European Journal of Molecular & Clinical Medicine, *2022,*
Volume 9, Issue 2, Pages 2955-2974

Hypertension with reference to Left Ventricular Hypertrophy with Reference to Left Ventricular Mass Index

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Abstract

Better understanding and early recognition of hypertrophy in hypertensive patient helps with effective treatment and prevention of cardiovascular events and morbidity.

It has been established that anti-hypertensive therapy may diminish LVH and hence lessen the risk of serious cardiovascular events in individuals with hypertension. Left ventricular hypertrophy (LVH) is an adaptation of the heart, specifically the left ventricle, to increasing ventricular stress.

Subjects with borderline isolated systolic hypertension in the Framingham Heart study also had modestly elevated left ventricular (LV) layer thickness.[1] It's widely recognized that left ventricular hypertrophy is linked to high blood pressure and the cardiovascular complications that come with it..

Key words: Ventricular, Hypertrophy, ventricle, adaptation, stress, process, distress, borderline, systolic, cardiovascular

Introduction

Left ventricular hypertrophy (LVH) is a process that demonstrates an adaptation of the heart to increasing ventricular stress, specifically the left ventricle in our research instance. In the Framingham Heart Study, patients with borderline isolated systolic hypertension exhibited modestly thickened left ventricular (LV) walls.[1] It is widely recognised that hypertrophy of the left ventricle is associated with hypertension and cardiovascular complications as a result.

Left ventricular mass (LVM) is a well-established metric for measuring left ventricular hypertrophy and predicting unfavourable cardiovascular consequences in patients.

Better understanding and early recognition of hypertrophy in hypertensive patient helps with effective treatment and prevention of cardiovascular events and morbidity.

"The reduction of Left ventricular hypertrophy (LVH) in patients with hypertension

treated with anti-hypertensive medication has been shown to reduce severe cardiovascular events later in the course of the disease."

Aim:

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

Objectives:

To correlate electrocardiography and two-dimensional echocardiographyfindings for detecting Left ventricular hypertrophy.

Review of Literature

Studies on various aspects of the subject and its allied areas are detailed below:

Hypertension:

"Blood Pressure is a quantitative trait that is highly variable".[2] "Clinically, Hypertension maybe defined as the level of blood pressure at which institution of therapy reduces blood pressure-related morbidity and mortality. Epidemiologically, however, there is no absolute level of blood pressure that defines hypertension".[3] It is a frequent, chronic, age related disorder, which often entails debilitating cardiovascular and renal complications.[4] Therefore, age-related increments in blood pressure are constantly happening in all individuals. The basal blood pressure is governed by many factors such as age, gender, race, genetics, environmental factors and lifestyle.[3]

Hypertension can be divided into two main categories depending upon underlying cause. "Essential, primary or idiopathic hypertension is defined as high blood pressure" in which secondary causes are:

1. Renovascular disease,
2. Renal failure,
3. Pheochromocytoma, aldosteronism, or other causes of secondary hypertension or mendelian forms (monogenic) are not present.
4.

"Essential hypertension accounts for 95% of all cases of hypertension. Essential hypertension is a heterogeneous disorder, with different patients having different causal factors that lead to raised blood pressure".[3] Although it has been widely said that "the origins of essential hypertension are unknown, this is only partly accurate since we know so little about genetic differences". A number of factors cause increased blood pressure, including:

1. Overweight,
2. Insulin sensitivity,
3. Excessive alcohol consumption,
4. Excessive salt consumption (in sodium people),
5. Ageing
6. Inactivity,
7. Stress, Poor potassium consumption,
8. Low calcium supplementation

are all risk factors for osteoporosis..[3]

Secondary causes of hypertension encompass all the systemic diseases that can result in raised blood pressure as a consequence of the disease process. These include

1. renal and renovascular causes,
2. diseases of the adrenals,
3. obstructive sleep apnea,
4. pregnancy induced hypertension,
5. miscellaneous endocrine disorders and some medicines.[4]

Diagnosis of Left Ventricular Hypertrophy :

Patients having hypertension all don't build up left ventricular hypertrophy. On physical examination, an S4 gallop can be heard which points to early diastolic dysfunction and possible LVH. "Electrocardiographic findings may include left atrial enlargement, prolongation of the QT interval, and LVH. If we do a graded exercise testing, patients with LVH will have a hyper adrenergic response with an increase in heart rate to about 150 beats per minute within the first minute, systolic blood pressure > 200mmHg and dyspnea on exertion." This can point towards the presence of a possible hypertrophy.[9]

