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Left Ventricular Mass Index As Reference For Left Ventricular Hypertrophy

Dr. Anil Bhattad1 Assistant Prof Dr. Jabbar Desai 2 Assistant Prof. Dr. Rohit Gosavi3 Assistant Prof.,Dr. Abhijeet Patil4 Assistant Prof .

Department of Medicine, Krishna Institute of Medical Sciences,
Krishna Institute of Medical Sciences "Deemed to be University", Karad
Email:-anilbhattad75@gmail.com

ABSTRACT

The risk of cardiovascular morbidity and mortality is observed to be two to three times in cases with ventricular hypertrophy as compared to cases with normal left Ventricular mass.3 Development of hypertension is dependant on variables like country of origin, race, population of different regions along with different environmental and genetic factors. All these influence the development of hypertrophy and its effect on mortality and morbidity. Pathological hypertrophy may be present for many years without any symptoms and then development of sudden severe symptoms.

Multiple diagnostic methods can be used to detect Left Ventricular hypertrophy (LVH). The Electrocardiogram (ECG) is the most common, and has many criteria for detection of LVH, but its sensitivity is less. Echocardiography is among the most sensitive, specific, non-invasive and repeatable investigations for LVH as compared to the Electrocardiogram.

INTRODUCTION

"When the heart faces increased hemodynamic burden, it compensates by different mechanisms, one being increased cardiac muscle which is commonly seen in response to hypertension. It is the first step towards development of clinical disease due to abnormal increase in the mass of the left ventricle owing to maladaptation resulting from chronic pressure overload to heart"[1]. "The prevalence of hypertrophy increases with duration of hypertension. Left Ventricular Hypertrophy is an independent indicator of morbidity and mortality, which can pre-disposed to cardiac failure, stroke, ventricular arrhythmias, etc. It is a major risk factor in patients of Hypertension as it can cause sudden death".[1,2]

The Left ventricular mass is assessed by two-dimensional transthoracic echocardiography and M-mode measurements to measure the Left Ventricular wall dimensions at the end of diastole.

Objectives:

(i) To study the Left ventricular hypertrophy (LVH) in patients diagnosed with hypertension with reference to left ventricular mass index and its correlation with the severity of the disease.

(ii) To correlate electrocardiography and two-dimensional echocardiography findings for detecting Left ventricular hypertrophy.

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MATERIAL AND METHODS

This study was conducted on 73 patients who were admitted for Hypertension in the inpatient medicine ward of Krishna Institute of Medical Sciences, Karad during the study period from November 2017 to May 2019.

Study Design: Cross-sectional (Observational) study

Study Framework:

Study Centre: Inpatient medicine ward of Krishna Hospital and Medical Research Centre, Karad.

Time Frame: Data was collected from November 2017 to May 2019.

Sample Size Determination:

According to previous studies ,the prevalence of Left ventricular hypertrophy (LVH) varies between 23 and 48% in clinical populations. Hence, we chose p= 25%, q= 1-p i.e. 75%. Using the formula for cross-sectional studies, with the absolute precision of 10 percentage points (d) at a 95% confidence interval, and p=25%, the sample size comes up to 73 patients.

Formula: $N=4pq/d^2$

Sample recruitment:

Simple Random sampling was done to recruit 73 patients who fit the inclusioncriteria.

Inclusion and Exclusion Criteria:

1. Inclusion criteria:

Both male and female patients who fulfilled the following criteria were includedin the study:

- Patient's age more than or equal to 18 years
- Diagnosed cases of Hypertension

2. Exclusion criteria:

Patients with any of the following criteria were excluded from the study:

- Patient's age less than 18 years
- Patients with Hypertrophic Obstructive Cardiomyopathy, valvular heart disease, congenital heart disease, other cardiomyopathies.
- Patients who are known case of any other cardiac diseases
- Patients who are newly detected case of any other cardiacdiseases

Study tool:

A pre-tested validated proforma was developed to collect data for theresearch purpose.

Procedure:

After obtaining the Ethical clearance, the study was initiated. Data was collected after taking informed and written consent of patients. A detailed casehistory of the patient was taken and a detailed clinical examination was done. This included demographic details of the patient, chief complaints, past medical history, and relevant family history, personal history about diet, appetite, sleep, bowel/bladder habits, addictions were taken.

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All the patients underwent a basic workup according to needs of the patient and standard 12 lead Electrocardiogram (ECG) was recorded along with two-dimensional trans-thoracic echocardiography. All the patients diagnosed with Hypertension were divided according to Grades of hypertension, and were further treated and managed according to standard treatment guidelines.

Left Ventricular mass (LVM) was calculated by using the LV dimensions using the Devereaux (cube) formula, according to the current American Society of Echocardiography (ASE) guidelines,

$$\text{“LVM= 0.8x 1.04 [} (IVS_d + LVID_d + PWT_d)^3 - (LVID_d)^3 \text{] + 0.6g”}$$

[Note: **$LVID_d$**: “left ventricular internal dimension”, **LVMI**: “left ventricular mass index”, **lVS_d**: “inter-ventricular septal thickness”, **PWT_d**: “posterior wall thickness”]

Patients were considered to have Left ventricular hypertrophy if Left ventricular mass index (LVMI) was >95 g/m^2 in females and .115 g/m^2 in males.

