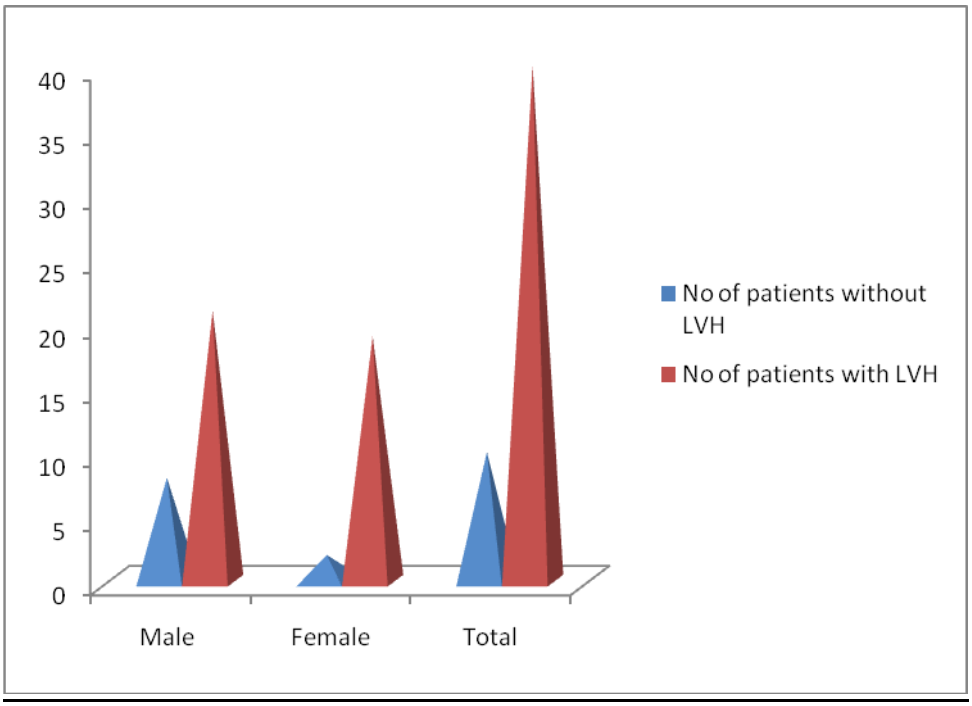
AGE GROUP(Years)	MALE	FEMALE	Total
<40	4	3	7
40-49	6	7	13
50-59	8	6	14
60-69	8	3	11
70-79	3	2	5
Total	29	21	50