Typical clinical features in a hypertensive woman are overweight, middle- aged, hypertensive, postmenopausal, presenting with a history of exertional chest pain like angina pectoris. The graded exercise test is positive indicating hypertensive heart disease in women. "Left Ventricular Hypertrophy (LVH) in women is a strong cardiovascular risk factor independent of blood pressure".[9]

"The introduction of chest x-rays and the electrocardiograph (electrocardiogram) provided objective information about the structure and function of the heart. Electrocardiography, nowadays, is an essential part of theinitial evaluation for patients presenting with cardiac complaints. As a first line diagnostic tool, health care

providers at different levels of training and expertise frequently find it imperative to interpret electrocardiograms. Specifically, it plays an important role as a non-invasive, cost-effective tool to evaluate arrhythmias and ischemic heart disease".[10]

Echocardiography: [12]

The science of cardiac ultrasonography has advanced over the last 50 years due to the efforts of several people. The emergence and development of echocardiography's multiple modalities, including as A-mode, M-mode, contrast, two-dimensional, Doppler, transesophageal, and intravascular applications, comprise its history. Echocardiography or cardiac ultrasonography depends on the creation, propagation, reflection, and reception of sound waves. Ultrasound refers to the section of the sound spectrum with a frequency higher than 20,000 cycles per second (20 KHz), which is much beyond the hearing range. Echocardiography is the study of the anatomy and function of the heart and major blood arteries using ultrasound. With the advent of piezoelectric transducers, the use of ultrasound for imaging has become feasible. When an alternating electric current is provided, piezoelectric material or crystals quickly change form or vibrate. The sound waves are produced by the fast alternating expansion and contraction of the crystal material.

Review of Literature

The following studies show the work done by other scholars in the field.

Aronow W.S et al, conducted a blinded prospective study in 476 patients of age ≥ 62 years to study the correlation between values of five ECG Criteria and Echocardiography in LVH. They found that 35% had LVH on Echocardiography. "The sensitivity of the ECG criteria ranged from 12 -29%, the specificity from 93-96 %, the positive predictive value (PPV) from 62-71%, and the negative predictive value (NPV) from 67-71 %".[13]

Koren M. J. et al conducted a study to assess the prognostic significance of LVM and geometry in persons with essential hypertension. They reported that LVM was > 125 g/m2 in 69 out of 253 patients (27%); the incidence of cardiovascular events was higher in patients with LVH than without LVH. (26% and 12% respectively; P = 0.006); risk for cardiovascular death was also higher in patients with increased LVM (14% compared to 0.5%; P < 0.001); LVH on ECG did not predict risk. On multivariate analysis, only age and LVM independently predicted cardiovascular events, cardiovascular deaths, and all-cause mortality; Gender, blood pressure and serum cholesterol levels wereinsignificant. [16]

LVMI:

This is necessary to allow for comparison between individuals of different body sizes. [17] The best method however is debatable. "Body surface area (BSA) and height are used to index LVM. BSA has shown stronger statistical correlation especially in the case of HTN related LVH. But, it is also shown that indexing by BSA minimizes the effect of obesity on LVM".[18] Therefore, even height is used for indexing purposes. Height alone can be used or height raised to the power of 1.7 or 2.7 is used.

De Simone G et al in two separate studies have proven that indexing LVM to height$^{2.7}$ i.e. (LVM/height$^{2.7}$) has better predictive value for CVD outcomes as well as better detection of obesity-related LVH.[19, 20]

Chirinos et al showed that indexing LVM to LVM/height$^{1.7}$ was the best method compared to BSA and height for prediction of CVD outcomes, all- cause mortality and identification of obesity-related LVH.[19]

Measurement of Left vetricular size:[17]

The most commonly used parameters are linear dimensions and volumes. They are usually reported for end-diastole and end-systole. They are reported indexed to BSA.

a. Linear methods of LVM measurement:

- Devereux and Reichek "cube" formula:

$LVM = 0.8 \times 1.4 \times [(LVID_d + IVS_d + PWT_d)^3 - LVID^3] + 0.6$

Where, "$LVID_d$ = LV internal diameter at end-diastole"

PWT_d = "inferolateral (posterior)" LV wall thickness at end-diastole IVS_d = "Interventricular septal thickness at end-diastole"

Normal values :

Women= 43-95 g/m^2 and Men = 49-115 g/m^2

- Original ASE formula : [17]

$LVM = 1.04 [(LVID_d + IVS_d + PWT_d)^3 - LVID^3] + 0.6$

- Penn convention formula: [17]