LV mass was indexed using body surface area (BSA). It was calculated usingformula,

$$LVMI = LVM/BSA\ (g/m^2)$$

Data Entry and Analysis:

Data entry was done in Microsoft Excel 2016 and analyzed using SPSSversion 21.0 (IBM). Descriptive statistics like mean, standard deviation, range, and proportions were used. The results were represented in tabular and graphical formats. The use of inferential statistics was limited. “Chi-square statistics were used to see associations between categorical variables. An unpaired t-test was used for comparing means of continuous variables. Pearson’s correlation coefficient was used to see the association between twocontinuous variables. The outcome of interest was calculated within 95% confidence limits. P-value of <0.05 was considered to be significant.”

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Figure 1: Comparison of latest ACC/AHA and ESC/ESH Hypertension Guidelines [Source: Chopra et al[5]]

Parameter	ACC/AHA	ESC/ESH
Definition of hypertension, mm Hg	>130/80	>140/90
Grading of normal pressure, mm Hg	Normal <120/80	Optimal <120/80
	Elevated 120–129/<80	Normal 120–129/80–84
		High normal 130–139/85–89
Grading of hypertension, mm Hg	Grade 1, 130–139/80–89	Grade 1 140–159/90–99
	Grade 2, ≥140/90	Grade 2, 160–179/100–109
		Grade 3, ≥180/110
Target blood pressure in various subsets	≤65 y, <130/80	<65 y, <130/80
	≥65 y, <130/80	≥65 y, <140/80

According to **Richard E. Katholi** and **Daniel M. Couri**, factors promoting left ventricular hypertrophy are,

a. Hypertension

b. Neurohumoral factors[6] - Angiotensin II, Aldosterone, Norepinephrine, Insulin, and other growth factors[7]

c. Genetic influences[8]

"Metabolic syndrome is associated with an increased Left ventricular mass (LVM)".[9] Therefore "it is important to screen obese hypertensive's for Left ventricular hypertrophy (LVH). Left ventricular hypertrophy (LVH) should also be suspected in patients with hypertension who have unilateral renal artery stenosis".[10] "Of the several adverse changes in cardiovascular morphology and function that occur in association with hypertension, left ventricular hypertrophy (LVH) is an established and independent prognostic factor for major cardiovascular events, including sudden cardiac death, acute myocardial infarction, stroke, and congestive heart failure".[11]

In individuals with left ventricular hypertrophy, the risk of cardiovascular morbidity and death is two to four times higher than in people with normal left ventricular mass, highlighting the importance of detecting this condition. Stroke, heart attack, and kidney failure are all reduced when hypertension is treated well over the long term. It is also crucial to detect left ventricular hypertrophy (LVH) and treat it in order to encourage regression.

Types of Left ventricular Hypertrophy:

- "Concentric left ventricular hypertrophy" (LVH): "increased left ventricular mass index (LVMI) with relative wall thickness ≥0.45"
- "Eccentric left ventricular hypertrophy"(LVH): "increased Left ventricular Mass Index with a relative wall thickness <0.45"

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Pathophysiology of Ventricular Hypertrophy:

An increase in the muscle mass to compensate for hemodynamic overload is a key mechanism used by the heart to compensate. The increase in muscle mass is due to hypertrophy rather than hyperplasia as the myocytes become terminally differentiated cells just after birth. Therefore, in case of pressure overload like hypertension, there is an increase in myocyte width due to the addition of sarcomeres. This type of remodeling which causes increased wall thickness leads to concentric hypertrophy.

Consequences of Left ventricular hypertrophy(LVH):

"The association of Left ventricular hypertrophy (LVH) with Hypertension and cardiovascular consequences due to it is well established. Left ventricular hypertrophy (LVH) can cause atrial fibrillation, heart failure, and sudden death. Echocardiography findings in hypertensive patients have revealed a concentric LVH very commonly. Left ventricular hypertrophy(LVH) involves changes in myocardial tissue architecture consisting of perivascular and myocardial fibrosis and medial thickening of intramyocardial coronary arteries, in addition to myocyte hypertrophy. The physiologic alterations which occur as a result of these anatomical changes include disturbances of myocardial blood flow, the development of an arrhythmogenic myocardial substrate and diastolic dysfunction."[12]

According to **William B. Kannell**, prevalence of Left ventricular hypertrophy (LVH) on electrocardiogram (ECG) increases with age and has male preponderance; cardiac enlargement on X-rays is twice as compared to LVH findings on ECG; in cases with systolic pressure >160 mm Hg presence of LVH on ECG was seen in 50%; the risk of cardiovascular morbidity and mortality was two to three times as compared to cases with normal LV mass.[13]

In the case of pathological hypertrophy, "there may be no symptoms for many years in most of the patients and they may develop congestive heart failure or unexpected sudden death. It is necessary to diagnose LVH. The diagnosis of LVH depends mostly on imaging techniques like echocardiography measurements".[14]

Clinical assessment of a hypertensive patient may not always tell us about the presence or absence of left ventricular hypertrophy. There two main methods that are used in the detection of hypertrophy. They are commonly employed, easily available and non-invasive; they are the electrocardiogram and echocardiography.