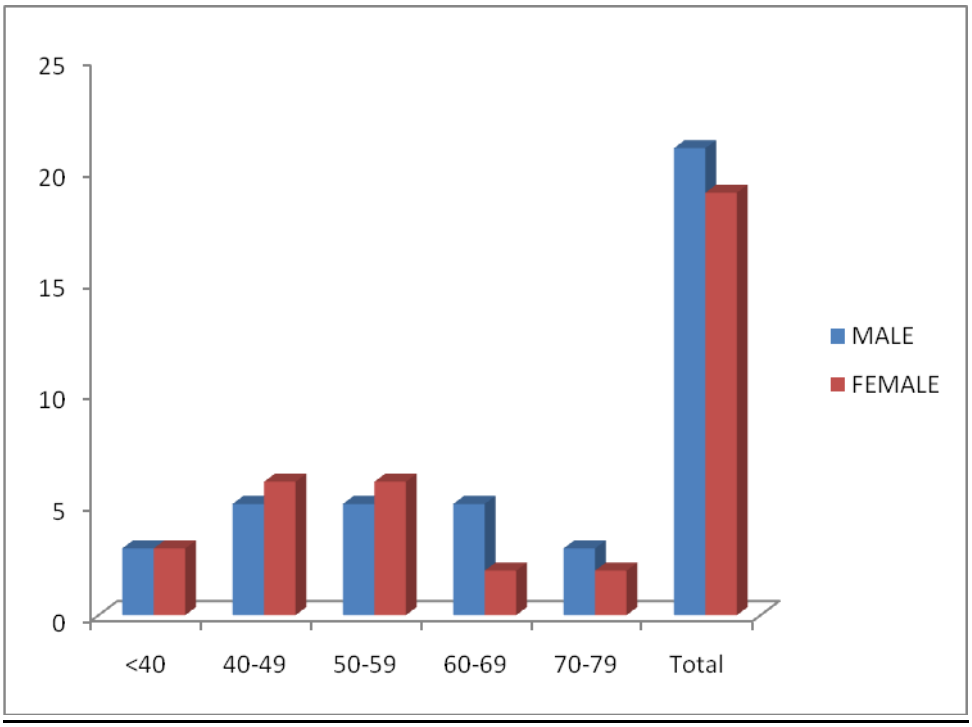
Prevalence of LVH in study population

	No of patients without LVH	No of patients with LVH
Male	8	21
Female	2	19
Total	10	40



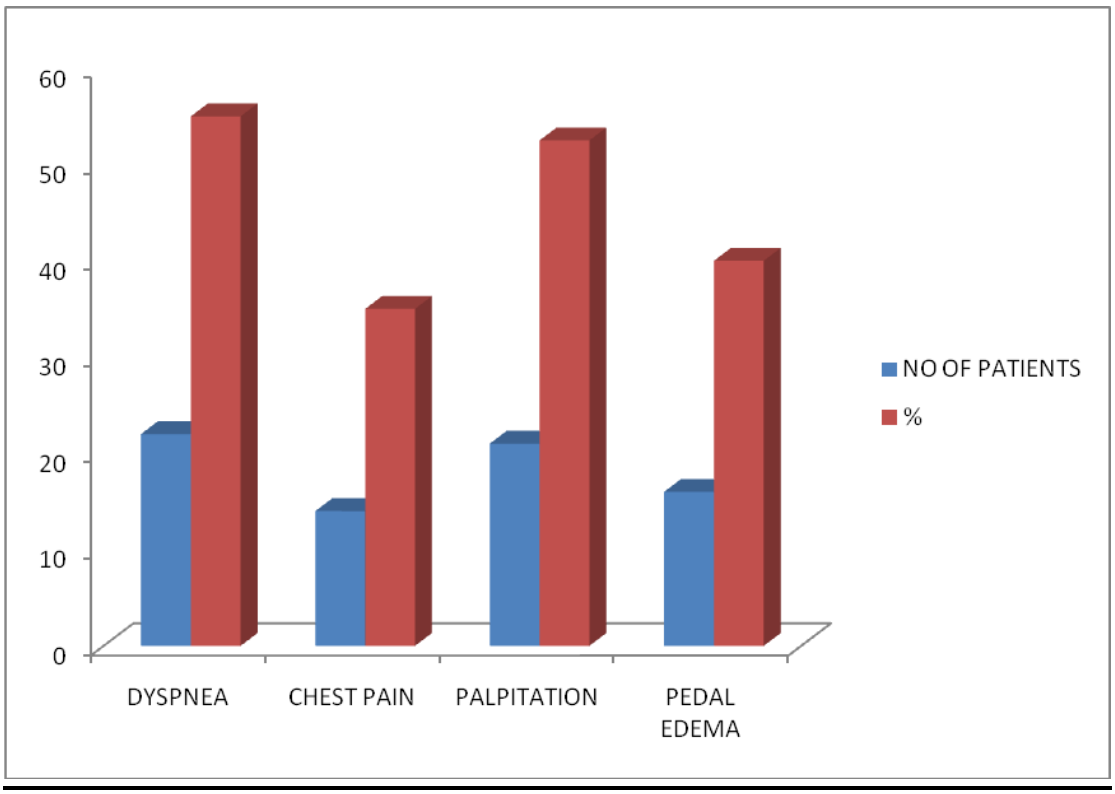
Age Distribution of patients with LVH

AGE GROUP(Years)	MALE	FEMALE
<40	3	3
40-49	5	6
50-59	5	6
60-69	5	2
70-79	3	2
Total	21	19