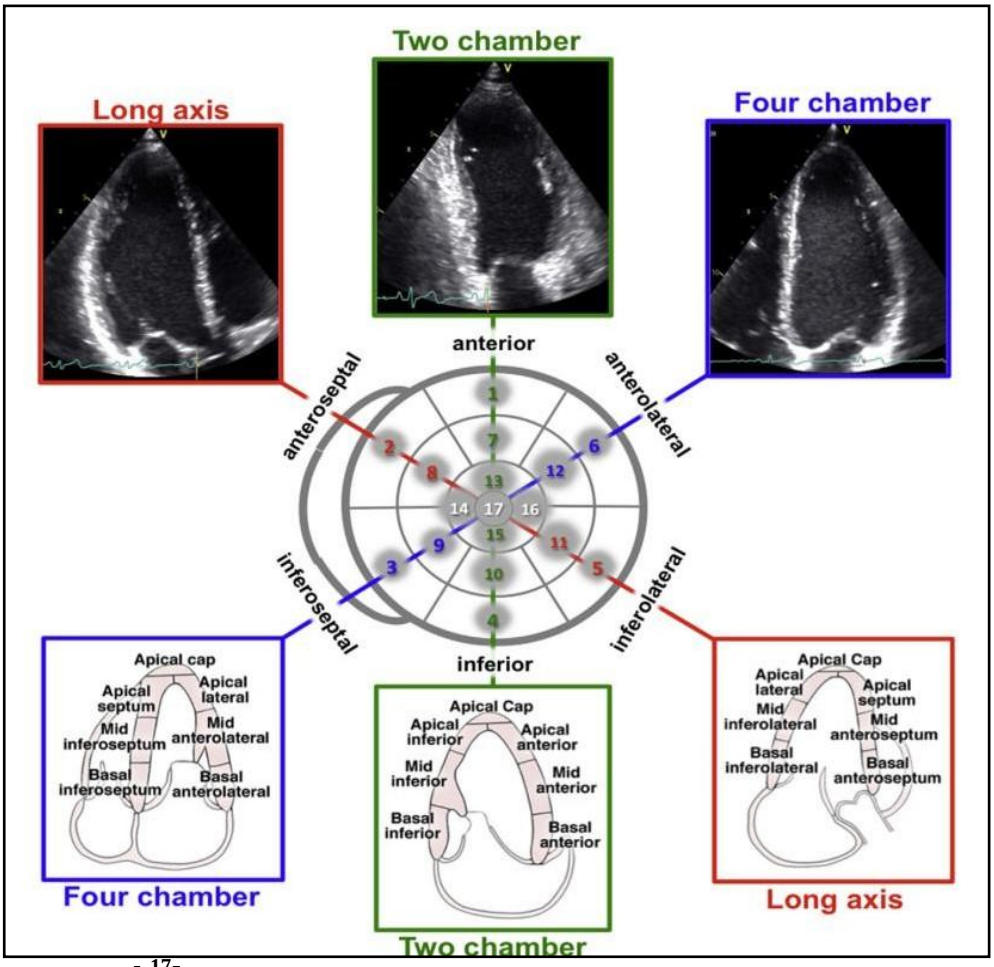
This includes endocardial echoes in the measurement of LVID and not IVSD and PWT. From the LVM calculated using the original ASE formula, 13.6 g has to be subtracted as there is an overestimation.

b. The three-dimensional formula for Left Ventricular Mass (LVM) using volume [17]

"These are based on tracings of the blood tissue interface in the apical four- and two-

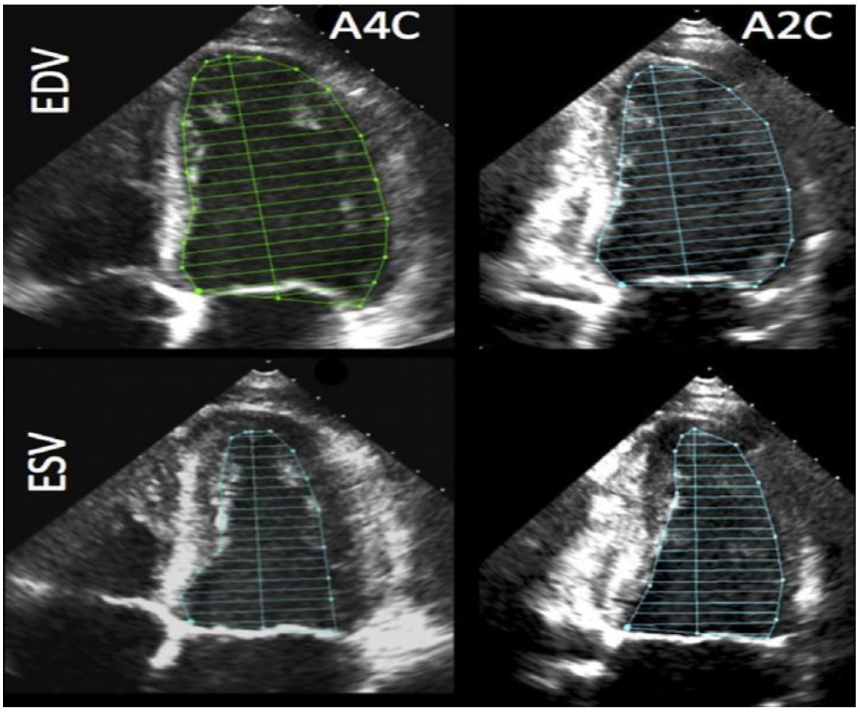
chamber views. (figure 10 and 11) At the level of the mitral valve, a straight line is drawn which connects the two opposite sections of the mitral ring. LV length is calculated as the distance between the middle of this lineand the most distant point of the LV contour".