In the 1786, Dr. Luigi Galvani, an Italian physician and physicist at the University of Bologna, first noted that "electrical current could be recorded from skeletal muscles. He recorded electrical activity from dissected muscles. In 1842, Dr. Carlo Matteucci, a professor of physics at the University of Pisa, demonstrated that electrical current accompanies every heart beat in a frog. Thirty-five years later, Augustus Waller, a British physiologist of St Mary's Medical School in London, published the first human electrocardiogram. It was recorded by Waller in 1887 with Lippmann's capillary electrometer, which revealed only 2 deflections. Being a physiologist, Waller labeled the waves, as one would expect a physiologist to do, he used letters that suggested the anatomic parts of the heart that produced them. Accordingly, he labeled the 2 waves V1 and V2 to indicate ventricular events".[15]

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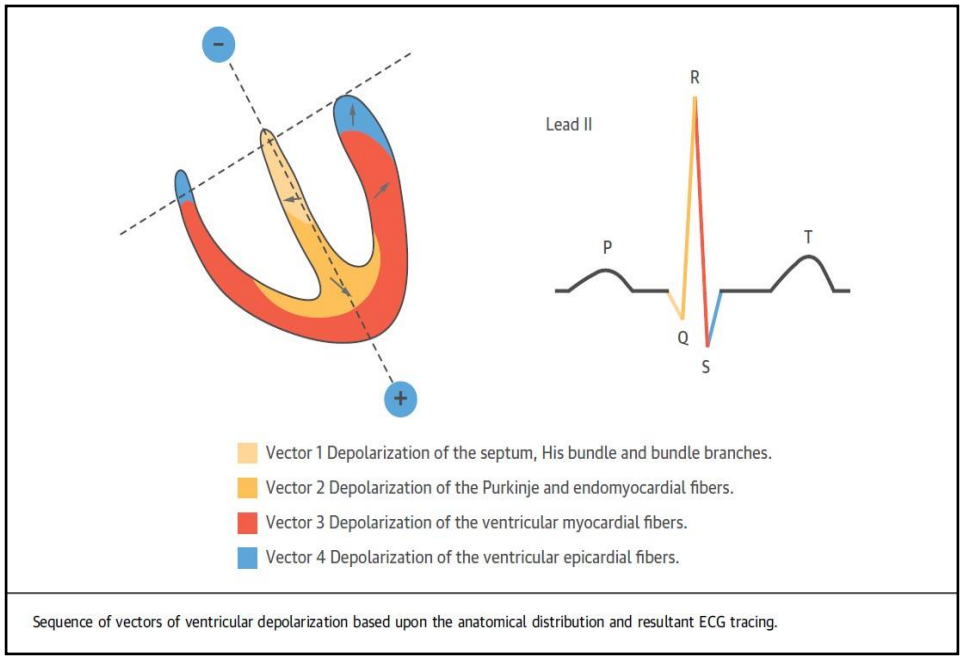
Dr. Willem Einthoven, a Dutch physiologist inspired by the work of Waller, "refined the capillary electrometer even further and was able to demonstrate five deflections which he named ABCDE. To adjust for inertia in the capillary system, he implemented a mathematical correction, which resulted in the curves that we see today. He used the terminal part of alphabet series (PQRST) to name these deflections."

LVH can be estimated by the electrical voltage changes detected on the surface electrocardiogram. "This principle makes the electrocardiogram an acceptable surrogate to detect changes in left ventricular mass. However, the cardiac electrical voltage does not exclusively depend on the amount of myocardium. Rather, it is dependent on active and passive electrical properties of the heart and torso. These in turn are modified by influencing factors such as distance of left ventricular cavity to electrode, the location of the surface electrode, individual anthropometric differences, conduction abnormalities, fibrosis of the myocardium, and lung pathology. In addition, it has been described that the ECG voltage may vary significantly from day to day, between patients, or even within the same patient. All these factors hamper in reproducibility of ECG findings and difficulty in quantification."

Figure 2: Mean vectors of ventricular depolarization [Source : Peguero et al[16]]



Sequence of vectors of ventricular depolarization based upon the anatomical distribution and resultant ECG tracing.

"Left ventricular Hypertrophy is not only the organ manifestation of hypertrophic growth of the cardiac myocytes but also of changes in the interstitium due to fibrosis and deposition of other material that may dampen the voltage expression of the hypertrophic myocardium and limit the diagnostic capability of the surface electrocardiogram. This inherent limitation of the electrocardiogram is an important contributor to the high false-negative rate that all ECG criteria share. Nonetheless, the electrocardiogram continues to be an important low-cost tool for early screening and detection of leftventricular hypertrophy".[17]

At present, 37 different ECG criteria have been endorsed by the American Heart Association.

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The most commonly used criteria for LVH on ECG are the following:-

Sokolow-Lyon Index: [17]

This was introduced in 1949.This is the most commonly used criteria for thediagnosis of LVH.

- "R in V5 or V6 +S in V1>35 mm"
- "R in aVL >11 mm"

Romhilt and Estes point scoring system :[18]

Developed in 1968.

- 3 points each
  - "P wave from left atrial abnormality"
  - "Any increase in voltage of the QRS complex"
  - "R or S in limb lead ≥20 mm"
  - "S in V1 or V2 ≥30 mm"
  - "R in V5 or V6 ≥30 mm"
  - "ST-T abnormalities"
  - "Any shift in the ST segment (without digitalis) =3"

- 2 points
  - "Left axis deviation of ≥30°"
- 1 point each
  - "Slight widening of the QRS complex of ≥0.09 seconds"
  - "Intrinsicoid deflection in V5 or V6 of ≥0.05 seconds"
  - "ST-T abnormalities with digitalis"

Score of ≥5 points LVH; score of 4 points probable LVH

Cornell voltage criteria

"R in aVL + S in V3 >28 mm in men and >20 mm in women."

Cornell product:

Cornell voltage multiplied by the QRS duration in milliseconds>2,440 milliseconds. (In women, 6 mm is added to Cornell voltage.)

Total QRS voltage:

Total QRS voltage or total amplitude of the QRS complex obtained from all 12 leads. The normal voltage averages 129 mm (range, 80 to 185 mm) with 175 mm as the upperlimits of normal. [19]

An electrocardiogram is less sensitive than the echocardiography in diagnosing Left ventricular hypertrophy (LVH).

