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Original Research Article

A study on the left ventricular hypertrophy among the patients of chronic kidney disease stage third to five

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ABSTRACT

Background: In Chronic kidney Disease (CKD) a significant risk factor for mortality is Cardiovascular disease (CVD) and the most prevalent cardiovascular risk factor is left ventricular hypertrophy (LVH). Anemia, hypertension and volume overload are risk factors for LVH in CKD. So, the present was aimed at comparing the risk factors between CKD with and without LVH.
Methods: A cross sectional study carried out over a 2 year period in Department Nephrology and General Medicine OPD, MIMS, Vizianagaram, Andhra Pradesh. A total of 120 patients are included in this study and divided in to CKD stage III to V based on estimated GFR. Based on 2D echocardiography data CKD cases are further divided in to CKD with LVH and CKD without LVH.
Results: The Left ventricular mass index was significant higher in CKD with LVH (128.89±19.28) when compared with CKD without LVH (108.20±10.28). The left ventricular mass index was noted in more number in stage V of CKD. It is also observed that the left ventricular mass index was negatively correlated with haemoglobin and eGFR and was positively correlated with systolic blood pressure and serum NT-proBNP.
Conclusions: Present study finding suggested that the incidence of LVH is higher in CKD patients. LVH was positively correlated with hypertension and NT-proBNP and negatively correlated with anemia and estimated GFR.

Keywords: Chronic kidney disease, Cardio vascular disease, Left ventricular hypertrophy, 2D echocardiography

INTRODUCTION

Chronic kidney disease (CKD), is a major chronic disease increasing globally.[1] Cardiovascular disease (CVD) is a significant risk factor for mortality and morbidity in CKD. Cardiovascular risk in CKD leads to premature death.[2] In CKD cardiovascular disease abnormalities are due to volume overload, hypertension, endothelial dysfunction, inflammation, uremic pericarditis, cardiomyopathy, anemia, dyslipidemia and oxidative stress.[3] Besides, these traditional risk factors C-reactive protein (CRP), asymmetric dimethyl arginine (ADMA) and Carotid intima media thickness (CIMT) abnormalities were also identified in CKD.[4] Cardio vascular disease related mortality is 15 to 30 fold higher in early stages of CKD in end stage kidney disease (ESKD) it is more than 500 times higher than healthy individuals.[5]

In CKD the most prevalent cardiovascular risk factor is left ventricular hypertrophy (LVH) and it can contribute arrhythmias, heart failure and sudden death. All these factors will have negative prognostic value.[6] CKD is generally associated with anemia, which leads to poor oxygen delivery and causes elevated heart rate and stroke volume and finally leads to LVH.[7]

The prevalence of LVH increases with decreased renal function. Previous studies shown that incidence of LVH in CKD stage 3 to 5 is 71% and stage 5 it is 96%.[8] LVH can be measured by using electrocardiography and echocardiography.[9] LVH can be assessed by estimating NT-pro BNP (N-terminal pro Brain Natriuretic Peptide). It is synthesised from its precursor Brain natriuretic peptide.[10] NT-pro BNP will be elevated in response to raised intravascular volume and promotes diuresis to maintain blood volume. In CKD, decreased renal function more NT-pro BNP is synthesised to achieve level of activity in the healthy kidney.[11]

LVH is a major predictor of cardio vascular mortality in CKD early prevention of LVH in CKD prevents deleterious events.[12] There is increased attention towards Left ventricular hypertrophy in CKD patients early measures can prevent its progression. This study will help to determine the incidence of LVH in CKD and highlight the risk in these patients.

METHODS

The present study is a cross sectional study carried out over a 2 year period that is from January 2017 to January 2019 in Department of Nephrology and General Medicine OPD, MIMS, Vizianagaram, Andhra Pradesh. Those who were diagnosed as CKD and attending nephrology unit over a one year period were taken as subjects for the present study. A total of 120 patients from stage III to V were included in this study. Patients attending nephrology unit other than CKD, Patients with any debilitating illness and CKD patients who did not provide inform constant were excluded. To all Participants the importance of the study and procedure to be performed were informed. Informed consent was obtained from all the participants. A questionnaire was given to all patients and detailed clinical examination was performed.

In all the participants blood pressure measured by using mercury sphygmomanometer both systolic and diastolic blood pressure was measured based on 1st and 5th korotkoff phase. An average of two readings was considered.

All the participants are subjected 12 lead electrocardiography and recorded at paper speed 25mm/s and 1-mV/cm calibration. LVH defined based on Sokolow-lyon voltage criteria the amplitude of the S wave in lead V1 and the R wave in lead V5 or V6 is more than 3.5mV was considered as LVH.[13] LVH was further confirmed by using 2D echocardiography the data was recorded based on American Society of echocardiography guidelines. LVH was confirmed based on Framingham criteria were LV mass index greater than 131g/m^2 in male and more than 100g/m^2 in female. Interventricular septum wall thickness or the posterior wall if >1.2cm was considered as LVH.[14]

In all these participants, blood urea was estimated by GLDH-urease method.[15] Serum creatinine was estimated by Jaffes method.[16] The NT-pro BNP was estimated using standard ELISA kit of SunRed Laboratories. Based on serum creatinine, estimated GFR (eGFR) was computed by the Modification of Diet in Renal Disease (MDRD).[17] The haemoglobin was measured by using Sysmex KX-21 analyzer.

