

Correlation of Hypertensive Left Ventricular Hypertrophy with Renal End Organ Damage

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ABSTRACT

Aim: To find a correlation, of patients of LVH (cardiac end organ damage) with renal end organ damage.

Settings: Cardiology Department Mayo Hospital Lahore .Pakistan

Duration: 06 months. February 2011 to August 2011.

Methods: This descriptive cross sectional study was conducted in the department of cardiology Mayo Hospital Lahore from February 2011 till August 2011. Sixty (60) Hypertensive patients, 38 (63.33%) male and 22 (35.48%) female were enrolled in for study after informed consent, from out- patient of department of cardiology Mayo Hospital Lahore. They were assessed by 2-D Echocardiography for presence of LVH using standard M-mode section for measurement of Left ventricular posterior wall and interventricular septum thickness in left lateral decubitus position..Urinary albumin excretion (microalbuminuria) for 24 hours and urinary creatinine clearance were measured by collecting urine for 24 hours.Both were used as markers for renal insufficiency.

Results: The mean age of patients was 50.58 ± 10.42 with minimum observed age of 30 years and maximum age of 70 years. There were 38 (63.33%) male and 22(35.48%) female patients included in the study. The microalbuminuria was found to be 52.64 ± 72.92 with minimum observed value of 4 and maximum value of 282.00 Out of total 60 patients microalbuminuria was present in 16(26.67%) and was absent in rest of 44 (73.33%) subjects .The mean creatinine clearance observed was 92.69 ± 18.35 with minimum observed value of 32.00 and a maximum value of 120 ml/min.

Conclusion: Our study revealed a significant relationship between left ventricular hypertrophy and renal end organ damage

Keywords: Left Ventricular Hypertrophy, Microalbuminuria, Renal End Organ Damage,,Chronic Kidney Disease

INTRODUCTION

The left ventricular hypertrophy is an established cardiovascular risk factor and its presence increases the risk of coronary atherosclerosis, arrhythmias and cardiac failure¹. LVH has also been found associated with renal insufficiency .Hypertensive left ventricular hypertrophy also causes a change in left ventricular geometry².Increased fluid volume, decreased renal function and increased risk of cardiovascular risk are all associated with LVH³. In patients with LVH renal volume has also been found to be increased as compared to people with normal blood pressure⁴. Preclinical end organ damage besides the kidneys has been noticed in patients with hypertension. Who have mild to moderate renal dysfunction?

Increased urinary albumin excretion and decreased creatinine clearance are indicators of worsening renal function⁵. The evaluation of these parameters may help in early identification of such individuals suffering from covert renal dysfunction. .Longstanding uncontrolled hypertension leads to renal dysfunction as a result of ischemic nephropathy even in the absence of malignant hypertension⁶. Similarly patients with microalbuminuria have greater degree of target organ damage, be it retinal changes or LVH or renal creatinine clearance⁷. Microalbuminuria is not only a strong predictor of renal end organ damage but also an indicator of increased capillary leakage causing abnormality not only in renal microcirculation but also it shows generalized endothelial barrier dysfunction predisposing to further cardiovascular complications⁸.

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LVH and renal dysfunction (microalbuminuria and decreasing creatinine clearance) are indicative of end organ damage in hypertensive individuals. Renal dysfunction has been noted in patients with LVH and vice – versa⁹.

This study was designed to find out correlation of hypertensive LVH with renal end organ damage. LVH, microalbuminuria, and decreased creatinine clearance are all markers of end organ damage due to hypertension .Identification of one marker of damage in a system may suggest presence of damage in the other system.

MATERIALS AND METHOD

The study was carried out in the cardiology department Mayo Hospital Lahore from February 2011 till August 2011. Nonprobability based purposive sampling was done and sixty hypertensive patients were taken in to study after proper informed consent. The study was approved by the institutional review board and ethical committee of the hospital.

Inclusion criteria: All hypertensive patients of either sex between 30 to 70 years age with evidence of left ventricular hypertrophy on echocardiography.

Exclusion criteria:

1. Patients with already established chronic renal failure.
2. All patients with acute renal failure
3. Patients with diabetes mellitus (Fasting Blood Glucose greater than 126mg/dl).
4. Patients suffering from urinary tract infection based on history and routine urinary examination if required.

Data collection: Sixty patients fulfilling the inclusion criteria were enrolled in study through outpatient department of Mayo Hospital Lahore. Informed written consent was obtained from each of them. After detailed history and

physical examination and routine lab investigation were performed then specific study oriented special investigations were done. This included 2D Echocardiography for documentation of Left Ventricular Hypertrophy, Left ventricular size, ejection fraction (EF) and function etc. Urinary creatinine clearance and microalbuminuria were also determined for every patient included in the study.