Review of Literature

In the present study the 2D echocardiography guided M-mode linear measurements to measure Left Ventricular dimensions at the end of diastole were used. 2D echocardiography is the visual compilation of the sound waves reflected back into the machine from the transducer to give a 2D image of the heart structures. "By positioning the transducer over different acoustic "windows" of the chest wall, single-dimensional images of cardiac structures could be recorded and inferences about structure, dimensions, and function could

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be made, now referred to as two-dimensional echocardiography." By definition, "the M-mode presentation depicts anatomy along a single dimension corresponding to the ultrasound beam creating what has been called the 'ice- pick' view of the heart. Amplitude of any object along the path of the ultrasound beam can be converted to brightness (B-mode), in which the strength of the echoes at various depths is depicted as relative brightness. Motion can be introduced by plotting the B-mode display against time. This is the basis of M-mode echocardiography."

Levy D et al conducted a study on the elderly population to see the association of LVM by echocardiograph with the incidence of coronary heart disease. They found that the risk for new coronary events with echocardiographic LVH was 1.67 times in men and 1.60 times in women per 50 grams increase in LVM. "Compared to electrocardiographic LVH, Echocardiographic LVH was 15.3 times more sensitive in predicting coronary events in men and 4.3 times more sensitive in women."[20]

Reichek N and Devereux RB studied the anatomic, ECHO and ECG findingsof LVH in 34 patients. They found good correlation between Echocardiography LV mass (LVM) and postmortem LV weight (r=0.96); sensitivity was 93% and specificity was 95%; sensitivity of Romhilt-Estes point score and Sokolow-Lyonvoltage criteria for ECG LVH were 50% and 21% respectively; specificity ofRE point score and SL criteria were 95%. They concluded that " ECG is specific but less sensitive criteria for diagnosing left Ventricular Hypertrophy (LVH) and M-mode echocardiography LV Mass is superior to ECG criteria for the diagnosis of Left Ventricular hypertrophy (LVH)".[21]

Casale P et al did a study in 140 men to assess whether detection of LVH by Echocardiography and ECG could predict cardiovascular events in patients with uncomplicated essential hypertension. After 4.8 years, they found that cardiovascular morbidity occurred more in patients with LVH on echocardiography than with normal ventricular mass ($p < 0.01$). LVMI had the highest independent relative risk for future cardiovascular events. The predictive value of ECG was low.[22]

Due to higher costs, even though Echocardiography is a more sensitive toolfor detecting LVH it is not being used in the routine evaluation of asymptomatic patients with uncomplicated hypertension.

Devereux, R B et al conducted a study to compare necropsy findings in 55 patients to determine the accuracy of echocardiographic assessment of LVH. They found that, LV mass from Penn- Cube formulae strongly correlated with necropsy LV mass ($r = 0.92$, $p < 0.001$). The overestimation was 6%. LV mass from ASE-cube formulae also stronglycorrelated with necropsy LV mass ($r = 0.90$, $p < 0.001$) and overestimation was 25%.

Correction formula was given as,
LV mass = 0.80 (ASE-cube LV mass) + 0.6 g.
LV mass and cross-sectional area are useful measures to detect LVH accurately.

There are standard methods for 2D M-mode echocardiographic measurements of LV dimensions and the calculation of LV mass.[14] Many studies have mentioned criteria for

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detecting LVH based on echocardiography. The criteria for LVM offered by **Levy D et al** based on the original cohort of Framingham Heart Study has the largest number of subjects n=6148.According to them, the prevalence of LVH in the Framingham Study population is 19% in men and 16% in women.

The formula for used for LV Mass was, $LVM=0.8x[1.04x(LVID+LVPWT+IVST)^3- LVID^3]$
Factors affecting LVM are: [23]

1. Body size: obese people have increased LVM
2. Gender: males have larger LVM than females
3. Physical activity: athletes have physiological increased LVM
4. Ethnicity: black men have larger LVM than whites or Asians
5. Age
6. Blood pressure

Body size, gender, ethnicity, and physical activity are also associated withincreased LV volume.[24]
LV "relative wall thickness" (RWT): "the ratio of twice the LV inferolateral wall thickness to the LV internal diameter measured at end-diastole".

a. Two-dimensional method of LV measurements[24]

