# Comorbidities and Left Ventricular Hypertrophy (LVH) – An Exploratory Study

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## Abstract

Hypertension has many adverse effects on multiple systems such as the heart, brain, kidney and vascular systems. While significant progress has been made in our knowledge of the biology of essential hypertension and the availability of viable treatment methods, essential hypertension continues to be a major modifiable risk factor for cardiovascular disease (CVD).. The adverse effects of Hypertension on cardiovascular health of the individual-"Hypertensive cardiovascular disease" was studied in detail by Janeway T C in 1913.

The thickness of arteriolar walls in chronic hypertension is expected to considerably contributes to the deterioration of coronary hemodynamics associated with adaptive ventricular hypertrophy and the subsequent decrease of coronary reserves, which causes ischemia insult to the myocardium. This is thecause of arrhythmias that arise from dead ischemic cardiac tissue and cardiacfailure seen as diastolic dysfunction. These compromise the cardiac functionand such a compromised heart is at riskfor sudden death and morbidity due to the effect on rest of the systems in the body.



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## Introduction

The leading cause of mortality in hypertension people is heart disease. Hypertensive heart disease is the outcome of structural and functional adaptations that contribute to left ventricular hypertrophy, congestive heart failure, atherosclerotic coronary artery disease, cardiac arrhythmias, and other conditions.

The recent categories of Hypertension based on Blood Pressure measurement are (ACC/AHA):

| | |
|---|---|
| Standard | ">120/80 mm Hg" |
| Upraised | "Systolic 120-129 and diastolic >80 mm Hg" |
| Stage-I | "Systolic 130-139 or diastolic 80-89" |
| Stage-II | "Systolic minimum 140 or diastolic min 90 mm Hg" |
| "Hypertensive-crisis" | "Systolic >180 and/or diastolic >120" |

Left ventricular hypertrophy (LVH) "is controlled not just by the physical loading of pressure overload, but also different neurohormonal chemicals that exert trophic effects separately on myocytes and nonmyocytes in the heart. Angiotensin II, aldosterone, norepinephrine, and insulin are trophic agents that directly promote

myocyte hypertrophy and matrix deposition regardless of their impact on systemic artery pressure. These trophic substances induce the generation of growth factors and cytokines, such as transforming growth factor beta, fibroblast growth factor, and insulin growth factor, which directly drive cardiac protein synthesis and hypertrophy. Systolic hypertension is related with the development of Left Ventricular Hypertrophy (LVH). In the Framingham Heart Study, even people with borderline isolated systolic hypertension showed moderately increased left ventricular (LV) wall thickness."

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.

Electrocardiogram:

In the latter decade of the nineteenth century, doctors began using technology in addition to traditional history collection and physical examination to diagnose cardiac disease. In the first part of the 20th century, a series of remarkable discoveries and developments led to the 12-lead ECG as it exists today. The advent of chest x-rays and the electrocardiograph (electrocardiogram) gave objective data on the anatomy and function of the heart. Electrocardiography is now a crucial component of the first examination of patients with cardiac symptoms. As a diagnostic tool of first resort, interpreting electrocardiograms is often a need for medical professionals of varying levels of training and experience. In particular, it serves a crucial role as a non-invasive, cost-effective method for evaluating arrhythmias and ischemic heart disease.[10]

**Review of Literature**

In the study, 61.6% of patients are males and rest 38.4% are females. A high prevalence of left ventricular hypertrophy(LVH)amongfemales i.e. 78.57% compared to 62.2 % in males. This finding is consistentwithpreviousliteraturewhereahigherproportionofwomenhaveleftventricular hypertrophy (LVH) compared to males.[1]

A study by **Gerdts Eet al** reported that "in the study population with 62% males and 38% females,prevalence of left ventricular hypertrophy (LVH) (according to LVMI) was80%in females and 70% in males inhypertension patients, seen both at thebeginning and at end point of their study.[85] However, they had defined leftventricular hypertrophy (LVH) by indexing LV mass to Height$^{2.7}$, unlike ourstudy where we indexed Left ventricular mass (LVM) with Body surface area(BSA)".[2]