For every study patient echocardiography study was performed in detail in the department echocardiography lab by an experienced echocardiographer and all measurements and different parameters were assessed in standard echocardiographic views using Real time 2D and M mode echo studies. Other causes of ventricular hypertrophy like Aortic stenosis and hypertrophic cardiomyopathy were excluded. Left ventricular hypertrophy was defined on the basis of thickness of left ventricular posterior and interventricular septal wall thickness in diastole (M. mode measurement, greater than 1.2 cm being taken as indicative of hypertrophy).

The twenty four hour urine collection was done to estimate 24 hour urinary creatinine clearance as well as to document microalbuminuria. At the same time serum creatinine was also determined. Creatinine clearance was calculated using the following formula

$$\text{Creatinine Clearance (CrCl)} = \frac{U \text{Cr} \times UV}{SCr \times 1440}$$

Where; CrCl is Creatinine clearance, UCr is urinary creatinine mg/dl

SCr is serum creatinine in mg /dl

UV is 24 hour urinary volume in ml

1440 are minutes in 24 hours.

BP Classification	Systolic BP(mmHg)	Diastolic BR(mmHg)
Normal	<120	And <80
Prehypertension	120-139	Or 80-89
Stage 1 hypertension	140-159	Or 90-99
Stage 2 hypertension	≥160	Or ≥100

Renal damage: Renal damage was assessed by documenting presence of microalbuminuria and measuring creatinine clearance. Microalbuminuria: was defined as presence of albumin excretion of 30 to 300 mg per 24 hour urinary excretion. Creatinine Clearance (CrCl) taken as volume of blood plasma that is cleared of creatinine per unit time. It's a highly reliable indicator of renal function.

Normal CrCl>greater than 90 ml /min

Mild Renal Insufficiency =60 to 89 ml /min

Moderate Renal insufficiency = 30 to 59 ml /min

Severe Renal insufficiency =less than 30 ml /min

Data analysis: The information collected from the patients and laboratory results were analyzed by using SPSS version 20. The descriptive statistics were calculated. The quantitative variables like age, microalbuminuria, creatinine clearance, left ventricular posterior and inter ventricular wall thickness etc. were presented as mean and standard deviation. The qualitative variables like sex, severity of left ventricular hypertrophy, microalbuminuria and renal insufficiency were presented in form of frequency and percentages along with Pie charts. Spearman's Rank correlation was applied to see the correlation of LVH with renal end organ damage .P value < than 0.05 was considered as significant.

RESULTS

The mean age of patients was 50.58±10.42 with minimum observed age of 30 years and maximum age of 70 years. (Table1.) There were 38(63.33%) male and 22(36.67%) female patients included in the study. The microalbuminuria was found to be 52.64±72.92 mg/dl with minimum observed value of 4 and maximum value of 282.00mg/dl (Table 2.) Out of total 60 patients microalbuminuria was present in 16(26.67%) and was absent in rest of 44(73.33%) subjects. The mean creatinine clearance observed was 92.69±18.35 with minimum observed value of 32.00 and a maximum value of 120ml/min (Table 3). The level of creatinine clearance was normal in 47(78.33%) patients and it was towards mild renal insufficiency in 12 (20%) patients. Moderate renal insufficiency was noted in 1(1.67%) patient.

The mean value of left ventricular posterior wall thickness as determined by echocardiography was 1.30±0.17cm with minimum observed value of 1.20 and a maximum value of 1.90cm. (Table 4) Mild LVH was present in 33(55%), moderate LVH was seen in 24(40%) and severe LVH was found in 3(5%) (Table 4).

Highly significant positive correlation was found between LVH determined by echocardiography and microalbuminuria level (Spearman's rho p value <0.591). Also a statistically significant negative correlation was observed between the LVH as determined by posterior wall thickness and creatinine clearance (Spearman's rho p value = 0.557(0.000) (Table-5).

Table 1:

	Age in years
Mean	50.58
STD deviation	10.42
Range	40.00
Minimum	30
Maximum	70

Table 2: Microalbuminuria:mg/dl

Mean	52.64
Std deviation	72.92
Range	278.00
Minimum	4.00
Maximum	282

Table 3: Creatinine clearance; ml/min

Mean	92.69
Std deviation	18.35
Range	88.00
Minimum	32.00
Maximum	120

Table 4: Left ventricular hypertrophy as determined by posterior wall thickness:

	Posterior wall thickness
Mean	1.304cm
Std deviation	0.17cm
Range	0.70cm
Minimum	1.20cm
Maximum	1.90cm

Table 5: Correlation of hypertensive left ventricular hypertrophy with renal end organ damage:

	Micro albuminuria	Creatinine clearance	LVH
Microalbuminuria			
Spearman's rho	1.000	-159	0.591
p- value		0.216	0.000
Creatinine Clearance			
Spearman's rho	-159	1.000	-557
p-value	0.216		0.000
LVH			
Spearman's rho	0.591	-557	1.000
p-value	.000	.000	

Correlation is significant at the 0.01 level (2-tailed)

Fig. 1: Gender distribution

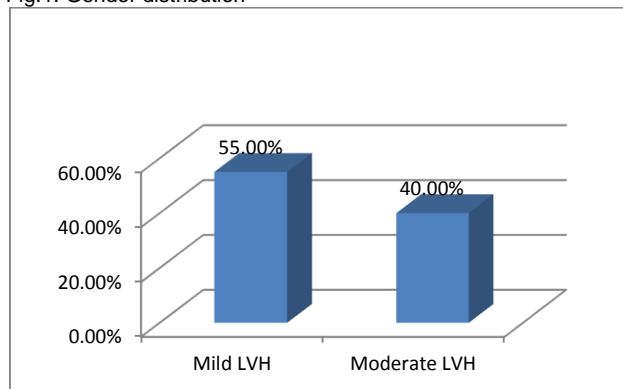


Fig. 2: Microalbuminuria

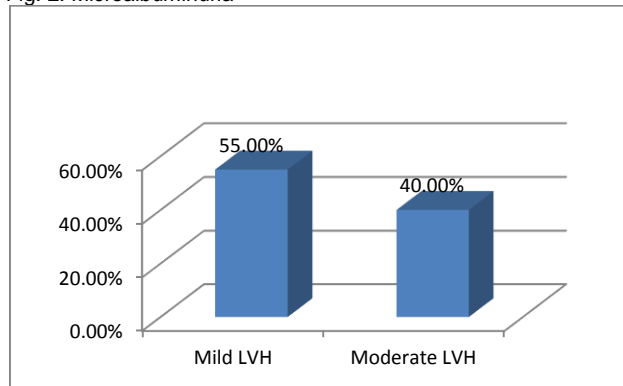


Fig. 3: Severity of renal insufficiency

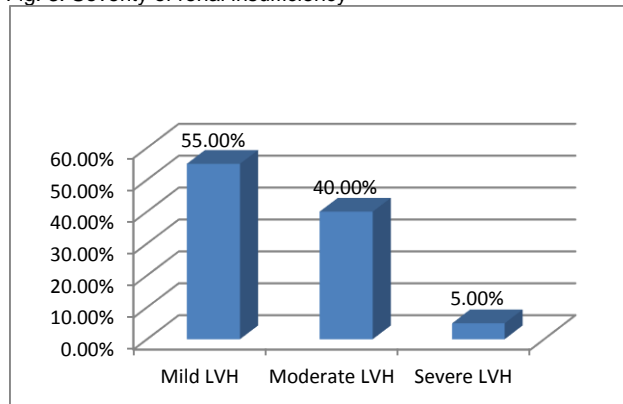
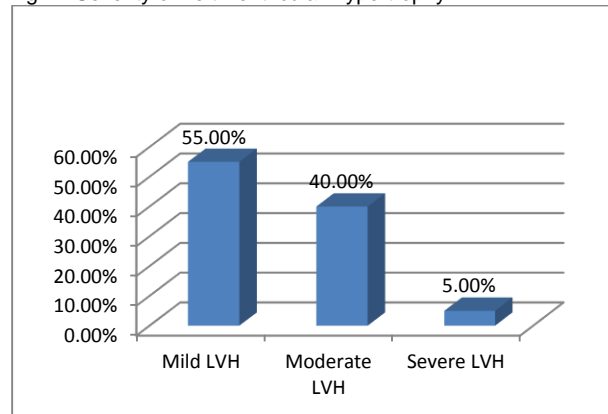


Fig- 4: Severity of Left Ventricular Hypertrophy



DISCUSSION

LVH is present in 60 to 80% of patients with End Stage Renal Disease on renal replacement therapy¹⁰. The risk of cardiovascular adverse events in form of coronary heart disease and cardiac failure is considerably greater in patients of renal disease as compared to general normal population¹¹. Cardiovascular mortality in patients with chronic kidney disease is 10 to 20 times higher as compared to normal individuals¹².

Echocardiography is still an investigation of choice for measuring LV mass and assessing LVH as it is easily available and quite cheap as compared to other modalities like Cardiac MRI. The mean age of patients in our study population turned out to be 50.58 ± 10, Minimum age was 30 years and maximum as 70. Out of total 60 patients 38 (63.33%) were male and 22 (35.48%) female. Our study population age matched with that seen in many other larger studies on cardiovascular diseases like Framingham Study and others which revealed that with advancing age mortality and morbidity increases due to cardiovascular problems like cardiac failure and hypertensive heart disease¹³.