- Truncated ellipsoid formula

$$\text{LV mass} = 1.05\pi\left\{(b+t)^2\right.$$

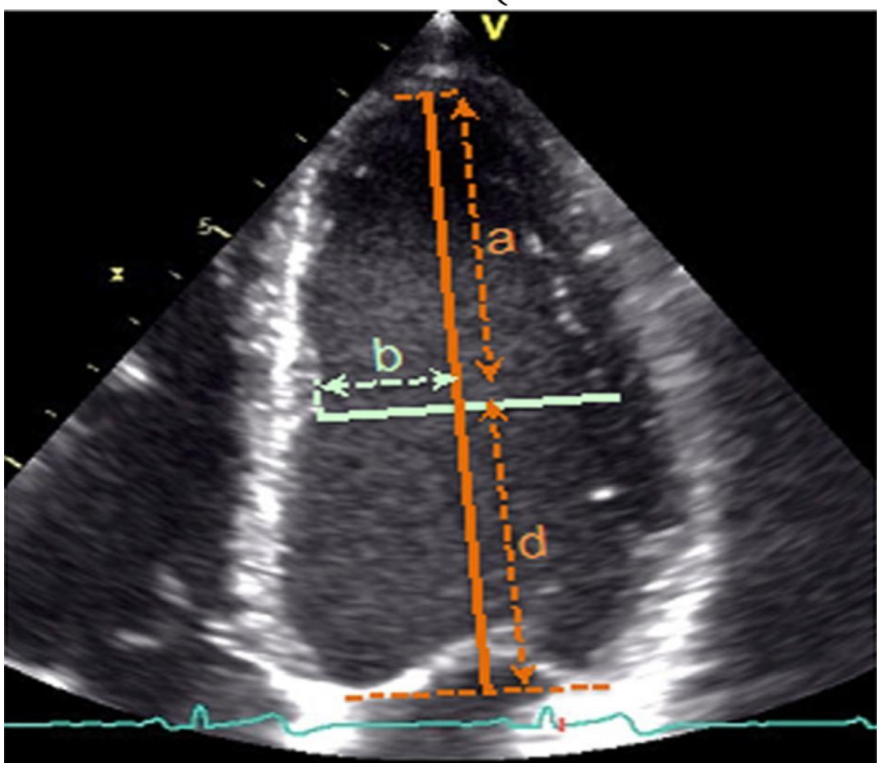

Figure 3: Measurement of LV dimensions in 2-D Echocardiography for truncated ellipsoid formula [Source: Lang et al [24]]

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- Area length method

$$\text{LV mass} = 1.05 \left\{ \left[\frac{5}{6} A_1 (a + d + t) \right] - \left[\frac{5}{6} A_2 (a + d) \right] \right\}$$

Where,
a – "distance from the minor axis to the endocardium at the LV apexb - LV minor radius"
d – "distance from the minor axis to the mitral valve planet - mean wall thickness".
A1= "epicardial cross-sectional areas A2= endocardial cross-sectional areas"
"Mean wall thickness is calculated from A1 and A2 in a short-axis view at the level of papillary muscle (top panel, green line, figure 1.7). Here the papillary muscles are considered part of the LV cavity."

"The short axis radius is calculated as: $b\sqrt{\frac{A_2}{\pi}}$"

"Mean wall thickness t is calculated as: $t = \left(\sqrt{\frac{A_1}{\pi}}\right) - b$"

"The cross-sectional area of the myocardium (Am) is: Am = A1 - A2"

Observations and Results

This cross-sectional study was conducted on 73 patients who were admitted for Hypertension and it yielded the following results.

Demographic Statistics

This a table of the age distribution of patients in our study group, ranging from 23 to 85 years of age. "The maximum number of patients i.e. 30.1% were in the age group of 61-70 years followed by 23.3% in the age group of 51-60 years, followed by 20.5% in the age group of 71-80 years. The mean age of patients was 61.89 years (SD ± 13.66, Range = 23-87 years). Of all the patients 2.7% patients were in the age group of 20-30 years, 5.5% in age groups of 31-40 years, 5.5% in age group of 81-90 years and 12.3% in 41-50 years."

Table 1: Age distribution of patients with Hypertension (n=73)

Age categories	Number	Percent
20 to 30 years	2	2.7
31 to 40 years	4	5.5
41 to 50 years	9	12.3
51 to 60 years	17	23.3
61 to 70 years	22	30.1
71 to 80 years	15	20.5
81 to 90 years	4	5.5
Total	73	100

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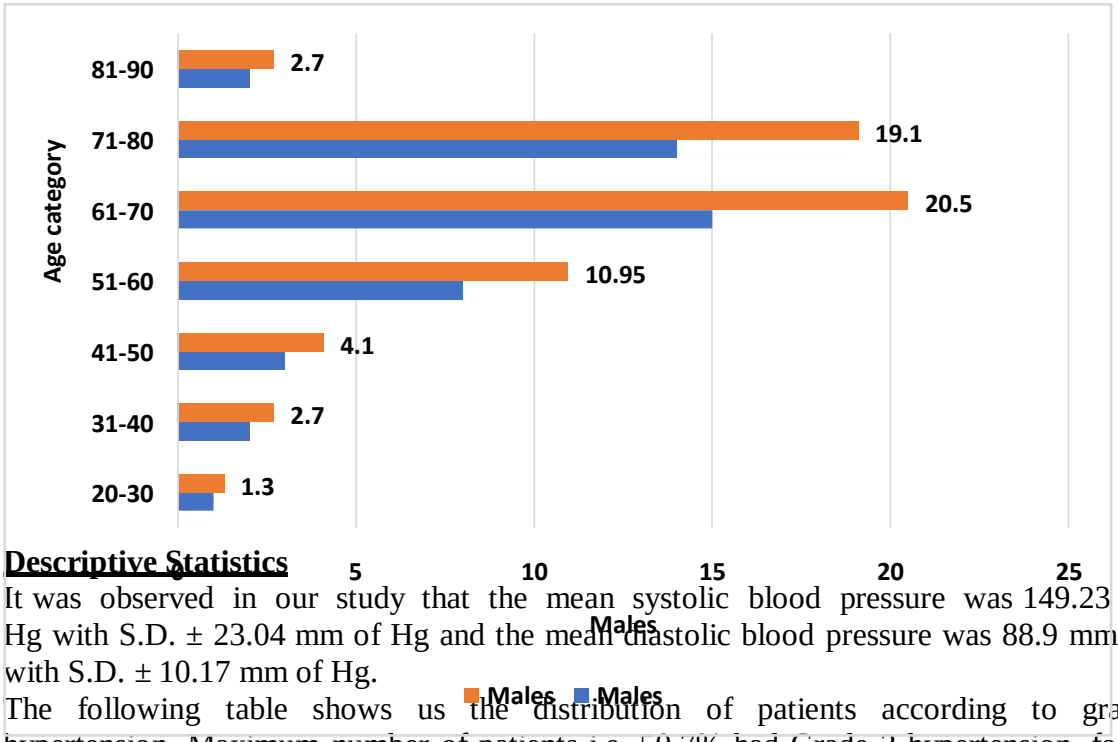
Table 2: Sex distribution of patients with Hypertension (n=73)

Sex	Number	Percent
(i) Male	45	61.6
(ii) Female	28	38.4
Total-	73	100

Figure 4: Bar graph showing age and sex distribution of male patients(n=73)



Descriptive Statistics

It was observed in our study that the mean systolic blood pressure was 149.23 mm of Hg with S.D. ± 23.04 mm of Hg and the mean diastolic blood pressure was 88.9 mm of Hg with S.D. ± 10.17 mm of Hg.