**Tingleff J et al** reported that in a study population with 56% malesand44%females, they found as light higher prevalence of Left ventricular hypertrophy(LVH) among hypertensive females (26%)thanhypertensivemales (25%).[3]

In contrast a study by **Ogah et al, "**males were 53.7% andfemales were 46.3%, but found no significant difference in the presence leftventricular hypertrophy (LVH) between males (18.5%) and females (20.3%) intheir study, with females having slightly higher presence of LVH.[4] Our studydidnotfindanystatisticallysignificantassociationbetweengenderandpresence of Left ventricular hypertrophy(LVH)."**Koren M. J. et al**[5] in theirstudy had reported"66% males and 44% females with males having 75% and femaleshaving25%Leftventricularhypertrophy."

Diabetes Mellitus (21.91%)wasthe most common comorbidity associated with

patients of Hypertension in our study followed by Cerebrovascular accident (CVA)(12.32%), Chronic kidney disease(CKD)(4%)andCOPD(4%).**VöllerHetal** reportedcomorbiditieslikecoronaryarterydisease,previous stroke or TIA, abnormal renal function, and diabetes in their study.Their most commonly associated comorbidity was Coronary artery disease(CAD) ranging from 37.2% to 54.7% followed by Diabetes Mellitus whichwaspresent in 22.7%-33.4% of the patients, which is similar to our study. We hadalso excluded patients of CAD inour study witha smaller study group.[6]**Ruilope L. M and Schmieder R. E** similarly reported diabetes in 24.1 % ofnormotensivesubjectsand32.9%ofhypertensivesubjects.[7]

"The mean Left ventricularejectionFraction(LVEF)inourpatientswas60.88± 8.88% and there was found to be no significant association between LVEFand presence of Left ventricularhypertrophy (LVH). Left ventricle ejectionfraction (LVEF) was normal in 90% of patients."**Gerdts et al** also reported"mean left ventricular ejection fraction (LVEF) of 60 ± 9% and majority patientswith normal Left ventricle ejection fraction LVEF (70%)".[8]**Ogah et al**[9] in theirstudy population had a "mean left ventricular ejection fraction of 58.9 ± 13.4%,and in contrast to our study, showed a negative correlation with left ventricularmass (LVM) which they had used to define LVH". **Pegueroet al**[10] reported amean LVEFof57±10%in hypertensivepatientsoftheirstudy.

The mean left ventricular mass index (LVMI) in males was 121.6±32.34 g/m$^2$and mean left ventricular mass index (LVMI) in females was 111.83±22.02g/m$^2$, but there was no statistical difference between the LVMI of males andfemales. The mean LVMI was seen to be higher in males. **Cuspidi et al**[11]studied "the prevalence of left ventricular hypertrophy (LVH) of various studiesand reported that mean LV mass index ranged from 96to 135g/m$^2$.In most ofthestudiesthatwerescrutinised,theyreportedthatmaleshadhigherprevalence of left ventricular hypertrophy (LVH) but there was no statisticalsignificance". In contrast to our findings, **Karakan S and Inan B**[12] report that"female patients in their study had higher left ventricularmass index(LVMI)than male patients, the mean LVMI in females was 94.8 ± 13.1g/m$^2$ and inmales was 89.2 ± 14.6 g/m$^2$, but in contrast to our study left ventricular masswas indexed to height$^{2.7}$". **Martinez et al**[13] reported that the mean LVMI inmales was 121.5± 30.6 g/m$^2$and it was 106.1 ± 27.7 g/m$^2$ in the females intheir study and found that LVMI was higherin males than in females.Incontrast,astudyby**Ogahetal**found nosignificantdifferencebetweentheleftventricularmassindex(LVMI)ofmenandwomenintheirstudypopulation. Though the study had the same framework comparable to ourstudy the difference was probably owing to racial and regional differences inthestudypopulation.[14]



