

# LEFT VENTRICULAR HYPERTROPHY IN CHRONIC KIDNEY DISEASE IN SULAIMANI CITY

Dana Ahmed Sharif <sup>a</sup>, Salar Mohammed Amin <sup>b</sup>,  
and Deler Shamsulddin Hamid <sup>a</sup>



Submitted: 24/3/2019; Accepted: 3/5/2019; Published: 21/6/2019

## ABSTRACT

### *Background*

Left ventricular hypertrophy (LVH) is an independent predictor for cardiovascular events in chronic kidney disease (CKD).

### *Objectives*

To find relationship between left ventricular hypertrophy and CKD stages during predialysis period and assess risk factors in CKD patients for left ventricular hypertrophy.

### *Methods*

This cross-sectional study of 125 participant with CKD was conducted at Shar Teaching Hospital of Nephrology department, Sulaimani, Iraq. Left ventricular mass index (LVMI) was measured by using two-dimensional echocardiogram in the left decubitus position. LVH was labeled when the left ventricular mass index was  $>115$  g/m<sup>2</sup> in men, and  $>95$  g/m<sup>2</sup> in women on echocardiogram. We analyzed the baseline characteristic in 125 patients with chronic kidney disease. All patients underwent laboratory investigations which included serum creatinine, complete blood count, serum calcium, phosphate, intact parathyroid hormone, high density lipoprotein cholesterol HDL-C, low density lipoprotein LDL-C, triglyceride, and total cholesterol with urine albumin to creatinine ratio.

### *Results*

The mean age was  $55.27 \pm 14.51$  years (male 60.0%, female 40.0%), and the prevalence of LVH was 68.0%, and it was increased with progressive decline in estimated glomerular filtration rate (eGFR) ( $P = 0.005$ ). independent risk factors for LVH were anemia ( $P = 0.000$ ), systolic and diastolic BP ( $P = 0.000$ ;  $P = 0.001$  respectively), intact PTH ( $P = 0.038$ ), body mass index BMI ( $P = 0.045$ ), serum calcium ( $P = 0.003$ ), serum phosphate ( $P = 0.001$ ) and majority of lipid profiles.

### *Conclusion*

There was a high prevalence of LVH in the CKD patients and it was increased with progressively decline in renal function. There was a significant association between systolic and diastolic BP, intact PTH, hemoglobin level, BMI, and minerals with LVH in CKD patients.

**Keywords:** *Chronic Kidney Disease, Left Ventricular Hypertrophy.*

---

<sup>a</sup> Department of Medicine , College of Medicine, University of Sulaimani, Kurdistan Region, Iraq.

<sup>b</sup> Kurdistan Board Candidate

Correspondence: [salar.mameen@yahoo.com](mailto:salar.mameen@yahoo.com)

## INTRODUCTION

Chronic Kidney Disease (CKD) is a worldwide public health problem with adverse outcomes of kidney failure, cardiovascular disease (CVD) and premature death<sup>(1)</sup>. CKD patients are at significantly increased risk for both morbidity and mortality from cardiovascular disease and has been attributed to various factors, such as the deleterious effects of retained uremic toxins, chronic micro-inflammation, increased sympathetic activity, premature atherosclerosis, fluid overload and hypertension<sup>(2)</sup>.

CVD risk factors such as, diabetes mellitus, smoking, hypertension and dyslipidemia are more common in individuals with CKD than those with normal renal function<sup>(3)</sup>. Left ventricular hypertrophy (LVH) is an independent risk factor for an adverse clinical outcome, both in the general population and in chronic kidney disease patients<sup>(4)</sup>. Most evidence suggests that LVH does not regress, but aggravates in this patient group over time<sup>(5)</sup>. From a hemodynamic view, LVH is an adaptive remodeling process that compensates for the increase in cardiac workload induced by an increased afterload (pressure overload), an increased preload (volume overload) or both<sup>(6)</sup>.

A high prevalence of LVH in patients with CKD is a consistent finding, and the relationship between increases in left ventricular mass and a subsequent increase in cardiovascular events indicate the potential value of LVH as a valid surrogate marker in CKD patients<sup>(7,8)</sup>.