The following table shows us the distribution of patients according to grades of hypertension. Maximum number of patients i.e. 50.7% had Grade 2 hypertension, followed by 31.5% that had Grade 1 hypertension, 9.6% had elevated blood pressures and 8.2% had normal blood pressure.

Table 3: Grading of Hypertension (n=73)

Grading of Hypertension	Number	Percent
Normal (less than 120/80 mmHg)	6	8.2
"Elevated (systolic BP 120-129 mmHg and diastolic BP <80 mmHg)"	7	9.6
"Stage 1 (systolic BP 130-139 mmHg ordiastolic BP 80-89 mmHg)"	23	31.5
Stage 2 (systolic at least 140 mmHg ordiastolic at least 90 mmHg)	37	50.7
Total	73	100.0

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Figure 5: Pie chart showing comorbidities associated with HTN (n=73)

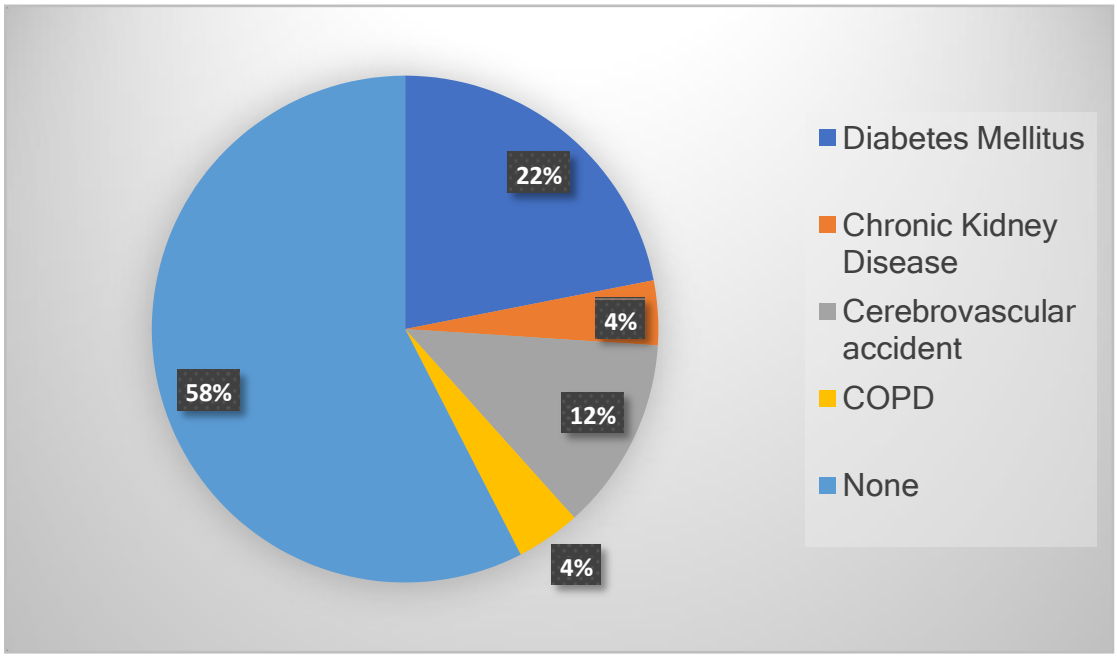
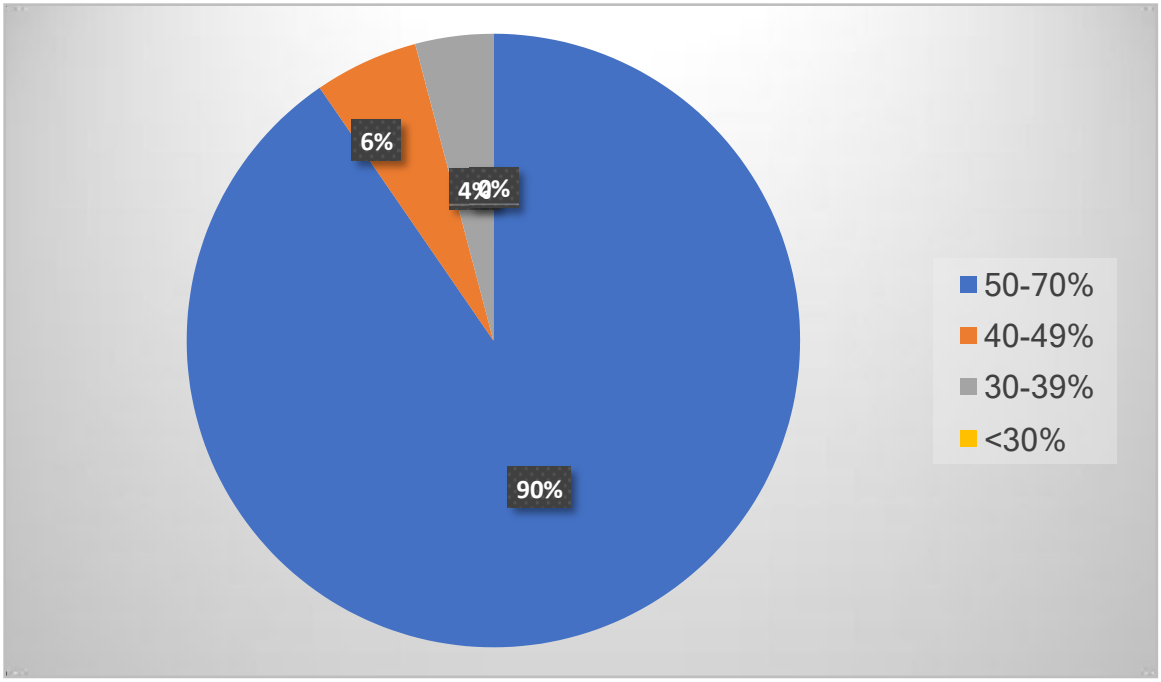