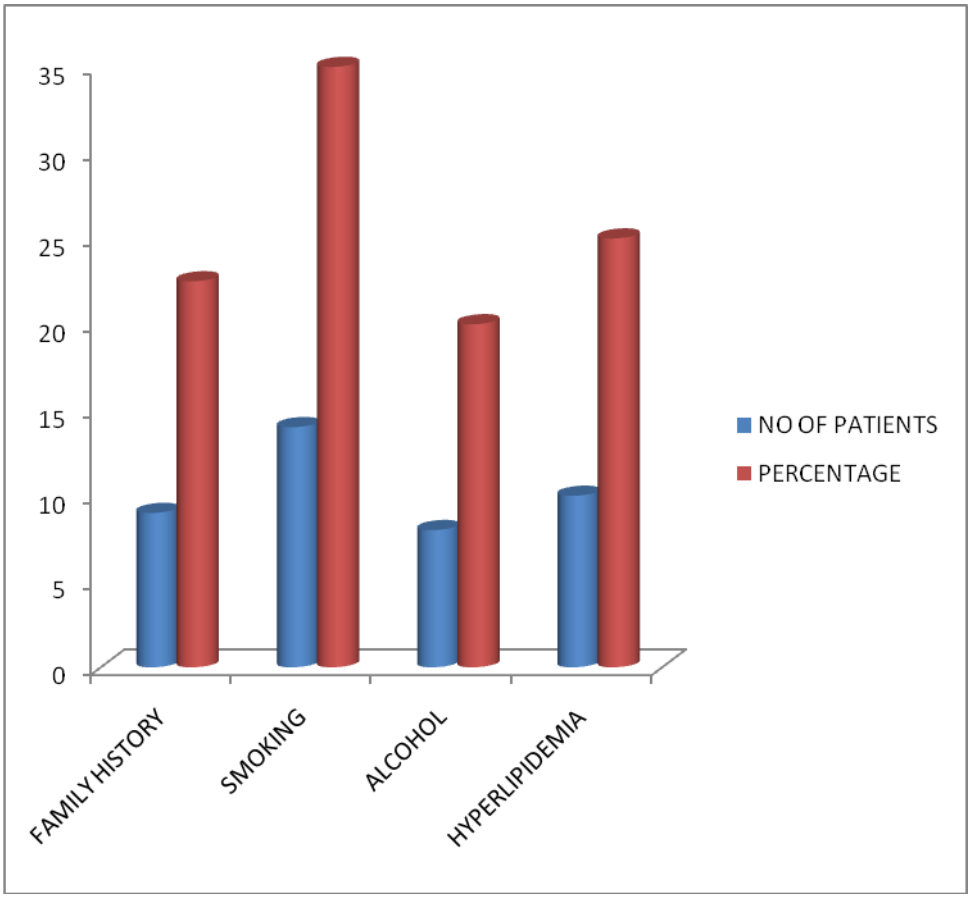
Symptoms in Patients with LVH(N=40)

SYMPTOMS	NO OF PATIENTS	%
DYSPNEA	22	55
CHEST PAIN	14	35
PALPITATION	21	52.5
PEDAL EDEMA	16	40



Risk factor in patients with LVH (N=40)