Figure-1: Orientation of views in relation to LV wall segment



[Source: Lang et al [17]]

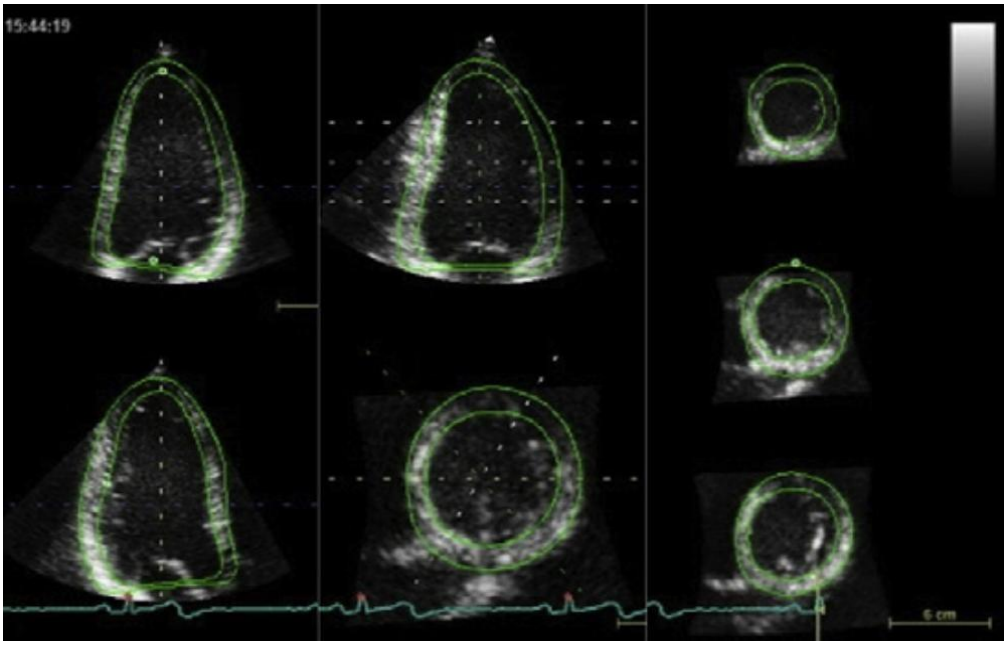
Figure-2: Tracings of the blood tissue interface in the apical four- and two-chamber views [Source: Lang et al [17]]



LV mass = 1.05 x (LV epicardial volume – LV endocardial volume)

= LV myocardial volume x 1.05

Figure-3: 3-D dataset used for LV volume and mass measurement [Source: Lang et al [17]]



In our study we have used the Linear method for LV mass calculation by echocardiography.

Both in Linear and 2D echocardiographic measures, the area is determined at the level of the middle papillary in the parasternal short-axis view and at the end of diastole. Postmortem findings indicate that 2D echo approaches (area-length and truncated ellipsoid) and 2D guided M-mode measures of Left ventricular mass are similar, with minor correlates with autopsy-derived Left ventricular mass.

Material and Methods

This study was conducted on 73 patients 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 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.[21] Hence, we chose p= 25%, q= 1-p i.e. 75%. Using the formula for cross-sectional studies,[22] 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 included in 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 the research purpose.

Procedure:

After obtaining the Ethical clearance, the study was initiated. Data was collected after taking informed and written consent of patients. A detailed case history 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.

Electrocardiogram (ECG):

A standard 12 lead ECG was used to detect LVH by the Sokolow-Lyon Index by voltage,

"S in V_1 + R in V_5/V_6 (whichever is greater) > 35mm/3.5 mV"

Two-dimensional trans-thoracic Echocardiography:

Combined two-dimensional echocardiography with M-mode measurements was done for all patients. "Patients were given a 30-degree left lateral position, and multiple readings were obtained from parasternal views in long and short axis, apical four-chamber view and long-axis views, and sub costal four-chamber and short-axis views. The left ventricular (LV) measurements were taken at end-diastole and the left ventricular internal diameter was measured at the level of mid papillary level."