Interestingly, recent studies have suggested that microvascular changes affect myocardial micro vessel structure, leading to abnormalities in the coronary microvascular response and the myocardium in patients with hypertension and LVH<sup>(9)</sup>. Echocardiography is a simple and non-invasive assessment of left ventricular structure and function and also gives information on both left ventricular contractility and left ventricular geometry. However, there are few data about pattern and prevalence of left ventricular hypertrophy in different stages of predialysis chronic kidney disease<sup>(9)</sup>.

In this study we evaluated the pattern and prevalence of left ventricular hypertrophy by left ventricular geometry through echocardiography in patients with chronic kidney disease and provide the relationship between LVH and various predisposing factors such as blood pressure, renal function, anemia, proteinuria and secondary hyperparathyroidism.

## PATIENTS AND METHODS

One hundred and twenty five Iraqi Kurdish speaking patients were enrolled from June 2018 to February 2019, all of them with chronic kidney disease (CKD), according to the National Kidney Foundation-Kidney Disease Outcomes Quality initiative (K/KOQI) guidelines<sup>(10)</sup>.

Patients were recruited from nephrology department at Shar Teaching Hospital and all participants gave their written informed consent. Participants before their inclusion in the study, estimated glomerular filtration rate (eGFR) was measured and diagnosed as chronic kidney disease (CKD) based on Modification of Diet in Renal Disease Formula<sup>(11)</sup>.

$[eGFR \text{ in mL/min per } 1.73 \text{ m}^2 = 175 \times \text{Serum Cr}^{-1.154} \times \text{age}^{-0.203} \times 1.212 \text{ (if patient is black)} \times 0.742 \text{ (if female)}]$ .

They were selected to participate in this study if they agreed to receive Echocardiographic examination. Demographic and medical data including age, sex, smoking history (ever versus never), weight, height were obtained from patients interviews or medical records. Exclusion criteria for all participants included kidney transplantation, kidney donors, patients were on hemodialysis or peritoneal dialysis, patients with human immunodeficiency virus (HIV) infection, liver cirrhosis, active cancer, age below 18 year and patients who refused to provide informed consent.

All participants underwent clinical investigation, laboratory testing and transthoracic echocardiography (TTE). Fasting blood samples were obtained under sterile and full aseptic technique from upper limb peripheral veins for hemoglobin by Medonic M-series M32 machine, intact parathyroid hormone (PTH) by Cobas e 411 analyzer machine, serum creatinine, calcium, phosphorus, total cholesterol, low density lipoprotein cholesterol (LDL cholesterol), high density lipoprotein cholesterol (HDL cholesterol) and triglyceride by Cobas c311 analyzer machine. Random urine samples were obtained and albumin to creatinine ratio (ACR) strip and laboratory quantitative tests were completed within 2 hr of urine collection. Urine ACR was calculated (expressed as milligrams of albumin per gram of creatinine). Microalbuminuria was defined as an ACR 30 to 300 mg/g, and macroalbuminuria (overt proteinuria) as ACR 300 mg/g or above<sup>(12)</sup>.

## Left Ventricular Hypertrophy in Chronic Kidney Disease in Sulaimani City

Blood pressure was measured in the sitting position with back support after 5 minutes of rest by mercury sphygmomanometers in Shar Teaching Hospital / Nephrology Department as outpatient. The mean of three blood pressure determinations obtained on the day of echocardiogram was reported. Hypertension was defined as systolic and diastolic blood pressure  $\geq 130/80$  mmHg according American college of cardiology (2017 Guideline).

### ECHOCARDIOGRAPHY

All patients underwent two-dimensional targeted M-mode echocardiograms recorded in the left lateral decubitus position by an experienced cardiologist using a GE VIVID E9 (Canadian ICES-001, GE Vingmed Ultrasound AS, N-3191 Harten, Norway). The cardiologist was blinded to the other data. The standard echocardiographic measurements from parasternal long axis view included the left ventricular internal diameter in diastole (LVIDd), left ventricular posterior wall thickness in diastole (LVPWTd) and interventricular septal wall thickness in diastole (SWTd). Left ventricular ejection fraction (LVEF) was calculated from M-mode recordings using the Teichholz method and impaired left ventricular systolic function was defined as left ventricular ejection fraction  $< 55\%$  according American Society of Echocardiography (ASE) guidelines<sup>(13)</sup>.

$$\text{LVEF}(\%) = \frac{(\text{LVIDD})^2 - (\text{LVIDS})^2}{(\text{LVIDD})^2} \times 100.$$

Left ventricular mass (LV mass) was calculated using linear measurements derived from 2D targeted M-mode or, more recently, from 2D linear LV measurements. (ie, LV mass =  $0.8 \times \{1.04[(\text{LVIDd} \times \text{PWTd} \times \text{SWTd})^3 - (\text{LVIDd})^3]\} - 0.6 \text{ g}$ ) by ASE.