	NO OF PATIENTS	PERCENTAGE
FAMILY HISTORY	9	22.5
SMOKING	14	35
ALCOHOL	8	20
HYPERLIPIDEMIA	10	25

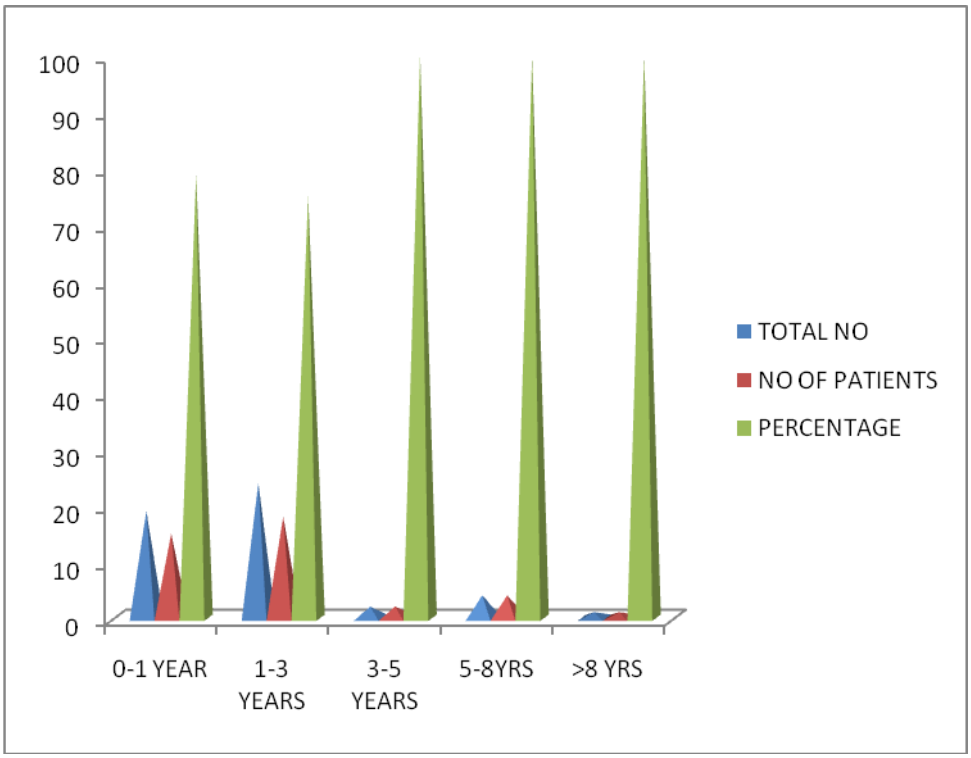


Validity of ECG in detection of ECG

	NO OF PATIENTS	SENSTIVITY	SPECIFICITY
RES	19	47.50%	100%
VOLTAGE CRITERIA	24	60%	100%

Correlation between duration of hypertension and prevalence of LVH

DURATION OF HYPERTENSION	TOTAL NO	NO OF PATIENTS	PERCENTAGE
0-1 YEAR	19	15	78.9
1-3 YEARS	24	18	75
3-5 YEARS	2	2	100
5-8YRS	4	4	100
>8 YRS	1	1	100



ECHO measurements and derived values

ECHO	**PATIENTS WITHOUT LVH** **Mean** ± SD	**PATIENTS WITH LVH** **Mean** ± SD	**"P" VALUE**
LVIDd	4.2±0.39	4±0.46	P<0.05
LVPWd	1.26±0.28	1.48±0.40	P<0.01
IVSd	1.17±0.30	1.48±0.26	P<0.05
LVM	182.8±43.1	320±98.1	P<0.001
LVMI	113.8±19.8	207.6±68.8	P<0.001

DISCUSSION

Males 29(58%), females 21(42%).

Male to female ratio1.38:1.

Prevalence of LVH in study population is 80%.

Anatomic validity of echocardiographic methods of determining left ventricular hypertrophy using the Penn and American society of echocardiography measurements was demonstrated in two independent correlation studies, using the sex-specific criteria, which showed high sensitivity of 97% and specificity of 96%[36].

In a previous study by Trivedi SK, Gupta OP, Jain AP.et al [48], LV mass index for Indian population was studied as 110g/m^2 in females and 121g/m^2 in males. These values were taken as reference values in this study and used as gold standard for left ventricular hypertrophy. The overall presence of left ventricular hypertrophy in hypertension as defined by sex-specific reference standard is reported to be 25 to 30%. With 97% specificity by Devereux RB et al [38]. Similarly Tingleff J et al [62]reported the prevalence of the left ventricular hypertrophy of 25-26% in hypertensives. Martinez et al [63]also reported LVH to be 26% in hypertension. The prevalence of LVH in the study population 80%. this can be explained by Left ventricular hypertrophy occurred in patients with sustained hypertension who also exhibited increased cardiac output due to delayed presentation, delay in treatment At similar levels of blood pressure, black patients were more likely than white patients to exhibit concentric left ventricular hypertrophy, especially among borderline hypertensive patients.

