Original Research Paper

Volume-9 | Issue-3 | March-2019 | PRINT ISSN - 2249-555X

Nephrology

IMPORTANCE OF LVMI AND LVH AS A CARDIOVASCULAR RISK FACTOR IN CKD PATIENTS

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ABSTRACT The objective of this study is to determine the prevalence of Left ventricular hypertrophy in patients with chronic kidney disease (CKD) and its possible association with patterns of blood pressure (BP), left ventricular mass index and mortality. Method: Two-dimensional and M-mode echocardiography was performed to determine Left ventricular mass by the method of Devereux and indexed (LVMI) to body surface area surface using Du Bois Formula on 40 patients who were on hemodialysis (HD) and 31 patients suffering from CKD. The patients were classified on the basis of having left ventricular hypertrophy (LVH) provided LVMI was ≥95 g/m2 for women or 115 g/m2 for men. Follow-up duration was calculated from the date of ambulatory blood pressure monitoring (ABPM) till the end of the study or to the date on which an outcome of death has occurred. Causes of death were classified into cardiovascular death (ischemic heart disease, cardiac arrhythmias, congestive heart failure, stroke and sudden death) and death not related to cardio vascular disease

KEYWORDS : Ambulatory BP, Left ventricular hypertrophy, cardio vascular disease, hemodialysis.

INTRODUCTION

Hypertension is prevalent in 80 to 90% of patients with chronic kidney disease[1] and is known to accelerate progression of CKD and increase the risk for cardiovascular events[2]. Despite the recommendations for strict BP control in patients with CKD only 27% of them achieve a BP goal of 140/90 mm of Hg. This is because of the volume overload as well as activation of numerous mechanisms due to the direct result of underlying kidney disease, this makes difficult to control BP with anti hypertensive drugs[3]. However, it is often ignored that hypertension may seem to be poorly controlled because of suboptimal BP assessment. Ambulatory BP measurement and self-measurements of BP allows a great number of measurements in the patients' natural environment which make these measurements more valid.

Left ventricular hypertrophy (LVH) is a well-recognized cardiac manifestation of long-term hypertension induced target organ damage[28]. In HD patients, LVH contributes substantially to high cardiovascular mortality[29]. The high prevalence of LVH among HD patients may be a consequence of inadequate diagnosis and treatment of hypertension.

The purpose of this study is to evaluate the relation between 24 hour ABPM, LVMI and cardiovascular mortality in CKD patients.

METHODOLOGY

A prospective hospital based study was carried on 40 HD patients and 31 CKD patients who have attended the hemodialysis unit and outpatient department of Osmania general hospital , Hyderabad from 2013 to 2015 .

Inclusion Criteria:

Men or women aged ≥18 years suffering from CKD of any etiology either in predialysis or on hemodialysis 2 to 3 times a week for at least 3 months and having haemoglobin ≥11 g/dl and ≤14 g/dl and single-pool Kt/V ≥1.2. Hypertension was defined as a mean of predialysis BP from three consecutive sessions ≥140 mmHg for SBP and/or ≥90 mmHg for DBP or who were using antihypertensive medication.

Exclusion Criteria:

The patients with visual or cognitive insufficiency, cardiac arrhythmias, severe heart or liver failure and pregnant women were excluded. Patients who had change in their antihypertensive medication within the last 3 months were also excluded.

ABPM Monitoring:

ABPM was performed on an interdialytic day after the midweek HD session. The 24hr Ambulatory BPs were recorded every 30 min during the day (06:00–22:00 hrs) and every 30 min during the night (22:00–06:00 hrs) using a Meditech ABPM 05 monitor. At least 70% of BP readings during daytime and night-time periods were satisfactory, or else the monitoring was repeated (according to ESH 2013). Pre-HD BP was measured after the patients had rested quietly for 15 min in the supine position, using a mercury sphygmomanometer on the upper portion of the non-fistula arm.