Left ventricular mass index (LVMI) was calculated by dividing the left ventricular mass by body surface area (BSA);  $\text{LVMI} = \text{LV mass (gm)} / \text{BSA (m}^2\text{)}$ .  $\text{BSA} = 0.007184 \times \text{W}^{0.425} \times \text{H}^{0.725}$  (where W is weight in kg and H is height in cm). Left ventricular hypertrophy (LVH) was defined as LVMI  $> 95 \text{ gm/m}^2$  for female and  $> 115 \text{ gm/m}^2$  for male, according to ASE recommendation.

Further characterization of LVH into eccentric and concentric hypertrophy was dependent on measurements of relative wall thickness (RWT) according to ASE criteria.  $\text{RWT} = [(2 \times \text{PWT}) / \text{LVIDD}]$ . In the presence of LVH Concentric hypertrophy was defined if the RWT  $\geq 0.45$ , and eccentric hypertrophy

was defined if the RWT  $< 0.45$ . In the absence of LVH, concentric remodeling was present of the RWT  $\geq 0.45$ , and normal geometry was defined if the RWT  $< 0.45$ .

### STATISTICAL ANALYSIS

Data were entered Microsoft Excel and coding, then for statistical analysis SPSS program (version 22) were used:

Descriptive approaches were used for calculation of frequencies, percentages and means.

Analytic approaches were used to find associations using, Chi square test, t- test, ANOVA test and Fisher's exact test.

P- Value of  $\leq 0.05$  regarded as statistical significant, while P- Value  $< 0.001$  regarded as statistically highly significant.

### RESULTS

We studied 125 participants. Table 1 describes population studied. Age, gender, smoking history, primary renal diseases and estimated GFR are presented. Patients were between 18 – 81 years old, The mean age of study participants was  $55.27 \pm 14.51$  year , 5 (60.0%) were male and 50 (40.0%) were female, and 24 (19.2%) were smoker. Underlying causes of renal insufficiency were diabetic nephropathy 60 (48%), hypertension 35 (28%), glomerular disease 18 (14.4%), obstructive uropathy 3 (2.4%), adult polycystic kidney disease 4 (3.2%), unknown 5 (4.0%).

The mean body mass index (BMI) was  $26.76 \pm 3.46$ , and mean estimated GFR was  $30.41 \pm 13.31$ , ranged from 10 to 74 ml/min per  $1.73 \text{ m}^2$ .

CKD was stage 2 in 4 patients (3.2%), stage 3 in 56 patients (44.8%), stage 4 in 54 patients (43.2%), and stage 5 in 11 patients (8.8%) (Fig 1; Table 2).

Patients with stage 4 – 5 CKD were older, had higher systolic BP, a higher prevalence of anemia, and higher level of Intact PTH than patients with stage 2 – 3 CKD (P = 0.000) but diastolic BP was not significant different between the CKD stages, and serum level of phosphate, and serum low density lipoprotein cholesterol LDL-C and triglyceride TG in stage 4 and 5 CKD patients were significant higher than those in stage 2 and 3 CKD patients (P = 0.000) . But the serum level of high density lipoprotein cholesterol HDL-C and total calcium were significant higher than in the early stages compared to the late stages (P = 0.000) as shown in table 2,3 . With

progressive decline in renal function the LV systolic dysfunction was increased (P = 0.002) (Table 3).