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	DEVEREUX ET AL	TINGELEFF ET AL	MARTINEZ ET AL	PRESENT STUDY
Presence of LVH in essential hypertension	20%	25-26%	32%	80%

Demographic features:

Sex:

	H.S CHIRMES et al	PRESENT STUDY
Males	14.9%	58%
Females	9.1%	21%

Slightly higher percentage (58%) of males than females (42%) had left ventricular hypertrophy in our study. In a study by Cohen et al[5], there was greater proportion of men than women with left ventricular hypertrophy. Also H.S Chirmes et al[64] reported greater proportion of left ventricular hypertrophy in men. However Hammond et al [38]reported a greater proportion in women, several factors may explain this, the upper normal limit by these criteria may be too high for men, the other reason may be hypertensive men with LVH who are known to be at risk of coronary artery disease probably are disproportionately excluded from this study.

Age:

In the present study the mean age of patients was 51.74 years

The mean ages for men and women were similar (56.0 ± 13.0 vs. 55.5 ± 13.5,) [75]

Mean age, 66.9 years (LIFE study) correlates with my study.

Hammond et al [38] also showed increased age is associated with LVH. There is no difference in the mean body surface area of the two groups.

Blood Pressure:

The duration of hypertension in patients with increased LVMI is more than patients with normal LVMI. The mean systolic and diastolic blood pressure was also more in these patients with increased LVMI. In a similar study Ross et al[65] also reported duration of hypertension as significant factor in the development of LVH. Glasser SP etal[66,] also showed duration of hypertension added significantly in predicting an elevated LVMI.

Clinical correlation also show that heaving apical impulse, loud first heart sound loud A_2 component of second heart sound and fourth heart sound are more often seen in patients with left ventricular hypertrophy. Ejection systolic murmur in aortic area may be due to sclerotic aortic valve. The sclerotic aortic valve may be due to the pro-atherogenic effect of hypertension. Similar findings have been reported in earlier studies[23].

Investigation:

Nkado RN et al [67]showed that Echocardiography is highly accurate for measurement of left ventricular mass compared to electrocardiography. In this study on comparing

the echocardiogram to chest x-ray and 12 lead ECG for detecting left ventricular hypertrophy, the echocardiogram is found to be more specific and accurate than the other two. The sensitivity being 65%, 67%, and 66% for chest x-ray, ECG and 2D-Echo respectively, and the specificity being 76%, 76% and88% for chest x-ray. ECG and 2D- Echo respectively.

In the standard 12 lead ECG on comparing the Sokolow-lyon criteria and Romhilt-Estes point score system, it is seen that sensitivity of 77% and specificity of 65% is found in Sokolov-lyon criteria whereas Romhilt-estes has sensitivity of 57% and specificity of 87%. Therefore Romhilt-Estes point score system becomes the ideal criteria for diagnosing left ventricular hypertrophy, if 2D- Echocardiography is not feasible. However, the electrocardiography and chest x-ray may convey other important information than echocardiogram; therefore echocardiogram should be used in conjunction with them, rather than instead of them[38].

Among patient with LVH breathlessness was found to be most common symptom (55%), followed by palpitation (52. %), pedal edema (40%), chest pain (35%).

Headache (36%), Chest pain (16%), Dyspnoea (11%), Palpitations (8), are the symptom of frequency in study done by Chowta K N, et al

Present study showed smoking (35%) is main risk factor followed by hyperlipidemia (25%), family history (22.5%) and alcohol (20%).all smokers are males. TOMH study shows prevalence of LVH in smokers as compared to nonsmokers.