**Discussion and Results**

The study showed that the mean Left ventricular mass (LVM) was 209.33 ±56.85g. The mean LVM in males and females was 226 ± 59.73g and 181.84 ±39.17g, respectively. We also found that the mean values of Left ventricular internal diameter (LVIDd), Interventricularseptalthickness(IVSd)andPosterior wall thickness (PWTd) were 4.34 ± 0.48cm, 1.26 ± 0.17cm, 1.21 ±0.19cm, respectively. Each of the above dimensions had positive correlationwith left ventricular mass and LVMI. **Cuspidiet al**[15], in their study found thatthe mean Left ventricular mass was 152 ± 45g. The mean Interventricularseptal thickness (IVSd) and Posterior wall thickness(PWTd)was1.12±0.15cm and 1.02 ± 0.16cm, respectively of the highest quintiles and they hadsignificant association

with left ventricular mass. **Ogah et al** in their study found that the mean of Left ventricular in ternaldiameter (LVIDd), Interventricular septal thickness (IVSd) and Posterior wall thickness (PWTd)was 4.86 ± 0.95cm, 1.07 ± 0.28cm and 0.997 ± 0.22cm,respectively. They had a statistically significant correlation with increased LVmassi.e.LVhypertrophy.[14] in contrast, **Peguero et al**[10], found no significant correlation between mean Left ventricular internal diameter (LVIDd)(4.40±0.8cm), Interventricularseptal thickness(IVSd)(1.48±0.35cm)andPosterior wallthickness (PWTd) (1.30±0.28 cm) and Left ventricular hypertrophy. But these values were significantly higher in their hypertensive group compared to normotensive group.

Thetable-1gives us the association between thepatients having comorbidities and those free of them with the presence of LV hypertrophy.Left ventricular hypertrophy was found to be 20% (10) in Diabetics, 4% (2) inchronic kidney disease, 8%(4) in stroke patients and 6% (3) in patients ofchronicobstructivepulmonarydisease(COPD).

**Table1:AssociationbetweencomorbiditiesandLeftVentricularHypertrophy**

| **Comorbidities** | **Leftventricularhypertrophy(LVH)** | |
|---|---|---|
| | **Present** | **Absent** |
| **DiabetesMellitus** | 10(20.0%) | 6(26.1%) |
| **CKD** | 2(4.0%) | 1(4.3%) |
| **CVA** | 4(8.0%) | 5(21.7%) |
| **COPD** | 3(6.0%) | 0(0.0%) |
| **None** | 31(62.0%) | 11(47.8%) |
| **Total** | 50(100.0%) | 23(100.0%) |

$_x$2statistic= 4.61,d.o.f=4andp= 0.33

OnapplyingtheChi-Squaretestwefoundthatthereisnosignificant associationbetweencomorbiditiesofthepatientandpresenceofLVhypertrophy.

ThefollowingTable-2givesustheassociationbetweenpresenceandabsenceoftreatmentwiththepresenceorabsenceofLeftventricularhypertrophyinthe 73 patients of the study. We see that 31 patients (62%) on treatment hadleft ventricular hypertrophy while 11 (47.82%) did not. We see that 19 (38%)patients who were not on treatment had left ventricular hypertrophy while 12(52%)didnot.

**Table2:AssociationbetweentreatmentandLV hypertrophy**

| **Treatment** | **Leftventricularhypertrophy** | |
|---|---|---|
| | **Present** | **Absent** |
| Yes | 31(62.0%) | 11(47.82%) |
| No | 19(38.0%) | 12(52.17%) |
| Total | 50(100.0%) | 23(100.0%) |

$_x$2statistic= 1.29,d.o.f=1andp= 0.25

OnapplyingtheChi-Square test we found that there is no significant association between the presence of treatment and presence of Left ventricular hypertrophy(LVH).

The following table-3 shows us the distribution of patients having left


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ventricularhypertrophy according to both electrocardiography and echocardiography. We find that 33(45.2%) patients had LV hypertrophy both on ECG and echocardiography. While 11 (15.1%) patients had LV hypertrophy absent on both ECG and echocardiography.