Figure 6: Pie chart showing Left Ventricular Ejection Fraction in patients with Hypertension (n=73)



The following table shows the distribution of patients according to left ventricular dimensions. We can see that most patients i.e. 68.5% had left ventricular internal diameter in the range of 4.1-5.0 cm, 91.8% patients had Interventricular septal thickness between 1.1-1.5

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cm and 90.4% patients and posterior wall thickness between 1.1-1.5 cm.

Table 4: Left ventricular dimensions as per 2-D ECHO (n=73)

Left ventricular dimensions	Number	Percent
LVIDd (cm)		
3.0-4.0	20	27.4
4.1-5.0	50	68.5
5.1-6.0	3	4.1
IVSd (cm)		
0.5-1.0	1	1.4
1.1-1.5	67	91.8
1.6-2.0	5	6.8
PWTd (cm)		
0.5-1.0	3	4.1
1.1-1.5	66	90.4
1.6-2.0	3	4.1
2.1-2.5	1	1.4

LVIDd- Left ventricular internal diameter (diastolic)PWT- Posterior wall thickness
IVSd- Interventricular septal thickness (diastolic)

According to the above data the mean Left ventricular Ejection fraction was 60.8% (S.D. ±8.88%), mean Left ventricular internal diameter was 4.34cm (S.D. ±0.48 cm), mean Interventricular septum was 1.26 cm (S.D. ±0.17cms), mean Posterior wall thickness was 1.21cm (S.D. ±0.19 cm) and mean left ventricular mass was 209.33gm (S.D. ±56.85gm)

Table 5: Descriptive statistics of Left ventricular dimensions

Left ventricular dimensions	Mean	Standard Deviation (S.D)	Range
LVEF (%)	60.88	± 8.88	30-75%
LVIDd (cm)	4.34	± 0.48	3.20-5.80 cm
IVSd (cm)	1.26	± 0.17	0.60 - 1.80 cm
PWTd (cm)	1.21	± 0.19	0.60 - 2.10 cm
LV mass (g)	209.33	± 56.85	85.00 - 413.32 g

Based on electrocardiographic findings 61.64% (45) had Left ventricular hypertrophy according to Sokolow-Lyon index. According to echocardiographic findings 68.49% (50) had Left ventricular hypertrophy.

Table 6: Distribution of patients according to the presence of left ventricular hypertrophy by electrocardiography and echocardiography.

Left ventricular hypertrophy (LVH)	Based on electrocardiography (Sokolow-Lyon Index)		Based on two-dimensional echocardiography (LVMI)	
	Number	Percent	Number	Percent
Present	45	61.64	50	68.49
Absent	28	38.35	23	31.50

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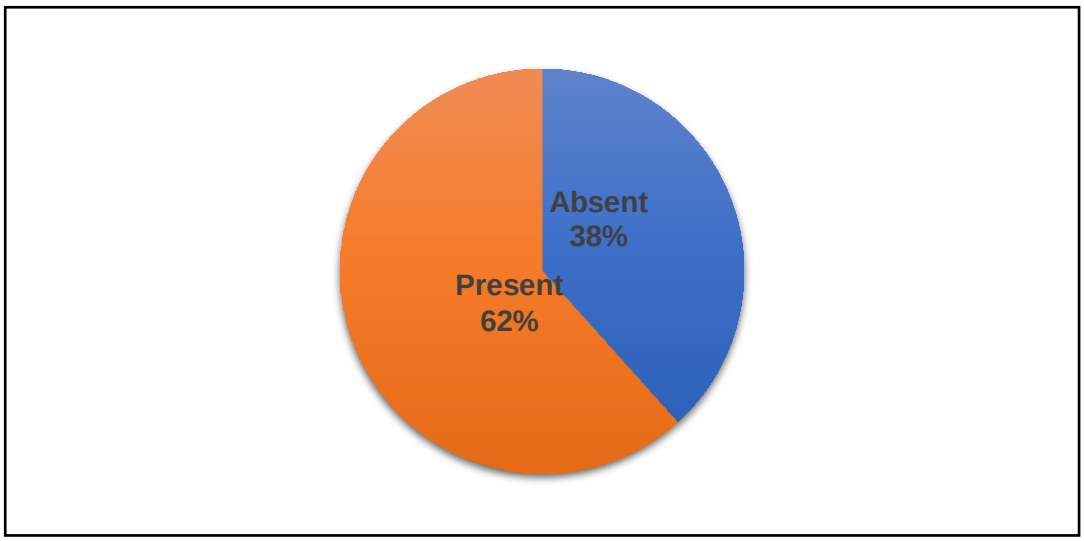
Total	73	100.0	73	100.0

The sensitivity of electrocardiography using the Sokolow-Lyon voltage index in detecting left ventricular hypertrophy in our study was 66% and specificity was 47.82%.