Data Entry and Analysis:

Data entry was done in Microsoft Excel 2016 and analyzed using SPSS version 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.

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

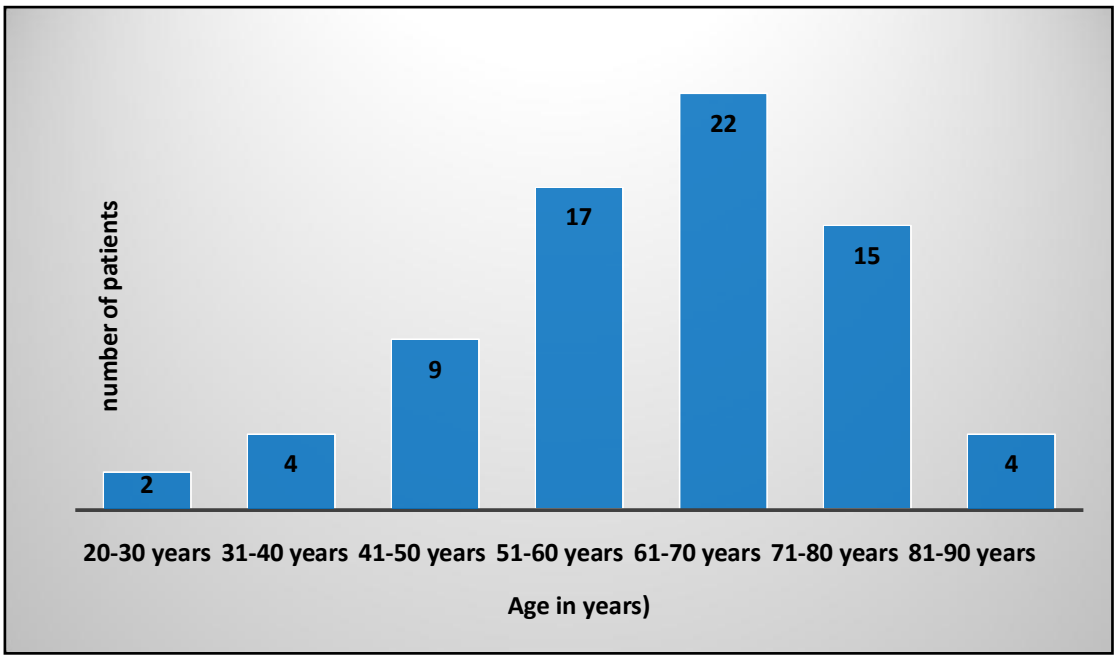
Table-1 of the age distribution of patients in our study group, ranging from 23 to 85 years of age.

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

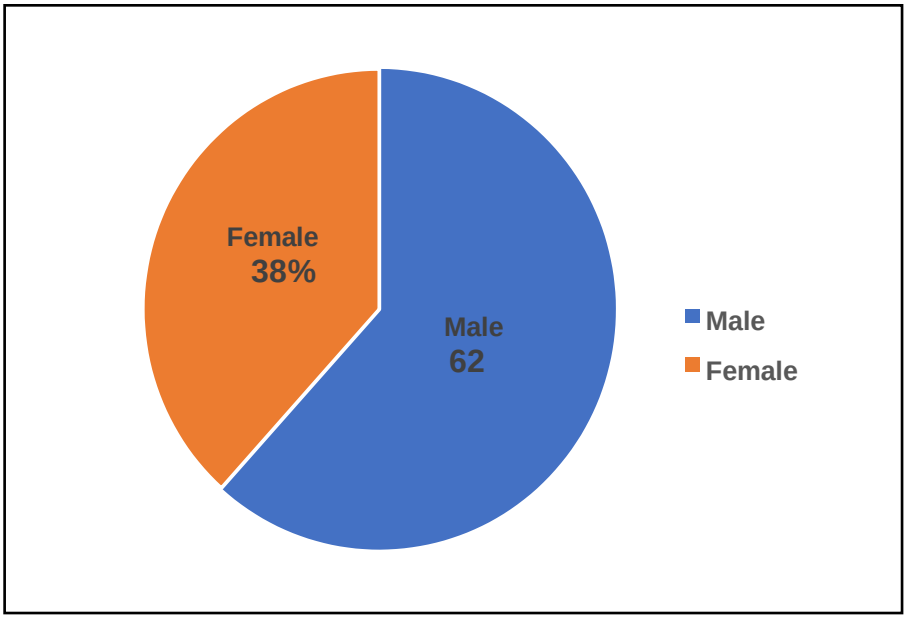
The maximum number of patients i.e. "30.1% were in the age group of 61-70 years followed by 23.3% in the age group of 51-60 years, followed by 20.5% in the age group of 71-80 years. The mean age of patients was 61.89 years (SD ± 13.66, Range = 23-87 years). Of all the patients 2.7% patients were in the 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."