The prevalence of LVH was 85 of 125 (68.0%) in the study predialysis CKD patients (Table 4). The prevalence of LVH in the stage 2, stage 3, stage 4, and stage 5 was 25.0%, 55.4%, 81.5%, and 81.8% respectively (P = 0.005; Fig. 1; Table 5). Those CKD Patients who are older and had higher BMI are more likely to have LVH than the other (P = 0.045) (Table 4). eGFR, hemoglobin, and level of intact PTH were significantly different in those patients with LVH compared to those without LVH (table 5), and there was significant correlation between anemia and LVH (P = 0.000). In the present study, hyperparathyroidism was more prevalent in patients with LVH (P = 0.038; Table 5). Urine ACR was not significant related to LVH in CKD patients (P = 0.117; Table 5).

As shown in Table 6, variables independently associated with LVH in CKD patients were systolic BP (P=0.000), diastolic BP (P = 0.001), serum HDL-C (P = 0.000), serum LDL-C (P = 0.002), serum TG (P = 0.002), serum total cholesterol (P = 0.008), serum calcium (P = 0.003), and serum phosphate (P = 0.001).

In the study of predialysis CKD patients, and among those with LVH the prevalence of concentric LVH was 64 in 85 (75.3%) and eccentric LVH was 21 in 85 (24.7%) (Fig. 2; Table 7) the prevalence of concentric was higher than eccentric LVH with more advance in CKD (Table 7), and among those CKD patients, the Systolic BP and intact PTH level in concentric LVH were higher in concentric LVH than in eccentric LVH (P = 0.017, P = 0.001 respectively; Table 8).

**Table 1. Demographics of the study Population.**

<b>Age , year (mean± SD)</b>	55.27±14.51
<b>Range</b>	18- 81
<b>Smoking , number (%)</b>	
<b>yes /</b>	24 (19.2)
<b>No/</b>	101 (80.0)
<b>Gender, number (%)</b>	
<b>Male</b>	75 (60.0)
<b>Female</b>	50 (40.0)
<b>Primary renal disease, number (%)</b>	
<b>Diabetic nephropathy</b>	60 (48.0)
<b>Hypertension</b>	35 (28.0)
<b>Glomerular disease</b>	18 (14.4)
<b>Obstructive uropathy</b>	3 (2.4)
<b>Adult polycystic kidney disease</b>	4 (3.2)
<b>Unknown</b>	5 (4.0)
<b>eGFR ml/min per 1.73 m<sup>2</sup></b>	
<b>(mean ± SD)</b>	30.41±13.31
<b>Range</b>	10 - 74
<b>BMI (body mass index) kg/m<sup>2</sup> (mean ± SD)</b>	26.76±3.46

*Left Ventricular Hypertrophy in Chronic Kidney Disease in Sulaimani City*

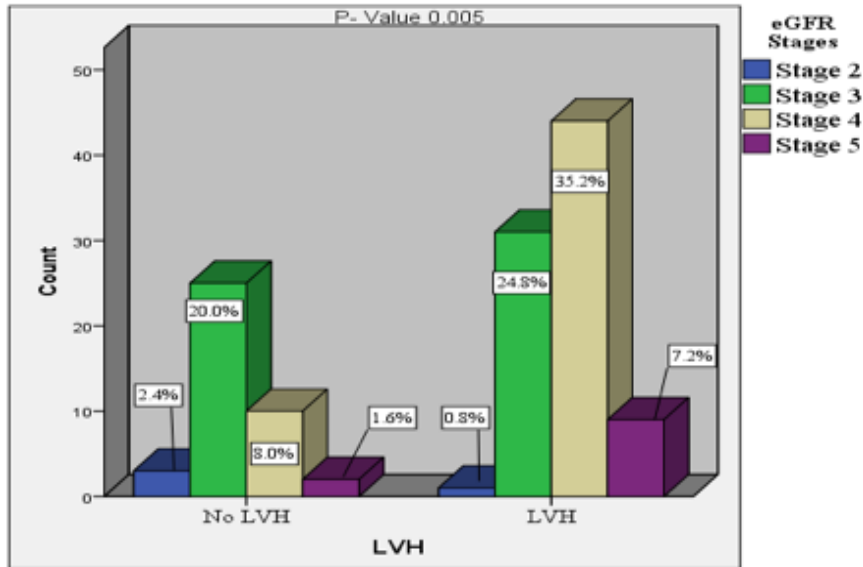


Figure 1. Categories of left ventricular hypertrophy by stages of chronic kidney disease. The prevalence of left ventricular hypertrophy is higher with more decline estimated glomerular filtration rate eGFR.