Table – 2: Definitions of hypertension by office and out of office blood pressure levels

Category	Systolic BP (mm Hg)		Diastolic BP (mm Hg)
Office BP	>140	and / or	>90
Ambulatory BP			
Day time (awake)	>135	and / or	>85
Night time (asleep)	>120	and / or	>70
24 h	>130	and / or	>80

Echocardiogram:

Two-dimensional and M-mode echocardiography was performed and interventricular septal thickness (IVS), left ventricular posterior wall thickness (LVPW), left ventricular end-systolic diameter (LVESD), and left ventricular end-diastolic diameter (LVEDD) were measured according to the American Society of Echocardiography guidelines. Left ventricular mass was determined from M-mode measurements by the method of Devereux and indexed to body surface area surface (Du BoisFormula):

- Left ventricular mass (grams) = 0.832 [(IVSTd+ LVIDd+ PWTd)^3−(LVIDd)^3] + 0.60, IVSTd= thickness of the interventricularseptum; LVIDd = left ventricular diastolic diameter; PWTd = posterior wall thickness of the left ventricle.
- Left ventricular mass index (g/m^2): left ventricular mass (gm)/body surface area (m^2).

The patients were classified as having left ventricular hypertrophy (LVH) if LVMI was ≥95 g/m^2 for women or 115 g/m^2 for men.

Follow-up duration was calculated from the date of ABPM till the end of study or to the date on which an outcome of death has occurred. Causes of death were classified as cardiovascular death (ischemic heart disease, cardiac arrhythmias, congestive heart failure, stroke and sudden death) and death not related to cardiovascular disease.

Statistical analysis:

The entire data has been analyzed using SPSS software 9. The univariate analysis has been expressed as mean and standard deviation.

The correlation between variables is expressed as **Pearson Correlation Coefficients Prob> |r| under H0: Rho=0.** The significance of variables between survivors and non survivors was done by two – tailed student T test; p- values less than 0.05 was considered significant.

RESULTS

40 HD patients and 31 CKD (non-HD or non-dialysis) patients formed the material of the study.
Their characteristics are shown in the following tables.

Table 1: Characteristics of Patients

	HD patients	non-HD patients
AGE (yrs)	46.05 ±12.44	42.5 ±11.7
GENDER	M 27(67.5%), F 13 (32.5%)	M 18(58.1%), F 13 (41.9%)
BMI (kg/m^2)	21.4 ±2.7	21.9 ±2.8
Mean GFR (ml /min)	7.6 ±1.4	43.9 ± 15.5
Mean OFFICE SBP mm Hg	153.9 ±17	138.7 ±16.6
Mean OFFICE DBP mm Hg	81.1 ±9.9	84.8 ±11.9
Mean 24 HR SBP mm Hg	139.9 ± 18.3	132±15.6
Mean 24HR DBP mm Hg	82.8 ± 10.05	79.8±9.24
Mean DAY SBP mm Hg	140.7 ± 18.2	134.7±14.7
Mean DAY DBP mm of Hg	83.97 ± 10.7	82.5±9.1
Mean NIGHT SBP mm of Hg	137.6 ± 19.5	126 ± 17
Mean NIGHT DBP mm of Hg	79.77 ± 10.86	73.2±11.18
Mean LVMI (g / m^2)	114.3 ± 23.2	96.48±22.6
LVH n (%)	31 (77.5%)	15 (48.4%)

The non-dialysis patients in general will have lower BP measurements and less prevalence of LVH

Table 2: Prevalence of hypertension patterns in HD and non HD patients

Patterns of hypertension	HD patients	non-HD patients
White coat hypertension	6 (15%)	4 (12.9%)
Masked hypertension	8 (20%)	6 (19.4%)
Nocturnal hypertension	32 (78%)	14 (45.2%)
Sustained hypertension	24 (60%)	10 (32.3%)