Figure-4: Bar chart showing the age distribution of patients with Hypertension (n=73)



Out of 73 patients in the study group,45 (61.6%) were males and theremaining 28 (38.4%) were females.

Figure-5: Pie chart showing the sex distribution of patients with Hypertension (n=73)



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.

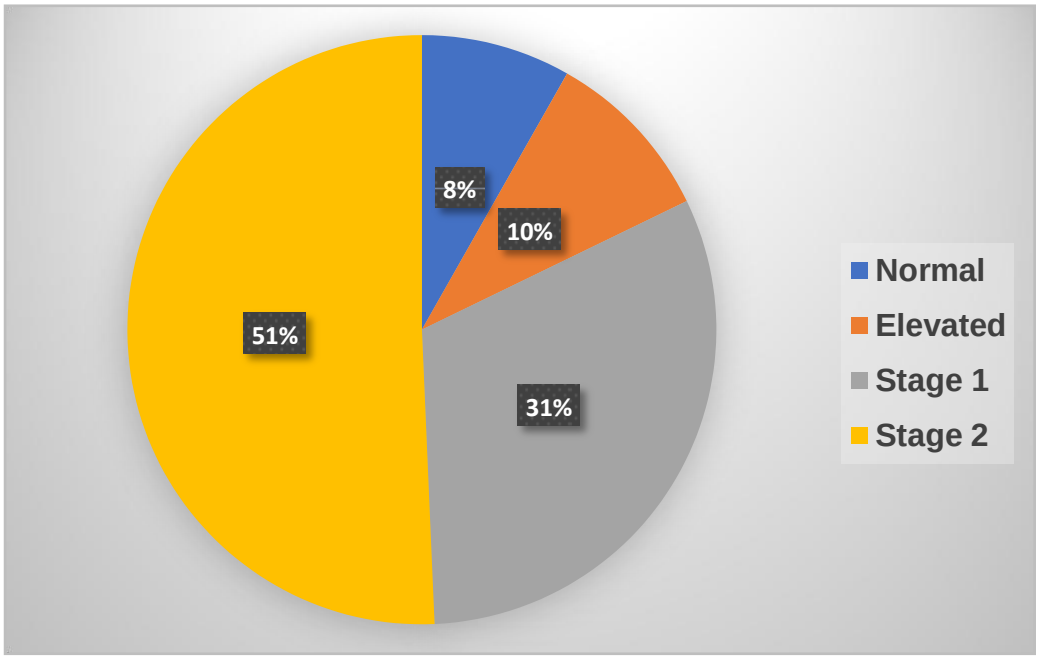
The following Table shows us the distribution of males and females in the study group according to age. It was observed, that maximum number of males i.e. 20.5% were in the age group of 61-70 years followed by 19.1% in age group of 71-80 years, 10.95% in the age group of 51-60 years, 4.1% in age group of 41-50 years, 2.7% in the age groups of 31-40 and 81-90 years and 1.3% in the age group of 20-30 years.

It was observed maximum number of females i.e.12.3% were seen in age group of 51-60 years, followed by 9.5% in 61-70 years, 8.2% in 41-50 years, 4.1% in 71-80 years, 2.7% in 31-40 years and 1.3% in 20-30 years. The majority of the patients among both males and females belonged to the age group of above 60 years.

Table 2: Age and sex distribution of patients with Hypertension (n=73)

	Males		**Females**	
	Number	**Percent**	**Number**	**Percent**
20-30	1	1.3	1	1.3
31-40	2	2.7	2	2.7
41-50	3	4.1	6	8.2
51-60	8	10.95	9	12.3
61-70	15	20.5	7	9.5
71-80	14	19.1	3	4.1
81-90	2	2.7	0	0

Figure 10: A pie diagram showing the grading of Hypertension in patients (n=73)



The following table shows us the distribution of comorbidities in the study group. Out of 73 patients 57.53% had no comorbidity. Most of them 21.91% had Diabetes Mellitus (DM) while 12.32% had Cerebrovascular Accident (CVA), 4.11% had Chronic Kidney Disease (CKD) and 4.11% had COPD.