Present study showed no significant relationship between staging of hypertension, and prevalence of LVH. Framingham study showed 30 yrs of average BP was a better predictor of LVM than a single recent blood pressure measurement.

Frank R Bauwens et al ,studied [36] untreated hypertensive patients a 24 hour ambulatory BP monitoring and determination of LV mass index according to Devereux formula.LV mass did not correlate with the office systolic or diastolic BP, but there was statistically correlation between 24 hr systolic BP and LV mass.

Cardiomegaly on x- ray chest was found in 20% it indicates only left ventricle dilatation, but not LVH.

ECG:

ECG failed to detect LVH both by RES and voltage criteria in 16 patients.

The sensitivity of ECG by RES is 47.5% and voltage criteria 60% both showed 100% specificity.

According to romhilt Estes the sensitivity of RE score was 54%[75.]

ECHOCARDIOGRAPHY

Echocardiography variables LVIDd, LVISd, IVSd, are increased in patients with LVH and more in case of LVM&LVMI which was statistically significant as compared to patients without LVH. Savage et al, study was in accordance with this study.

CONCLUSION

1. Mean age of patients 51.74%, male: female ratio=1.38.1

2. Prevalence of LVH is 80%.

More in male compared to female

3. Prevalence of LVH increased with age

4. Smoking is significant risk factor for LVH.

5. Retinopathy is more prevalent in patients with LVH as compared to patients without LVH.

6. The sensitivity of ECG –RES was 47.5% &voltage criteria 60%; specificity of both is 100%

7. Echocardiography is most sensitive in detection of LVH than ECG.

8. Echocardiography variables LVM and LVMI significantly increased in patients with LVH as compared to patients without LVH, which was statistically significant.

SUMMARY

- This study was done on 50 patients with hypertension.
- Detailed clinical evaluation, laboratory investigation ECG, chest x-ray, and echocardiography are done.
- Mean age of patients was 51.74%, male: female ratio=1.38.1.
- Prevalence of LVH is 80%.
- Patients with secondary hypertension, ischemic heart disease. Cardiomyopathies, valvular heart disease, congenital heart diseases and diabetes were excluded from the study.
- Mean duration of hypertension is more in patients with increased LVMI.
- Mean Systolic blood pressure and Diastolic blood pressures were also found to be more in patients with increased LVMI.
- Comparative study of LVH, showed high specificity and accuracy with echocardiography when compared to ECG.
- End organ damage like Retinopathy is more in patients with increased LVMI.