Table 3: Correlation of BP measurements with LVMI

		LVMI in HD patients	LVMI in non dialysis patients
OFFICE SBP	r value	.355*	.610
	p value	.025	.000
24 HRS SBP	r value	.602**	.733**
	p value	.000	.000
DAY SBP	r value	.549**	.697**
	p value	.000	.000
NIGHT SBP	r value	.647	.680**
	p value	.000	.000
OFFICE DBP	r value	.278	.465**
	p value	.082	.008
24 HRS DBP	r value	.367*	.579**
	p value	.020	.001
DAY DBP	r value	.265	.531**
	p value	.098	.002
NGHT DBP	r value	.524**	.595**
	p value	.0005	.0000

Multivariate analysis was performed between left ventricular mass index and blood pressures, it was observed that:

- There was a significant positive correlation between SBP, office and ambulatory BP and LVMI both in hemodialysis and non dialysis patients.
- There was a significant positive correlation between DBP, office and ambulatory BP and LVMI both in hemodialysis and non dialysis patients.

Thus though both DBP and SBP showed correlation, SBP showed a better correlation. Night BP readings showed the best correlation with CV markers.

Table 4: Comparison between prevalence of LVH in patients with different patterns of hypertension

LVH	White Coat HTN present		Controlled HTN	
	No	%	No	%
Present	2	20	1	7.7
Absent	8	80	12	92.3
Total	10	100	13	100
Chi square	0.75	p value	.384	

Table 5: Comparison between prevalence of LVH in patients with Masked HTN

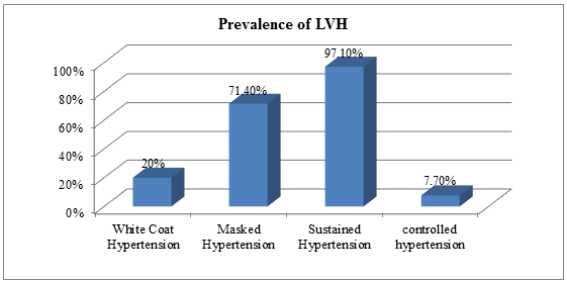
LVH	Masked HTN present		Controlled HTN	
	No	**%**	**No**	**%**
Present	10	71.4	1	7.7
Absent	4	28.6	12	92.3
Total	14	100	13	100
Chi square	11.3	p value		0.0007

Table 6: Comparison between presence of LVH in patients with Sustained HTN

LVH	Sustained HTN present		Controlled HTN	
	No	%	No	%
Present	33	97.1	1	7.7
Absent	1	2.9	12	92.3
Total	34	100	13	100
Chi square	37.56	p value		<0.05

Prevalence of LVH in patients with whitecoat hypertension was higher than controlled Hyper tension (HTN) though the numbers did not achieve statistical significance. There was a increased incidence of LVH in patients with masked HTN, and the increase was statistically significant. In the present study LVH was significantly higher in patients who had sustained hypertension p <0.05.

Figure 1: Prevalence of LVH in different patterns of Hypertension



Outcomes and mortality:

Follow up: The mean duration of follow up of the entire cohort was 18.9+5.18 months. During this time 14 patients on dialysis and 2 patients on conservative therapy died. Of them 10 (63%) patients died due to CVS disease (first most common cause). Of them 4 (25%) patients died due to sepsis (second most common cause).

Table 7: Incidence of adverse events and deaths

Adverse events	Numbers	Percentage
Acute coronary syndrome	4	5.6%
Cardiac failure	11	15.4%
Cerebrovascular accident	3	4.2%
Total deaths	16	23%
Cause of death		
Sudden cardiac death	3	19%
MI	3	19%
Cerebrovascular accident	2	12.5%
Cardiac failure	2	12.5%
All CVS deaths	10	63%
sepsis	4	25%
Upper GI bleed	1	6%
Accident	1	6%

Predictors of survival:

Table 8: Comparison of variables between survivors and non - survivors

Parameters	Non survivors 16		Survivors 55		t value	Sig. (2-tailed)
	Mean	Std. Deviation	Mean	Std. Deviation		
Age (yrs)	45.25	14.6	44.34	11.48	-.26	.79
eGFR(ml/min)	9.6	6.9	27.4	21.8	-3.22	0.001
OFFICE SBP	150.5	17.26	146.33	18.7	.795	.42
OFFICE DBP	90.75	7.19	84.8	11.4	1.97	.052
24 HR SBP	144.06	15.99	134.27	17.53	2.0023	.049
DAY SBP	143.5	14.89	136.56	17.33	1.45	.15
NIGHT SBP	143.44	18.7	129.4	18.59	2.65	.009
24 hrs DBP	85.5	8.43	80.36	9.88	1.88	.06
DAY DBP	85.75	8.54	82.64	10.36	1.09	.27
NIGHT DBP	83.25	9.75	75.05	11.27	2.633	.015
LVMI	125.7	23.13	100.95	22.11	3.90	0.0002

On comparing patients who survived with died. The 24 hr SBP, NIGHT SBP, Night DBP and LVMI were higher in patients who died. In this study Ambulatory BP monitoring proved to be a better predictor of all cause mortality than office BP. Night BP is a better predictor of all cause mortality than Day BP readings.

DISCUSSIONS

Correlation between BP measurements and LVMI:

Table 9: Association of BP with LVMI in hemodialysis

Study	S. Ertiirk et al[6] N=40	Present study
Predialysis SBP	R = .3961	R = .355
	P = .011 S	P =.025
Pre dialysis DBP	R = .3213	R= .278
	P= .043 S	P= 0.82NS
24 Hr SBP	R = .5440	R = .602
	P=.0002 S	P= 0.000
24Hr DBP	R=.4399	R = .367
	P=.004 S	P = .020
Day SBP	R=.5119	R = .549
	P=.0007S	P = .000
Day DBP	R=.4099	R=.265
	P=.0086S	P=.09
Night SBP	R= .5746	R= .647
	P=.0001	P= .000
Night DBP	R=.5111	R =.524
	P=.0007S	P=.0005

*S: significant, NS: non-significant

Optimal BP control is a main objective in treatment to improve renal and cardiovascular prognosis in CKD patients. However, the achievement of SBP target recommended by International Guidelines (<140 mmHg) remains largely inadequate.

The mean age in HD patients group was 46 years $\pm$ 12.4 years comparable to Robert Ekart[6] and Jacques Amar[7]. The mean age in ND group was 42 years $\pm$ 11.7 years comparable to R Agarwal[8] and Roberto Minutolo[9].Males account for 67.5% in hemodialysis group and 58.1% in non hemodialysis group.

The prevalence of white coat hypertension in HD patients was found to be 15% which was same when compared with R Agarwal[10] study i.e. 15%. The prevalence of white coat hypertension in non-HD patients was found to be 12.9% which was similar when compared with Kuriyama[11] study i.e. 12.5%. The prevalence of masked hypertension in HD patients was found to be 20% which was similar when compared with R Agarwal[10] study i.e. 15%. The prevalence of masked hypertension in non-HD patients was found to be 19.4% which was lower when compared with Kuriyama[11] study i.e. 31%.

Table 10: Correlation between BP measurements and LVMI in non-dialysis patients

Study	B. Tucker et al[12]	Present study
clinic SBP	R = .25	R= .610
	P = .03 S	P =.000
clinic DBP	R = .22	R= .465
	P= .03S	P = .008
24 Hr SBP	R = .52	R = .733
	P=.0001 S	P = .000
24Hr DBP	R=.42	R = .579
	P=.0001	P = .001
Night SBP	R= .64	R= .680
	P=.0001	P = .000
Night DBP	R=.55	R = .565
	P=.0002	P=.000

ABPM shows a significant correlation with LVMI in most of the clinical studies with the coefficient of correlation being greater for systolic BP than for diastolic BP. Based upon data from meta-analysis of 19 studies performed by Fagard et al[13], the strength of the correlation between LVMI and ABPM is greater than for office or clinic BP as seen in this study. The present study shows that the night time blood pressure correlates better with target organ damage in CKD patients similar to Wang C et al[14].