Table 2. Severity of Renal Dysfunction and Mean Values of Clinical, and Laboratory Variables.

Variables (mean ±SD)	Stages of CKD				Total	P- Value
	Stage 2	Stage 3	Stage 4	Stage 5		
<b>Age/ year</b>	4	56	54	11		
<b>N</b>	24.00	53.75	58.30	59.55	55.27	0.000
	3.916	15.159	11.908	10.976	14.519	
<b>Weight (kg)</b>	70.75	75.46	73.83	71.09	74.22	0.463
	11.177	9.437	10.235	9.534	9.831	
<b>Height (cm)</b>	171.25	167.45	164.41	164.18	165.97	0.111
	11.147	7.818	8.067	8.46	8.181	
<b>SBP (mmHg)</b>	144.00	151.48	165.83	173.00	159.34	0.000
	13.711	15.917	20.757	17.944	19.936	
<b>DBP (mmHg)</b>	86.0	89.41	84.7	87.08	87.06	0.099
	9.201	10.495	8.927	10.296	9.923	
<b>LDL-C (mg/dl)</b>	112.5	156.73	174.09	183.73	165.19	0.000
	11.733	22.034	30.481	23.605	29.113	
<b>HDL-C (mg/dl)</b>	38.00	33.73	31.19	28.36	32.3	0.000
	3.376	4.052	3.832	3.585	4.331	
<b>TG (mg/dl)</b>	169.75	182.86	225.78	265.09	208.22	0.000
	13.72	32.38	29.478	21.375	40.463	
<b>Total Cholesterol (mg/dl)</b>	219.06	238.21	238.3	253.18	238.95	0.237
	16.553	29.542	32.104	23.878	30.191	

Abbreviations: SBP, systolic blood pressure. DBP, diastolic blood pressure. HDL , high density lipoprotein. LDL, low density lipoprotein. TG, triglyceride

Table 3. Severity of Renal Dysfunction and Mean Values of Laboratory, and echocardiographic Variables.

Variables (mean± SD)	CKD Stages				Total	P- Value
	Stage 2 eGFR ≥60	Stage 3 eGFR 30-59	Stage 4 eGFR 15-29	Stage 5 eGFR <15		
<b>BMI</b>	24.12 1.36	26.92 2.96	27.25 2.61	26.36 2.65	26.92 2.78	0.134
<b>Hb(gm/dl)</b>	13.6 0.39	11.46 1.64	10.28 1.49	9.37 1.37	10.83 1.74	<0.001
<b>Urine ACR</b>	222 130.51	713.05 890.63	982.81 702.97	1320.45 640.3	867.33 802.95	0.024
<b>LVMI</b>	136.09 78.86	158.27 69.79	188.16 120.91	198.73 64.24	176.95 96.45	0.057
<b>Intact PTH</b>	65.02 9.83	185.25 61.9	326.5 65.61	439.09 37.93	264.76 110.25	<0.001
<b>Phosphate (mg/dl)</b>	3.3 0.18	4.13 0.48	4.45 0.37	4.81 0.38	4.3 0.5	<0.001
<b>Calcium (mg/dl)</b>	9.10 0.95	8.23 0.59	7.68 0.53	7.44 0.31	7.94 0.65	<0.001
<b>EF%</b>	53.25 1.5	53.18 6.276	50 5.22	47.27 4.75	51.29 5.9	0.002
<b>LVPWTd(cm)</b>	0.97 0.28	1.13 0.33	1.28 0.24	1.3 0.26	1.2 0.3	0.013
<b>SWTd (cm)</b>	1.05 0.31	1.12 0.32	1.34 0.73	1.22 0.28	1.22 0.54	0.183
<b>IVIDd(cm)</b>	5.7 0.45	5.83 0.34	5.81 0.38	5.7 0.32	5.81 0.36	0.683

Abbreviations: BMI, body mass index. Hb, hemoglobin. Urine ACR, urine albumine to creatinine ratio. LVMI, left ventricular mass index. EF, ejection fraction. LVPWTd, left ventricular posterior wall thickness during diastole. LVIDd, left ventricular internal dimension during during diastole. SWTd, septal wall thickness during diastole. Intact PTH,intact parathyroid hormone.