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KEY TO MASTER CHART

BSA : body surface area

CP : chest pain

PALP : palpitation

PD EDEMA: pedal edema

D.HTN: duration of hypertension

F/H: family history

BP: blood pressure

RETINO: retinopathy

M: Male

F: Female

+ : Present

_ : Absent

X-CARD : Chest xray

RES: Romhilt –estes score

VOLTAGE : voltage criteria

LVIDd : Left ventricle internal dimension diastole

IVSD: Inter ventricle septal diastole

LVPWd: Left ventricle posterior wall diastole

LVM : Left ventricle mass

RWT : Regional wall thickness

LVMI : Left ventricle mass index

MASTER CHART

Sl.NO	IP NO	AGE	SEX	BSA	DYSPNEA	CP	PALP	PD EDEMA	D.HTN	F/H	SMOKING	ALCOHOL	DYSLIPDEMIA	PULSE	BP	S3/S4	RETINO	X-CARD	RES	VOLTAGE	LVIDd	IVSD	LVPWd	LVM	RWT	LVMI
1	552157	60	M	1.6	-	-	-	-	7	-	+	+	-	88	160/90	-	2	-	0	0	4.1	1.4	2	220	58	134
2	563110	35	F	1.6	-	-	-	+	ND	-	-	-	-	72	150/100	-	-	-	6	+	4.3	1.2	1.3	223	60	139
3	561593	50	F	1.5	+	+	+	-	2	+	-	-	+	86	180/94	-	-	-	0	0	4.2	1.2	1.4	228	66	152
4	506713	35	F	1.8	-	_	_	-	ND	-	-	-	-	86	170/100	-	1	+	0	0	4.6	1.8	1.5	374	65	203
5	523101	45	M	1.8	+	+	-	-	1	-	+	+	-	68	160/90	-	-	-	9	+	4.8	2.3	1.8	560	75	325
6	561038	35	M	1.3	+	-	-	+	ND	-	-	-	-	80	180/90	-	1	-	6	+	4.5	1.6	1.6	345	71	262
7	557100	50	M	1.7	-	-	-	-	2	-	+	+	-	80	190?120	-	2	-	0	0	3.9	1.4	1.2	204	60	118
8	550831	66	F	1.5	-	-	-	-	ND	-	-	-	-	78	190/100	-	-	-	5	+	3.9	1.5	1.3	231	67	154
9	458693	43	F	1.4	+	-	+	+	1	-	-	-	-	90	170/90	-	-	+	6	+	4.9	1.2	1.2	258	50	187
10	560173	53	F	1.6	+	-	+	_	1	-	-	-	+	88	170/90	-	-	-	5	+	3.6	1.7	1.2	215	60	134
11	561252	50	M	1.5	-	+	_	+	ND	_	+	-	-	80	190/120	-	2	+	0	0	4.7	1	1.9	318	80	212
12	558843	55	M	1.6	+	-	-	-	2	+	-	-	-	96	140/90	-	2	-	0	0	3.6	1.5	1.2	202	50	126
13	551093	45	F	1.4	+	+	-	_	1	_	-	-	-	100	180/100	-	-	-	0	0	4.7	1	1.2	268	51	186
14	551094	50	F	1.6	-	-	-	+	1	+	-	-	+	120	200/100	-	-	-	0	0	3.2	1.2	1.4	223	80	138
15	535048	50	M	1.5	+	+	-	-	2	-	+	-	-	80	180/96	-	3	+	9	+	5	1.5	1.3	331	52	217
16	553224	60	M	1.3	-	+	-	-	1-	-	_	-	+-	92	150/100	-	-	-	0	0	3.7	0.9	0.6	86	32	66
17	563937	40	F	1.6	+	_	+	_	4	_	_	_	_	78	150/100	-	-	-	0	0	4.1	1.6	1.1	256	53	160
18	564930	72	M	1.5	-	-	+	+	ND	+	-	-	-	110	160/120	-	-	-	0	+	4.5	1.3	1.5	251	60	169
19	554467	63	M	1.6	+	+	-	-	1	-	+	-	+	84	170/100	-	2	+	6	+	4.5	1.4	1.7	299	75	181
20	552692	55	M	1.4	-	-	-	-	2	+	_	_	_	96	220/100	-	3	-	0	0	4.5	0.9	1.1	175	40	131
21	474254	35	M	1.6	-	-	+	-	1	_	+	_	_	100	160/110	-	2	+	8	+	5.7	2.03	1.95	688	60	424
22	563792	40	F	1.6	_	-	-	-	ND	+	-	_	_	80	150/96	-	1	_	0	0	3.9	1.4	1.2	204	60	113
23	555121	45	M	1.6	-	-	-	+	2	-	-	-	-	88	170/100	-	-	-	0	0	3.6	1.7	1.2	215	60	134
24	551615	40	F	1.4	+	-	+	-	ND	+	_	_	+	88	190/120	-	_	_	0	0	4.5	1.6	1.1	207	48	186
25	506698	68	M	1.6	--	-	-	-	1	-	-	-	-	78	170/90	-	-	-	0	0	4.2	1.2	1.3	194	60	119
26	438075	60	M	1.6	-	-	-	-	2	-	-	-	-													
27	574353	55	M	1.6	+	-	+	+	ND	+	+	_	_	80	170/90	-	3	-	6	+	4.7	1.8	1.5	318	60	198
28	494387	60	F	1.3	-	-	-	-	ND	-	-	-	+	90	150/90	-	-	-	0	0	3.5	1.2	1	112	57	85
29	572152	51	F	1.6	+	+	+	-	3	_	_	_	_	96	160/90	-	3	+	9	+	5.1	1.5	1.6	397	60	248
30	568830	50	F	1.5	_	_	_	+	ND	_	_	_	_	100	190/96	_	1	_	0	0	4.1	1.8	1.3	287	65	186
31	520652	70	F	1.6	+	-	+	_	6	_	_	_	+	104	150/100	_	3	_	6	+	4	1.8	1.3	277	65	172
32	568570	50	F	1.5	_	+	_	+	1	_	_	_	_	80	150/90	_	2	-	6	+	5	1.6	1	298	40	199
33	565217	57	M	1.6	+-	_	+	-	2	+	+	_	_	110	190/102	_	3	_	3	+	4.8	1.6	1.4	306	58	196
34	577606	70	M	1.5	-	-	-	-	2	_	_	+	_	86	150/90	_	_	_	0	0	4.2	1.2	0.8	139	38	91