Figure 7: Pie chart showing the presence of LVH by electrocardiography



In our study, 42 patients (57.53%) were on treatment at the time of the studywhile the rest 31 (42.46%) were either not on treatment.

Table 7: Distribution of patient according to treatment history (n=73)

On treatment	Number	Percent
Yes	42	57.53
No	31	42.46
Total	73	100.0

Inferential statistics

In the following table we see that as age advances the presence of left Ventricular hypertrophy also increases.

Table 8: Association between age and left ventricular hypertrophy (byLV mass index)

Age categories	Left Ventricular Hypertrophy	
	Present	**Absent**
20-30 years	0	2 (2.73%)
31-40 years	0	4 (5.47%)
41-50 years	3 (4.1%)	6 (8.21%)
51-60 years	9 (12.3%)	8 (10.9%)
61-70 years	20 (27.39%)	2 (2.73%)
71-80 years	16 (21.9%)	1 (1.36%)
81-90	2 (2.73%)	0
Total	50	23

$x2$ statistic= 2.89, d.o.f =3 and p =0.41

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On applying the Chi-Square test we found that there is no significantassociation between the age of the patient and LVH.

In the study group, 28 (62.2%) males of 45 had left ventricular hypertrophy and 22 (78.57%) females of 28 had left ventricular hypertrophy.

The following table shows us that out of 45 males. In the study 28 (62.2%) had Left ventricular hypertrophy (LVH) on echocardiography and out of 28 females 22 (78.57%) had LVH on echocardiography.

Table 9: Association between sex and Left ventricular hypertrophy (by LV mass index)

Sex	**Left ventricular hypertrophy (LVH)**		**Total**
	Present	**Absent**	
Male	28 (62.22%)	17 (37.77%)	45 (100%)
Female	22 (78.57%)	6 (21.42%)	28 (100%)

$x2$ statistic= 2.14, d.o.f =1 and p =0.19

On applying the Chi-Square test we found that there is no significant association between the sex of the patient and Left ventricular hypertrophy.

The following table shows the presence and absence of Left ventricular hypertrophy (LVH) in patents with the following levels of blood pressure. We see that as the level of blood pressure rises, more number of patients have LVH.

CONCLUSION

The mean age of the patients in the study was 61.89 years with a S.D of ±13.66 years with age of the patients ranging from 23-87 years.The majority of them belonged to the age group of 60 years and above. In previous studies like **Hammond et al**[29]**,** the mean age of hypertensive patients was similar to our study, 53±9.8 years. This study differed to ours in the classification of the patients into groups according to hypertension for the purpose of the study. In the study by **Cuspidi et al**[30] mean age of study group was 53±13 years. Similar findings were found by **Ogah et al** with mean age of 58.2±13.7 years in Nigerian hypertensives.[31] **Peguero et al**[32] reported a mean age of 66±17 years in the hypertensive patients of their study which was similar to the our study.

The diagnosis of Left ventricular hypertrophy (LVH) can be done by electrocardiogram(ECG), X-ray, echocardiographic measurements and MRI. An electrocardiograph (ECG) is less sensitive than echocardiography or MRIin diagnosing Left ventricular hypertrophy (LVH). Commonest electrocardiographic criteria for the diagnosis of left ventricular hypertrophy (LVH) are Sokolow-Lyon voltage criteria, Romhilt and Estes point score system and Cornell voltage criteria. In our study, we have used the Sokolow-Lyon voltage criteria**.** Using this we found that 61.64% patients in our study had left ventricular hypertrophy(LVH). "The sensitivity of electrocardiogram (ECG) in detecting left ventricular hypertrophy(LVH) was 66% and specificity was 47.82%. We also found no significant difference between the proportions of Left ventricular hypertrophy(LVH) detected by electrocardiography and echocardiography."

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Reichek N and Devereux RB reported 21% prevalence of left ventricular hypertrophy (LVH) and with a sensitivity of Sokolow-Lyon voltage criteria being 21% and specificity was 95% in their study. This is in contrast to our study probably considering older methods of LV mass detectionand higher cut off values.

Aronow W.S et al in their study on patients ≥ 62 years reported that the sensitivity of the echocardiography (ECG) criteria ranged from 12-29% and the specificity ranged from 93-96 %.[33]

Recently, **Peguero et al**[32], studied a new technique of electrocardiographic Left ventricular hypertrophy (LVH) detection. In their study electrocardiographic detection of LV hypertrophy by Sokolow-Lyon criteria showed a sensitivity and specificity of 23% and 97% respectively and LVH of 32%.

The present study revealed that the screening for Left ventricular hypertrophy is important for hypertensive patients. In the present study there was a positive correlation between presence of left ventricular hypertrophy and Left ventricular mass.

The pathophysiology of essential hypertension depends on the primary or secondary inability of the kidney to excrete sodium at a normal blood pressure. "Although monogenic forms of blood pressure dysregulation exist, hypertension mostly arises as a complex quantitative trait that is affected by varying combinations of genetic and environmental factors.[4] The sympathetic system, the body sodium levels, volume state, the renin-angiotensin system, functional and structural characteristics of the heart and blood vessels, and some other components are important complementary factors in blood pressure regulation. A deviation from the normal equilibrium among these components, with a persisting non-physiologic increase in pressor factors or in the basal vascular tone and/or cardiovascular reactivity to pressor factors, leads to hypertension".[3]

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