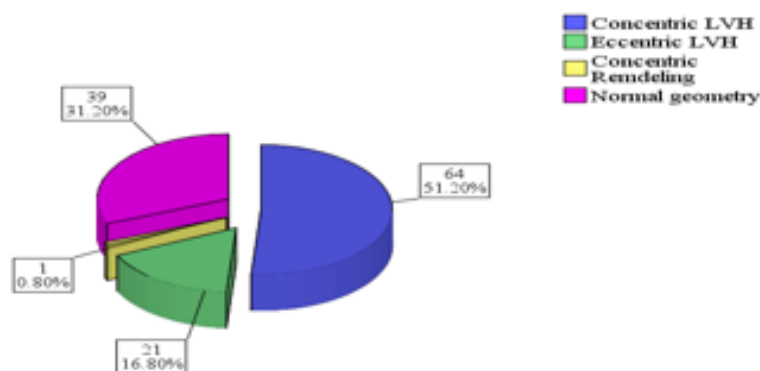


Figure 2. Pattern of LVH in chronic kidney disease patients. LVH, left ventricular hypertrophy.

*Left Ventricular Hypertrophy in Chronic Kidney Disease in Sulaimani City*

**Table 4. Baseline characteristics of study population by LVH.**

Variable		NO LVH	LVH	Total	P-Value
Age Groups (year )	Less than 40	14	7	21	0.001
		66.70%	33.30%	100.00%	
	40-65	19	51	70	
		27.10%	72.90%	100.00%	
More than 65	7	27	34		
Total		40	85	125	
		32.00%	68.00%	100.00%	
Gender	Male	28	47	75	0.084
		37.30%	62.70%	100.00%	
	Female	12	38	50	
		24.00%	76.00%	100.00%	
Total		40	85	125	
		32.00%	68.00%	100.00%	
Body mass index (BMI) Groups	Normal (18.5-24.9)	10	17	27	0.045
		37.00%	63.00%	100.00%	
	Overweigh (25-29.9)	29	52	81	
		35.80%	64.20%	100.00%	
	Obese ≥30	1	16	17	
5.90%		94.10%	100.00%		
Total		40	85	125	
		32.00%	68.00%	100.00%	

LVH, left ventricular hypertrophy.

**Table 5 . Baseline laboratory characteristics of study population by LVH**

Variable		LV mass		Total	P- Value
		NO LVH	LVH		
hemoglobin level	No Anemia	13	5	18	0.000
		32.50%	5.90%	14.40%	
	Anemia	27	80	107	
		67.50%	94.10%	85.60%	
Total		40	85	125	
		100.00%	100.00%	100.00%	
Intact PTH	High	38	85	123	0.038
		95.00%	100.00%	98.40%	
	Normal	2	0	2	
		5.00%	0.00%	1.60%	
Total		40	85	125	
		100.00%	100.00%	100.00%	
Urine ACR	Moderate increase	18	23	41	0.117
		43.90%	56.10%	100.00%	
	Normal	0	1	1	
		0.00%	100.00%	100.00%	
Stages of chronic kidney disease(CKD)	Severe increase	22	61	83	0.005
		26.50%	73.50%	100.00%	
	Stage2 N. (%)	3 (75.0)	1 (25.0)	4 (100.0)	
	Stage3 N.(%)	25 (44.6)	31 (55.4)	56 (100.0)	
	Stage4 N.(%)	10 (18.5)	44 (81.5)	54 (100.0)	
Stage5 N.(%)	2 (18.2)	9 (81.8)	11 (100.0)		
Total	Total N.(%)	40 (32.)	85 (68.0)	125 (100.0)	

Abbreviations: LVH, left ventricular hypertrophy.

Table 6. Baseline characteristic of study population by LVH.