Prevalence of LVH:

In this study LVH was present in 48.4% of pre dialysis population and 77.5% of dialysis population. LVH was significantly high in patients on dialysis than predialysis population with increased prevalence of LVH with increased stage of chronic disease observed in studies by Levin et al[15] i.e. 38.9% and lower when compared with P Dangri[16] i.e. 68.3%. LVH in dialysis population was higher than as observed in studies by Rajaram Barde[17] i.e. 57%.

Prevalence of LVH in different BP patterns in CKD:

The prevalence of LVH in masked hypertension was 71.4% and in sustained hypertension 97.1% which was significantly higher than in normotensive patients. Thus in the present study it can be concluded that the target organ damage in masked hypertension is similar to sustained hypertension. So the present study signifies the importance of ambulatory BP monitoring in CKD patients.

Target organ damage in masked hypertension:

Several studies demonstrated more target organ damage in treated patients with masked hypertension than in those whose blood pressure is controlled. Among hypertensive diabetics with mild to moderate kidney disease, those with masked hypertension had higher LVMI (138±15 g/m^2) than those with well-controlled hypertension (105±8 g/m^2)[11]. Pierdomenico[18] found that treated patients with masked hypertension were almost twice as likely to have LVH and had doubled the cardiac event rate, compared with the well-controlled group. Tomiyama[19] reported that masked hypertension was an independent determinant of LVH. Finally, in a recent analysis of 617 patients in the African American Study of Kidney Disease (AASK) trial, Pogue[20] reported higher left ventricular mass and prevalence of LVH among masked hypertensives compared with true normotensives.

Target organ damage in white coat hypertension:

In the present study prevalence of LVH in white coat hypertension was found to be 20% when compared to 7.7% in patients with controlled hypertension, the difference in the two groups was not statistically significant. However the issue of cardiovascular risk in subjects with untreated white-coat hypertension is still controversial. The major finding of the 2012 IDACO population study with a mean follow-up time of 10.6 years was that the sex and age-standardized, Incidence rate of cardiovascular events in 334 participants with untreated white-coat hypertension was no greater than in the untreated normotensive control population[21]. Similarly, the 2-year mortality in white-coat hypertensive patients with end-stage renal disease requiring hemodialysis was less than in masked and sustained hypertensives, but found to be somewhat higher than in sustained normotension patients as diagnosed by ABPM in a study done by Agarwal et al[10].

All cause mortality:

The mean duration of follow up was 18.9 months. Cardio vascular disease was the leading cause of death in patients with advanced CKD. The present study had a mortality of 23% in the entire cohort. 14 (35%) patients on hemodialysis and 2 (6.1%) patients on conservative therapy died. The most common cause of death was cardiovascular disease 63% (10) followed by sepsis 25% (4).

Liu et al[21] documented a mortality of 39% in 80 dialysis patients after 4 years follow up. Agarwal et al[8] documented a mortality of 31% over a

median follow up of 32 months.

In India Prakash et al[22] observed mortality of 24.3% in 41 CKD patients; 16% in patients on conservative therapy and 35% in patients on hemodialysis. Chandrasekharan et al[23] observed a mortality of 19.8% in 96 patients after 2 years. The two most common causes were CVS deaths and sepsis as observed in the present study.

Association of all cause mortality with office Vs ABPM measurements:

In the present study on comparison of patients who survived and died ; 24 hr SBP,NIGHT SBP, Night DBP,24Hr MAP and LVMI was higher in patients who died ($p < 0.05$). On the other hand Office BP was not a good predictor of mortality. This study signifies the importance of ambulatory BP recordings in CKD patients and ABP is independently associated with all cause mortality. The results of this study are consistent with studies done by Robert Ekart etal[6], Liuet al[21], Amaret al[24], Prakash et al[25]and Tripepi et al[26].

CONCLUSIONS

It can be concluded from the above study that 24 hr ambulatory BP is useful in CKD patients:

1. To diagnose masked hypertension. White coat hypertension patients should not be over treated.
2. It was found to be a better predictor of CVS risk factors and target organ damage than conventional BP.
3. ABPM and LVMI were found to be better predictors of all cause mortality than conventional BP.

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