Sl.NO	IP NO	AGE	SEX	BSA	DYSPNEA	CP	PALP	PD EDEMA	D.HTN	F/H	SMOKING	ALCOHOL	DYSLIPDEMIA	PULSE	BP	S3/S4	RETINO	X-CARD	RES	VOLTAGE	LVIDd	IVSD	LVPWd	LVM	RWT	LVMI
35	575978	47	F	1.5	_	_	+	_	3	_	_	_	_	88	160/120	_	4	_	3	+	3.5	1.8	1.7	283	90	191
36	575976	60	F	1.4	+	+	+	+	10	+	_	_	_	100	160/110	_	2	_	5	+	5.2	1.8	1.8	504	60	371
37	567464	40	M	1.7	+	_	_	_	ND	_	+	_	_	80	150/154	-	2	_	3	0	4.9	1.9	1.3	229	53	132
38	561252	68	M	1.6	-	+	+	_	2	_	+	+	+	90	160/100	_	_	_	3	0	4.3	1.4	1.1	223	65	137
39	575934	35	M	1.5	-	-	-	-	3M	_	_	+	_	76	140/90	_	_	_	0	0	4.1	1	1.3	172	50	113
40	572313	38	M	1.6	-	_	+	_	ND	_	_	_	+	80	176/90	_	_	-	0	0	4.2	1.2	1.4	228	60	140
41	576686	45	F	1.2	+	_	_	+	ND	_	_	_	+	96	150/90	S3	1	+	3	+	4.3	1.8	2.1	445	97	364
42	571654	50	M	1.5	+	_	+	_	1	+	+	_	_	80	160/94	_	2	_	0	0	4.2	1.2	1.5	241	70	158
43	571634	70	M	1.6	_	+	_	+	4	_	_	_	+	94	170/100	_	3	+	7	+	5.1	2.4	1.7	560	60	345
44	570086	45	M	1.6	+	_	+	_	IM	_	_	+	_	110	180/106	-	2	_	5	+	4.5	1.4	1.7	329	75	203
45	572422	68	M	1.6	_	_	_	_	ND	_	_	_	_	80	160/94	_	3	-	0	0	4	1.5	1	200	50	123
46	575520	70	F	1.7	_	+	-	_	8	+	_	+	+	88	170/90	__	_	-	5	+	4	1.7	1.8	366	85	215
47	571395	65	M	1.5	+	_	+	+	6	_	+	+	_	96	160?100	_	2	_	6	+	4.8	1.4	1.1	269	45	176
48	57003	42	M	1.9	+	_	_	_	ND	_	+	_	_	110	146/96	-	3	-	6	+	4	1.7	1.5	292	75	157
49	567988	35	F	1.6	-	+	+	+	2M	_	-	_	_	84	140/96	_	4	_	3	+	4.3	1.5	1.6	274	74	192
50	552157	46	M	1.6	+	_	+	_	1	+	+	_	_	96	160/100	_	_	+	0	0	4.1	1.36	1.4	240	68	135