Table 3: Associated Comorbidities of Hypertensive patients (n=73)

Comorbidities	**Number**	**Percent**
Diabetes Mellitus	16	21.91
Chronic Kidney Disease (CKD)	3	4.11
Cerebrovascular accident (CVA)	9	12.32
Chronic Obstructive Pulmonary Disease (COPD)	3	4.11
None	42	57.53
Total	73	100.00

Most of the patients in the study i.e. 90.4% (66) had left ventricular ejection fraction (LVEF) between 50-70%, 5.5% (4) had ejection fraction between 40-49% and 4.1% (3) had ejection fraction between 30-39%.

Table 4: Left Ventricular Ejection Fraction assessment of patients with Hypertension (n=73)

Left Ventricular Ejection Fraction (LVEF)	**Number**	**Percentage**
50-70%	66	90.4
40-49%	4	5.5
30-39%	3	4.1
<30%	0	0
Total	73	100.0

The following table gives us the distribution of Left Ventricular mass in the study group. In the study group 29 (39.7) patients had Left ventricular mass in the range of 201-250 grams, followed by 25 (34.2%) having left ventricular mass in the range of 151-200 grams. Out of 73 patients in the study 8 (10.9%) had LV mass in 251-300g, 6 (8.2%) in the range of 101-150g, 2 (2.7%) in range of 301-350g, 2(2.7%) in the range of 401-450g, 1(1.3%) in the range of 50-100g and 0 in the range of 351-400g.

Table 5: Left Ventricular Mass in patients with Hypertension on Echocardiography (n=73)

Left ventricular mass (LVM)	Number of patients	Percent
50-100	1	1.3
101-150	6	8.2
151-200	25	34.2
201-250	29	39.7
251-300	8	10.9
301-350	2	2.7
351-400	0	0
401-450	2	2.7

Figure 1 3 : Pie chart showing the distribution of patient according to treatment history

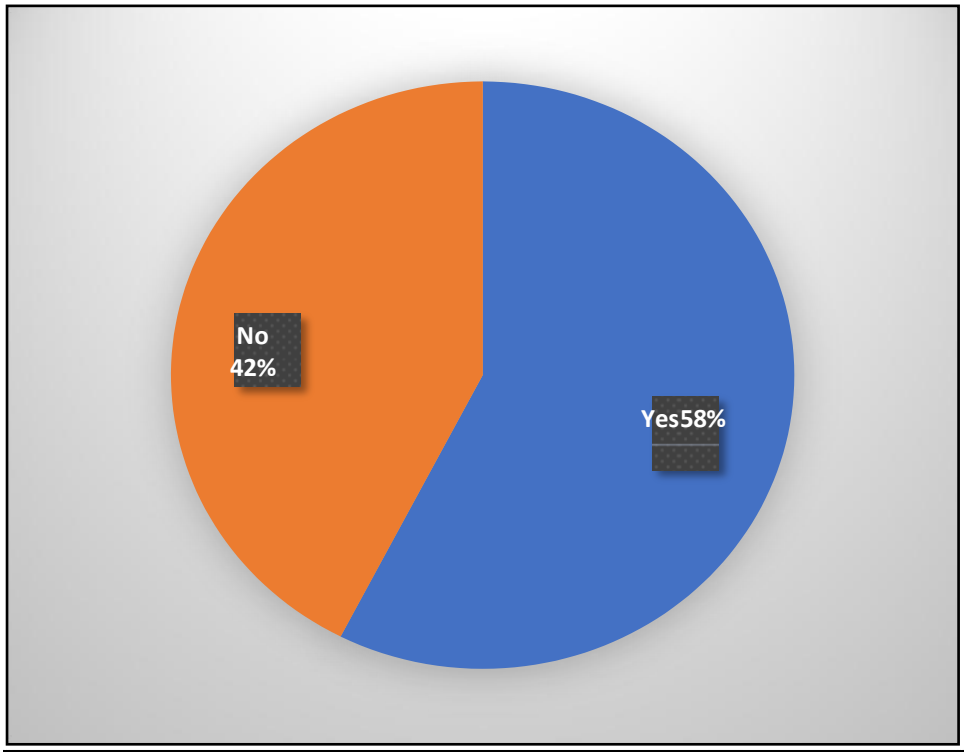


Table-6 shows the presence and absence of Left ventricular hypertrophy (LVH) in patents with the following levels of blood pressure. We see that as the level of blood pressure rises, more number of patients have LVH.

Table 6: Association between the grade of Hypertension and Left ventricular hypertrophy (by LVMI)

Grade of Hypertension	Left ventricular hypertrophy (LVH)	
	Present	Absent
Normal(<120/80mmHg)	5 (10.0%)	1 (4.3%)
Elevated (SBP- 120-129 mm of Hg; DBP- <80 mm of Hg)	3 (6.0%)	4 (17.4%)
Stage 1 (SBP- 130-139 mm of Hg; DBP- 80-89 mm of Hg)	16 (32.0%)	7 (30.4%)
Stage 2 (≥140/90 mm of Hg)	26 (52.0%)	11 (47.8%)
Total	50 (100%)	23 (100%)

SBP- systolic blood pressure

DBP- diastolic blood pressure

Mm of Hg- millimetres of mercury.

