ORIGINAL ARTICLE

Correlation of Hypretensive 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.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 LV posterior wall thickness as determined by echocardiography was 1.30±0.17 with minimum observed value of 1.20cm and a maximum value of 1.90cm.
Keywords: Left Ventricular Hypertrophy, Microalbuminuria, Renal End Organ Damage,

INTRODUCTION

The left ventricular hypertrophy is an established cardiovascular risk factor and its presence increases the risk of coronary atherosclerosis, arrhythmias and cardiac failure[1]. LVH has also been found associated with renal insufficiency .Hypertensive left ventricular hypertrophy also causes a change in left ventricular geometry[2].Increased fluid volume, decreased renal function and increased risk of cardiovascular risk are all associated with LVH[3]. In patients withLVH renal volume has also been found to be increased as compared to people with normal blood pressure[4].Preclinical end organ damage besides the kidneys has been noticed in patients with hypertension. Who have mild to moderate renal dysfunction?

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Increased urinary albumin excretion and decreased creatinine clearance are indicators of worsening renal function[5]. 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 hypertension[6]. Similarly patients with microalbuminuria have greater degree of target organ damage, be it retinal changes or LVH or renal creatinine clearance[7]. 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 complications[8].

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 versa[9].

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 30-70 years of 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 microalbuminurea 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 microalbuminurea. At the same time serum creatinine was also determined. Creatinine clearance was calculated using the following formula:

Creatinine Clearance(CrCl)= U Cr x UV/SCr 1440

Where;

- Crcl is Creatinine clearance
- UCr isurinary creatinine mg/dl
- SCr is serum creatinine in mg /dl
- UV is 24 hour urinary volume in ml
- 1440 are minutes in 24 hours.

Hypertension definition: Patients were labeled as Hypertensive after measuring arm blood pressure under complete resting conditions according to the prevailing JNC 7 guidelines on diagnosis and treatment of hypertension.

BP classification	Systolic BP(mmhg)	Diastolic BP (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.

Microalbuminurea: 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 t0 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, microalbuminurea 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 (Table 1). 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 282.00 mg/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 01(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 vetricular hypertrophy as determined by posterior wall thickness