Variables (mean ± SD )	LVH		Total (125)	P- Value
	No LVH (40)	LVH (85)		
<b>SBP (mmHg)</b>	144.32 19.55	166.4 15.85	159.34 19.93	<0.001
<b>DBP (mmHg)</b>	82.85 11.17	89.04 8.66	87.06 9.92	
<b>LDL (mg/dl)</b>	153.38 27.18	170.75 28.46	165.19 29.11	0.002
<b>HDL (mg/dl)</b>	34.25 4.37	31.38 4.01	32.3 4.33	
<b>TG (mg/dl)</b>	191.83 34.59	215.93 40.90	208.22 40.46	0.002
<b>Cholesterol (mg/dl)</b>	228.65 29.60	243.8 29.4	238.95 30.19	
<b>Calcium (mg/dl)</b>	8.2 0.73	7.83 0.59	7.949 0.65	0.003
<b>Phosphate (mg/dl)</b>	4.06 0.53	4.421 0.45	4.306 0.51	

Abbreviations: SBP, systolic blood pressure. DBP, diastolic blood pressure. HDL , high density lipoprotein. LDL, low density lipoprotein. TG, triglyceride. LVH, left ventricular hypertrophy.

Table 7. Characterization of Left Ventricular Hypertrophy in Relation to Renal Function.

CKD Stages	Types of LVH (N=85)		Total N. (%)	P- Value
	Concentric LVH N.(%)	Eccentric LVH N. (%)		
<b>Stage 2 eGFR ≥60</b>	1 (100)	0 (0.0)	1 (100)	0.073
<b>Stage 3 eGFR 30-59</b>	19 (61.3)	12 (38.7)	31 (100)	
<b>Stage 4 eGFR 15-29</b>	35 (79.5)	9 (20.5)	44 (100)	
<b>Stage 5 eGFR &lt;15</b>	9 (100)	0 (0.00)	9 (100)	
<b>Total</b>	64 (75.3)	21 (24.7)	85 (100)	

**Table 8. Characterization of Left Ventricular Hypertrophy in Relation to Systolic BP, Parathyroid hormone and hemoglobin**

Variables (mean ± SD)	Types of LVH (N=85)		Total (85)
	Concentric LVH (64)	Eccentric LVH (21)	
SBP (mmHg)	168.73	159.29	166.40
	14.22	18.66	15.88
PTH (pg/ml)	311.0	216.52	287.66
	100.02	78.41	103.19
Hb (gm/dl)	10.13	10.65	10.260
	1.57	1.69	1.60

## DISCUSSION

In this cross-sectional study we have demonstrated a high prevalence of LVH in CKD patients prior to institution of dialysis. We have described relationship between the presence of LVH and different stages of CKD, blood pressure, anemia, intact PTH, and minerals. The present study showed a linear relation between ageing and progressive decline in renal function and this is similar to other published study<sup>(1)</sup><sup>(14)</sup><sup>(15)</sup>. Based on events that CKD risk factors are increasing with ageing process like diabetes mellitus and hypertension. We have found that prevalence of LVH was increasing with ageing process among CKD patients largely due to increased risk factor for LVH with age (P =0.001).this is similar to Regina C. R. M. Abdulkader et al study<sup>(15)</sup>.

In this study we also found an overall prevalence of LVH was 68.0% in patients with predialysis CKD and the prevalence of LVH was increased with progressively decline in renal function ( P =0.005), and this is similar to Aktas Yilmaz et al. study, reported that prevalence of LVH in CKD was detected in 67.6% of patients with CKD stages-3 and -4 at the baseline and increased to 89.7% in one year in Turkey (P = 0.011)<sup>(16)</sup>.

However, some previous study have reported an unusually very high prevalence of LVH, like in the study of Sung-Kyu Ha et al. in which it was reported the prevalence of LVH was 87.0% in CKD patients<sup>(17)</sup>, the reasons for this might be suboptimal medical treatment of studied patient population and different age population with different risk factors parameter for example level hemoglobin, intact PTH, blood pressure and proteinuria. In the present study, all participants were recruited from a dedicated nephrology department,

which may guarantee better treatment according to the current guidelines. But result of present study is near to Amoako Y A et al. study found that prevalence of LVH in CKD patients was (64.5%)<sup>(1)</sup>. In the other study from Pakistan showed 56.3% of CKD patients had LVH<sup>(18)</sup>, the comparatively lower frequency of LVH in their study could be due to the exclusion of patients with high BP and mean age their study patients was 42 year. Also result of present study was near to Richard J. et al.study,in a randomized controlled trial, reported that CKD progresses, the prevalence of LVH increases, and by stage5 CKD,prior to initiation of renal replacement therapy, about 70–90% of patients exhibit LVH of varying degrees of severity<sup>(19)</sup>.