2 statistic= 2.81, d.o.f =3 and p =0.42

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

Discussion

The association of left ventricular hypertrophy (LVH) with Hypertension and the cardiovascular consequences due to it is well established. It can cause atrial fibrillation, heart failure, and sudden death. The risk of cardiovascular morbidity and mortality is two to three times in cases with left ventricular hypertrophy(LVH) as compared to cases with normal Left ventricular mass. Left ventricular hypertrophy (LVH) is a strong cardiovascular risk factor independent of blood pressure.

The study found "no significant association between the grade of hypertension and presence of Left ventricular hypertrophy (LVH), however the presence of Left ventricular hypertrophy was more with higher levels of Blood pressure. The mean systolic and diastolic blood pressures were 149.23 ± 23.04 mm of Hg and 88.9 ± 10.17 mm of Hg, respectively." In contrast to our study, **Martinez et al** [23] reported "significant association between systolic and diastolic blood pressure and Left ventricular mass index (LVMI). They had mean systolic blood pressure 144.7±13.4 mm of Hg and 143.2±11.5 mm of Hg in males and females respectively. These values were similar to the values in our study". **Ogah et al**[98], reported "mean systolic blood pressure as SBP (mmHg) 153.2 ± 29.5 mm of Hg and diastolic blood pressure (DBP) as 94.2 ± 18.0 mm of Hg.They found a positive correlation between mean systolic and diastolic blood pressures, and duration of hypertension with LV mass". **Sagie, A et al** reported that "even in subjects of Framingham Heart Study with borderline isolated systolic hypertension, they had mildly increased left ventricular (LV) wall thickness"[4]**. Cuspidi et al** reported "a significant association between the blood pressure levels and Left ventricular mass index with mean systolic BP of 124± 19 mm of Hg and mean diastolic BP of 76± 11 mm of Hg." **Cohen A et al** reported "similar findings to our study, that the blood pressure levels do not influence the presence of left ventricular hypertrophy (LVH). [24] the difference in study results was probably due to a smaller study population and single blood pressure reading in our

study."

Echocardiographic demonstration of Left ventricle hypertrophy (LVH) by measuring Left ventricle mass (LVM) provides prognostic information. Body surface area (BSA) using Dubois formula [25] and was used to index Left ventricular mass, as BSA has shown stronger statistical correlation especially in the case of hypertension-related left ventricular hypertrophy (LVH). It reduces variability due to sex and body size. Based on echocardiographic measurements 68.49% (50) of patients in our study had Left ventricle hypertrophy (LVH) according to Left Ventricular mass Index (LVMI).Various studies have reported the prevalence of LV hypertrophy in Hypertension from 26- 51%.

Cohen et al[24] reported a prevalence of 51% patients with left ventricular hypertrophy (LVH) comparable to our study, however in contrast to our study they had used posterior wall thickness and cross sectional LV area for defining presence of left ventricular hypertrophy (LVH) rather than Left ventricular mass index.

The parameters of age, sex, the grade of hypertension, comorbidities, height, weight and body surface area were not significantly associated with the presence of Left ventricular hypertrophy. **Ogah et al** also found that there was no significant difference in most of the echocardiographic parameters based on gender.[28] In contrast,

Karakan S and Inan B report that age, weight, and body fat percentage are independent factors affecting presence left ventricular mass index (LVMI).[29] this difference was probably owing to the factthat we had not considered such parameters for our study.

Summary of Results and Conclusion

Following conclusions can be drawn from the study-

1. A majority of the patients (50.7%) in our study were in Stage 2 Hypertension followed by Stage 1 Hypertension (31.5%) and 8.2% patients had normal blood pressure.
2. There were 62.2 % of males had have Left ventricular hypertrophy (LVH) and 78.57%of females had Left ventricular hypertrophy (LVH).
3. There was no significant difference between the proportions of leftventricular hypertrophy LVH) detected by ECG and echocardiography (p =0.31).
4. There was no significant association between the age (p =0.41), sex (p=0.19), grade of hypertension (p=0.42) and comorbidities of the patient (p= 0.33) with the presence of Left ventricular hypertrophy (LVH).

There was positive correlation between Left ventricular dimensions and left ventricular hypertrophy. It was perceived that patients with left ventricular hypertrophy had higher values of interventricular septal thickness, posterior wall thickness and Left ventricular mass compared to those with normal left ventricle.

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