**Table3:DifferencebetweentheproportionofLeftventricularhypertrophy (LVH) by ECG findings and left ventricular hypertrophy byechocardiography(LVMI)**

| Electrocardiogramfindings | tricularhypertrophy(LVH)byechocardiography | | Total |
|---|---|---|---|
| | Present | Absent | |
| LVHPresent | 33(45.2%) | 12(16.4%) | 45(61.6%) |
| LVH Absent | 17(23.3%) | 11(15.1%) | 28(38.4%) |
| Total | 50(68.5%) | 23(31.5%) | 73(100.0%) |

$_{X}$ 2statistic= 1.27,d.o.f= 1andp=0.31

OnapplyingtheChi-Squaretestwefoundthatthereisnosignificant difference between proportions of Left ventricular hypertrophy (LVH) detectedbyelectrocardiographyandtranstho racicechocardiography.



Table-4 shows us “the relation between Left Ventricular ejectionfraction,Leftventricularinternal diameter,Interventricularseptalthicknessan d posterior wall thickness with Left ventricular mass and LV mass indexindividually. The table shows us that there is a significant correlation betweenleftventricularinternaldiameter,Int erventricularseptalthicknessandposteriorwa ll thickness,each with leftventricularmassindex(LVMI)”

**Table4:CorrelationbetweenLVEF,LVIDd,IVSd,PWTdwithleftventricularmass(LVM) andLeft ventricularmassindex(LVMI)**

| Variablesforcorrelation | on Correlationcoefficient | p-value |
|---|---|---|
| LVejectionfraction andLVmass | -0.016 | 0.894 |
| LV ejectionfraction andLVmassindex | -0.062 | 0.602 |
| LVIDdandLVMass | 0.475 | <0.001 |
| LVIDdandLVmassindex | 0.425 | <0.001 |
| IVSdand LVmass | 0.710 | <0.001 |
| IVSdand LVmass index | 0.710 | <0.001 |
| PWTdand LVmass | 0.757 | <0.001 |
| PWTdandLV mass index | 0.747 | <0.001 |

LVIDd- left ventricular internal diameter;
LV- left ventricle
IVSd-interventricularseptalthickness
PWTd-posteriorwallthickness
LVEF-Leftventricularejectionfraction

The following tables show us the association between 2 groups i.e. one ofpatientshavingLVhypertrophyandtheotherofpatientsnothavingLeftventricularhypertrophy(LVH).

The following table-5 shows us that the mean LV mass in patients with LVH andinthosewithout LVHwas230.14 ±53.33gand164.09 ±33.46g, respectively.

**Table5:RelationofLeftventricularmasswithLeftventriclehypertrophy**

| LVhypertrophy | Number | /mass(g) | S.D (g) |
|---|---|---|---|
| Present | 50 | 230.14 | ±53.33 |
| Absent | 23 | 164.09 | ±33.46 |

S.D-standarddeviation;t-statistic=-5.45,p<0.0001i.e.highlysignificant

Onapplyingindependentsamplet-testitisseenherethatthereisasignificant difference in the mean values of Left ventricular mass between thetwo groups i.e. one having Left ventricular hypertrophy and one without Left ventricular hypertrophy.

The following Table-6 shows us that the mean LV mass index in group with Leftventricle hypertrophy is 130.64g/$m^2$ with S.D ± 24.63 g/$m^2$ and the mean LVmass index in group with no Left ventricle hypertrophy is 90.18g/$m^2$ with S.D±15.48g/$m^2$.

**Table 6: Relation of Left ventricular mass index with Left ventricularhypertrophy**

| Left ventricularhypertrophy(LVH) | Number | an LVMI(g/$m^2$) | S. D |
|---|---|---|---|
| Present | 50 | 130.64 | ±24.63 |
| Absent | 23 | 90.18 | ±15.48 |

t-statistic=-7.233,p<0.0001i.e.highlysignificant

On applying independent sample t-test it is seen that there is a significantdifference in values of Left ventricular mass index between the mean of twogroups i.e. one with Left ventricular hypertrophy (LVH) and the other with noleftventricularhypertrophy(LVH).

The following table shows us that, the mean left ventricle internal diameter(LVIDd) in patients with LV hypertrophy is 4.43cm with S.D± 0.48cm, and inpatientswithoutitis4.14cm withS.D±0.42cm.