The mean microalbuminuria in our patients was found to be 52.64±72.92 with a minimum of 4.00 mg per 24 hours and a maximum value of 282.00mg/24 hours. Of 60 patients, microalbuminuria was present in 16 (26.67%) subjects and in rest of 44 (73.33%) it was absent. The mean creatinine clearance observed was 92.69±18.35 with minimum observed value of 32.00ml/min and a maximum value of 120ml/min. The level of creatinine was normal in 47 (78.33%) patients, whereas mild renal insufficiency was noted in 12 (20%) patients. Moderate renal impairment was seen in only 1 (1.69%) individual. The mean LVH value was 1.307±.17 cm with minimum as 1.20cm and maximum as 1.90cm. Mild LVH was present in 33 (53.23%), moderate LVH in 24 (38.71%) and severe LVH seen in 3 (5%) of the patients.

In one study the urine albumin creatinine ratio (UACR) and echocardiographic measure of LV structure and function were obtained in 833 patients with Stage 1 to 3 Hypertension and LV hypertrophy determined by electrocardiogram. Overall 23% had microalbuminuria, whereas patients with eccentric or concentric hypertrophy of LV had higher prevalence of microalbuminuria (average 26% to 30% vs 9%, p value less than .001). In multiple regression analysis higher LV mass was associated with

higher UACR ($B = 169$, P less than .001) and patients with microalbuminuria had a significantly higher LV mass and lower endocardial and mid wall fractional shortening¹⁴. Yet another study of Stage 2 and 3 Hypertension showed the presence of microalbuminuria in 23%.

Microalbuminuria was more prevalent patients with ECG left ventricular hypertrophy by both ECG-criteria (29%)¹⁵. Another research conducted on 175 subjects showed that mean creatinine was $403 \pm 207 \mu\text{mol/L}$, representing a creatinine clearance (Crcl) of $25.5 \pm 17 \text{ mL/min}$. Left ventricular hypertrophy was present in 38.9% of the population studied. They demonstrated that the prevalence of LVH increases with progressive renal decline; 26.7% of patients with Crcl greater than 50 mL/min had LVH, 30.8% of those with Crcl between 25 and 49 mL/min had LVH, and 45.2% of patients with severe renal impairment (Crcl < 25 mL/min) had LVH ($P = 0.05$). The mean LVMI was significantly different among the three groups (97.58 g/m^2 , 114.4 g/m^2 , respectively; $P < 0.001$)¹⁶. All reported results of both microalbuminuria and creatinine clearance were similar to our findings. Highly significant positive correlation was found between LVH as determined by posterior wall thickness and microalbuminuria level (spearman's rho (p-value) = 0.591 (0.000)). Also a statistically significant negative correlation was observed between the LVH and creatinine clearance (spearman's rho (p-value) = -0.591 (0.000)). Similar results were found in another study that examined the prognostic significance of left ventricular hypertrophy determined by echocardiography in a cohort beginning renal replacement therapy. The relative risk for all-cause mortality and for cardiac mortality, based on comparison of upper and lower quintiles of left ventricular mass index, was 3.7 for both. Whilst the independent risk for cardiac mortality was 2.7. Therefore they concluded that left ventricular hypertrophy was an important, independent, determinant of survival in patients receiving therapy for end-stage renal failure¹⁷.

In another study, 162 subjects with chronic kidney disease (CKD) underwent echocardiograms to see if there existed some association between left ventricular hypertrophy and coronary artery calcification in patients with chronic kidney disease (CKD) with elevated FGF-23 concentrations. It was found that risk of left ventricular hypertrophy (odds ratio per 1-SD increase in log FGF-23, 2.1; 95% CL: 1.03 to 4.2)¹⁸. Yet another study showed that end stage renal disease (ESRD) patients have a higher cardiovascular mortality rate. They showed that among all patients included in their study 14% had coronary artery disease, 19% angina pectoris, 31% cardiac failure, 7% dysrhythmia and 8% peripheral vascular disease. On echocardiography 15% had systolic dysfunction, 32% left ventricular dilatation and 74% left ventricular hypertrophy.

It appears evidently from the results of our study and their compatibility with existing literature that progressive renal disease may occur in patients with cardiovascular disease that may worsen the complications and obscure therapeutic strategies.

CONCLUSION

Our study revealed a significant relationship between left ventricular hypertrophy and renal end organ damage (i.e.,

increased microalbuminuria and decreased creatinine clearance)

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