All the Data was expressed in Mean and Standard deviation (mean ±SD). Statistical significance between control and cases groups Z test was performed using Microsoft Excel and SPSS software 16.0. The statistical significance was determined at 5% ($p<0.05$) level. Spearmen correlation conducted between Left ventricular mass index with hemoglobin, eGFR, systolic blood pressure and serum NT-pro BNP.

RESULTS

The present study was conducted at Maharajah's Institute of medical sciences, Vizianagaram, Andhra Pradesh, India. A total of 120 CKD patients were included. The CKD cases were further divided into 2 groups that are CKD with LVH (72) and CKD without LVH (48).

Table 1: Profile CKD with LVH and CKD without LVH.

	CKD with LVH	CKD without LVH
Number	72	48
Age (mean±SD) years	43.27±10.09	44.8±10.78
Sex		
Males %	60	63
Females%	40	47
Blood urea (mg/dl)	88.22±22.90	76.72±18.01**
Serum creatinine (mg/dl)	6.82±2.12	5.21±2.02**
eGFR (mL/min)	28.67±9.89	48.57±8.73**
Blood pressure (mm Hg)		
Systolic	153.75±21.97	130.28±19.28**
Diastolic	93.12±7.83	80.21±8.83**
Hemoglobin (g/dl)	8.52±0.78	9.86±0.93**
NT-pro BNP (pg/ml)	392.52±71.22	210.67±32.50**
Left ventricular mass index (g/m2)	128.89±19.28	108.20±10.28**
Stages of CKD		
Stage III	2	18
Stage IV	28	21
Stage V	42	9

Table 1 shows the mean age of the CKD with LVH was 43.27years±10.09 CKD without LVH it was 44.8years±10.78. As regards the sex distribution, the majority of subjects were male in CKD with LVH 60%

and CKD without LVH 63%. The diagnostic criteria for CKD like blood urea, serum creatinine and eGFR were significantly higher in CKD with LVH when compared to CKD without LVH. In the present study systolic and diastolic blood pressure was significantly increased in CKD with LVH compared with CKD without LVH ($p<0.001$). There is also significantly increased levels of serum NT-proBNP in CKD with LVH when compared with CKD without LVH. The Left ventricular mass index was significant higher in CKD with LVH (128.89±19.28) when compared with CKD without LVH (108.20±10.28). The left ventricular mass index was noted in more number in stage V of CKD.

Table 2: Correlation of left ventricular mass index with hemoglobin, eGFR, systolic blood pressure and serum NT-pro BNP.

Parameters	Left ventricular mass index, r value	p value
Hemoglobin	r= -0.8234	p<0.001
eGFR	r= -0.6808	p<0.001
Systolic blood pressure	r=0.7896	p<0.001
Serum NT-pro BNP	r=0.8071	p<0.001

Table 2 shows the left ventricular mass index was negatively correlated with haemoglobin and eGFR and it is statistically significant ($p<0.001$). It is also observed that left ventricular mass index was positively correlated with systolic blood pressure and serum NT-proBNP and it is statistically significant ($p<0.001$).

DISCUSSION

The causes for raised serum creatinine and blood urea were due to decreased glomerular filtration and there is inverse relation between eGFR and LVH. The increased blood pressure in CKD with LVH is due to hypervolemia and the uncontrolled hypertension. Anemia is the independent risk factor for LVH. In anemia there is poor oxygen delivery which causes increased stroke volume and that causes LVH.

NT-proBNP was introduced as marker for heart failure but it is also marker for acute coronary syndromes and mortality in myocardial infarction.[18] The main reason for markedly raised NT-pro-BNP due to left ventricular structural and functional abnormalities.[19] The pro brain Natriuretic Peptide (BNP) synthesized and released from left ventricles due to myocardial stretch.[20] Once it entered in to blood circulation it converted into C-terminal side peptide and N-terminal pro BNP (NT-pro BNP). These peptides act on renal system and causes tubular natriuresis and diuresis. These peptides act against the renin-angiotensin-aldosterone system (RAAS).[21] If intravascular volume is increased due to renal or cardiac dysfunction the BNP peptides causes diuresis and maintain normal blood volume. In Kidney failure, high BNP is synthesized to reach the level of activity in the normal healthy kidney. CKD combined with cardiac dysfunction causes elevated secretion of NT proBNP. Earlier studies shown that increased NT-proBNP with decreased renal function. This is due to decreased clearance of NT-proBNP with progression of CKD.[22] Previous studies also shown that markedly elevated levels of NT-pro BNP in stage V cases and concluded that systolic dysfunction and LVH contributed for raised NT-pro BNP.[23]

In the present study 60% of CKD patients showed Left ventricular hypertrophy. In advanced stages of CKD LVH is still higher due to increased complication of anemia, hypertension, fluid overload and arterial stiffing. Levin studies demonstrated that LVH increased with progressive decline of renal function.[24] Yilmaz et al, studies revealed that LVH was reported in 67.6% of CKD stage III and stage IV.[25] A study done in Nigeria shown 95.5% of CKD stage IV and stage V patients show LVH.[26] Similarly a Brazil study also demonstrated 60% of stage V CKD shown LVH.[27]

From the findings of present study, it was concluded that there is a high prevalence of LVH in CKD patients. Low hemoglobin, decline in eGFR, increased systolic blood pressure, high NT-proBNP were found to be significantly associated with LVH in CKD patients. Early detection of LVH and correction of hypertension and anemia assist in the reducing progression deleterious effects.

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