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

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

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

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

Fig 1: Gender distribution of the patients

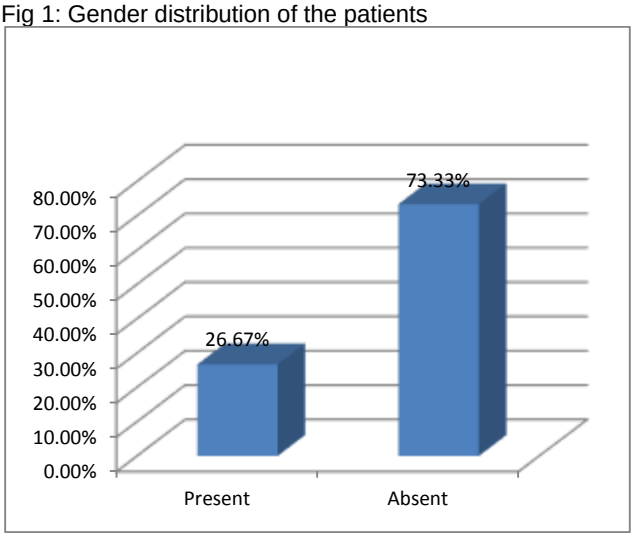


Fig. 2: Microalbuminuria

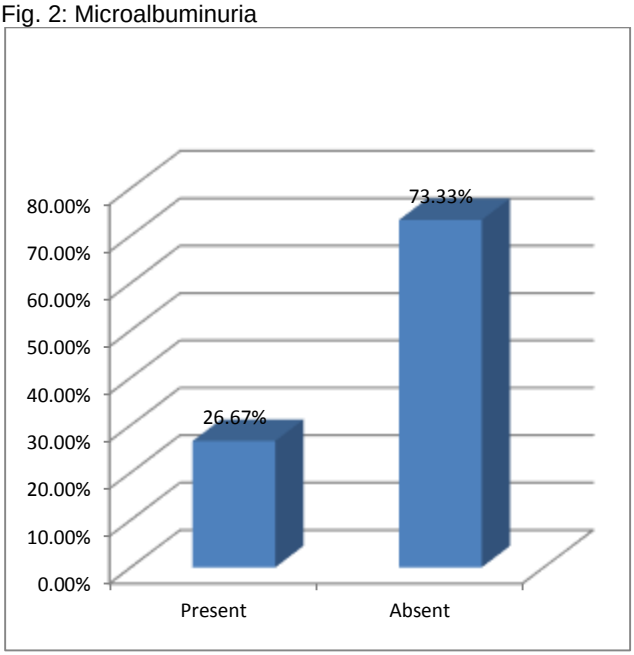


Fig. 2: Severity of renal insufficiency

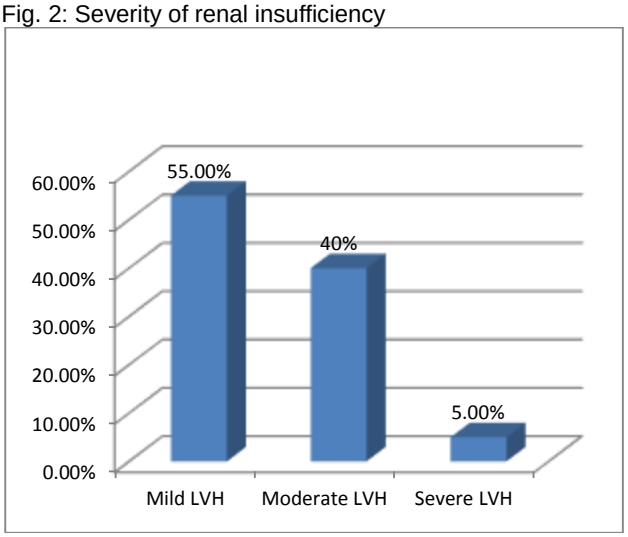
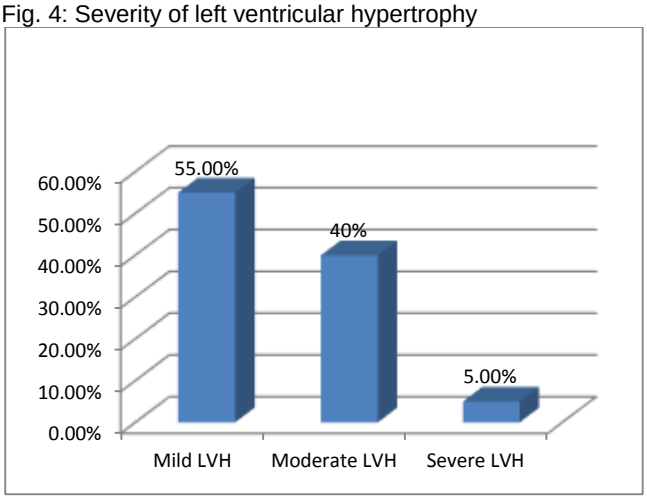


Fig. 4: Severity of left ventricular hypertrophy



DISCUSSION

LVH is present in 60 to 80%of patients with End Stage Renal Disease on renal replacement therapy[10]. 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 population[11]. Cardiovascular mortality in patients with chronic kidney disease is 10 to 20 times higher as compared to normal individuals[12].

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 disease[13].

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 was92.69+18.35 with minimum observed value of 32.00ml/min and a maximum value of 120 ml / min. The level of creatinine was normal in 47(78.33%) patients, whereas mild renal insufficiency was noted in12 (20%) patients. Moderate renal impairment was seen in only 01 (1.69%) individual. The mean LVH value was 1.307+.17 cm with minimum as 1.20 cm and maximum as 1.90 cm. Mild LVH was present in 33(53.23%) ,moderate LVH in 24(38.71%)and severe LVH seen in 3(5%) of pts.

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 shortening[14]. 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±207 µmol/L, representing a creatinine clearance (Crcl) of 25.5±17 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 50mL/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<25mL/min) had LVH (P=0.05). The mean LVMI was significantly different among the three groups (97.58g/m^2), 114.4g/m^2, respectively; P < 0.001)[16]. All reported results of both microalbuminuria and creatinine clearance was 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 failure[17].

In another study, 162 subjects with chronic kidney disease (CKD)underwent echocardiograms to see if there existed some association between let 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. Hence further detailed studies are suggested in future to establish this relationship and improve clinical outcomes for patients.

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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