**Table 7: Relation of Left Ventricular internal diameter (LVIDd) with leftventricularhypertrophy(LVH)**

| Left ventricularhypertrophy(LVH) | Number | n LVIDd(cm) | S. D |
|---|---|---|---|
| Present | 50 | 4.43 | ±0.48 |
| Absent | 23 | 4.14 | ±0.42 |

t-statistic=-2.420,p=0.018

On applying independent sample t-test it is seen that there is a significantdifference in values of left ventricle internal diameter (LVIDd) between the mean of two groups i.e. one with Left ventricular hypertrophy (LVH)andanotherwithnoLeftventricularhypertrophy(LVH).


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FromthefollowingTable-8, it is seen that the mean Interventricular septal thickness(IVSd) in the group of patients having LVhypertrophyis1.32cmwith S.D. ± 0.16cm, and it is 1.13cm with S.D.± 0.13cm in those patients nothavingLVhypertrophy.

**Table 8: Relation of Interventricular septal thickness (IVSd) with Leftventricularhypertrophy(LVH)**

| **Left ventricularhypertrophy( LVH)** | **Number** | **MeanIVSd(cm)** | **S. D** |
|---|---|---|---|
| Present | 50 | 1.32 | ±0.16 |
| Absent | 23 | 1.13 | ±0.13 |

t-statistic=-5.229,p<0.0001i.e.highlysignificant



On applying independent sample t-test it is seen that there is a significantdifference in values of interventricular septal thickness (IVSd) between themean of two groups i.e. one with LV hypertrophy and another with no LVhypertrophy.

Fromthefollowingtableweseethat,themeanposteriorwallthickness(PWTd) of patients with LV hypertrophy is 1.27 cm with S.D.± 0.19cm and inthose withnoLVhypertrophyitis1.09cmwithS.D.±0.12cm.

**Table9:Relationofposteriorwallthickness(PWTd)withLeftVentricularhypertrophy(LVH)**

| **LVH** | **Number** | **Mean PWTd (cm)** | **S. D** |
|---|---|---|---|
| Present | 50 | 1.27 | ±0.19 |
| Absent | 23 | 1.09 | ±0.12 |

t-statistic=-3.857,p<0.0001i.e.highlysignificant

On applying independent sample t-test it is seen that there is a significantdifference in values of PWTd between the mean of two groups i.e. one with Left ventricular hypertrophy (LVH)andtheotherwithoutleftventricularhypertrophy(LVH).

- Forotherparameterslikeheight,weightandbodysurfacearea,nosignificant difference was found between the mean of two groups i.e. onewithLVHandanotherwithnoLVH.(all p>0.05)
- ThemeanLVMinmaleswas226.44±59.73 andinfemaleswas181.84± 39.17. On applying independent sample t-test, this difference was found tobestatisticallysignificant.(p=0.001)
- ThemeanLVMIinmaleswas121.66±32.34g/$m^2$andinfemaleswas 111.83±22.02g/$m^2$.Thisdifferenceisnotstatisticallysignificant.(p=0.161)
- Mean BSA in males was 1.75 ± 0.15 $m^2$ and females was 1.77 ± 0.13 $m^2$.Thisdifferenceisnotstatisticallysignificant.(p=0.622)

**Summary and Conclusions**

Following conclusions can bedrawn fromthe study-

- Diabetes Mellitus (21.91%) was the most common comorbidity associatedwith patients of Hypertension in our study, followed by CVA (12.32%).There was no associated comorbidity in 57.53% of patients.

- The mean Left ventricular rejection fraction(LVEF)inthe studywas60.88%(S.D±8.88%)
- In the study, 42 patients (57.53%) were on treatment, but there was foundto be no significant association between treatment and presence of Leftventricularhypertrophy(LVH).
- There is a significant difference inmean values of LV parameters likeLVIDd, IVSd, and PWTd in patients with Left ventricular hypertrophy(LVH)and with no Left ventricular hypertrophy(LVH).(p<0.0001)

Itwillbeofprodigioushelpinscreeninghypertensive patientsfor the parameters ofleftventricular hypertrophy andLeft ventricular mass which can in long term help in better management ofhypertensionandfutureoutcomes.

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