There was strong association between decline renal function and increasing LVMI with greater strength association at lower levels of renal function. In considering patterns of LV geometry, it has been hypothesized that continuous increment of cardiac pressure led to concentric hypertrophy, whereas continuous increment of cardiac volume produced eccentric hypertrophy<sup>(17)</sup>. Sambhi R S et al. reported the main pattern of LVH among patients with CKD, was concentric hypertrophy based on echocardiographic evaluation<sup>(20)</sup>. In the present study, the main pattern of LVH was concentric hypertrophy 75.3% and this result is similar to Sambhi R S et al.<sup>(20)</sup>.

However, Levin et al. showed that main pattern of LVH was eccentric hypertrophy 57.8% in Canada<sup>(21)</sup>. While concentric LVH has been shown to predominate in predialysis CKD patients<sup>(22)</sup>, and prevalence of systolic hypertension and hyperparathyroidism were higher in concentric LVH<sup>(21)</sup>. An eccentric LVH was far more often associated with mortality than concentric LVH among CKD patients<sup>(2)</sup>. chronic kidney disease (CKD)

has been associated with an increased incidence of heart failure, even in subjects with early declines in kidney function<sup>(23)</sup>. In the present study, LVEF was  $53.25 \pm 1.50\%$  in mild kidney dysfunction with  $eGFR \geq 60$  ml/min per  $1.73m^2$  and LVEF was  $49.54 \pm 5.21\%$  those CKD patient with  $eGFR < 30$  ml/min per  $1.73m^2$  (P-value=0.002)<sup>(23)</sup>. the study findings are consistent with previous studies of Devereux et al. and Asp A M et al.<sup>(24) (25)</sup>. Although Asp A M et al. used Tissue Doppler Imaging that is a more sensitive tool than conventional echocardiography for detection of impaired LV function in the CKD patients.

We have described the relationship between the presence of LVH and eGFR, hemoglobin, systole and diastole BP, intact PTH, HDL cholesterol, LDL cholesterol, Triglyceride and total cholesterol. In the present study, eGFR had the strongest negative linear correlation with LVH, followed by hemoglobin. Also in regression analysis, systolic and diastolic BP has the strongest linear correlation with LVMI, followed by intact PTH and for some degree minerals. In the present study, we have found that those with anemia are more likely to have LVH than those without anemia (P-value=0.000).of note, anemia or low level hemoglobin has been consistently associated with LVH in end stage renal disease<sup>(26)(27)</sup> and the result of this study also similar to those in literature.

Systolic blood pressure is consistently associated with LVH in studies in the general and dialysis populations<sup>(28) (29) (30)</sup>. Elevated systolic BP has a continuous, graded, and independent association with risk of coronary heart disease, stroke, and ESKD<sup>(31) (32)</sup>. Nitta K et al. found that systolic BP was higher in patients with LVH than in patients without LVH in CKD-JAC study<sup>(32)</sup>. Also we reported mean SBP and mean DBP were higher among LVH group compared to CKD patients without LVH. from the pathophysiologic standpoint, an increase in afterload induces concentric LVH, whereas volume overload leads to concentric LVH. In patients with CKD, the simultaneous coexistence of all factors (hypertension, arterial stiffness, volume expansion, and anemia) may preclude the development of specific alterations in LV geometry<sup>(32)</sup>.

We have noted that the level of intact PTH was elevated in CKD patients with LVH compared with those without LVH, and this result is similar to Randon, R B et al. study<sup>(33)</sup>. The mean serum calcium level was low and mean serum phosphorus was normal in the present study. Hypocalcemia was associated with LVH by

logistic regression analysis. Although its mechanism is not completely known, but hypocalcemia followed by vitamin D deficiency may be related with pathogenesis of LVH.

The presence of microalbuminuria has also been shown to be predictive of LVH in patients with essential hypertension using echo<sup>(12)(34)</sup>. We investigated the relationship of urinary albumin to creatinine ratio (urine ACR) with LVH in CKD patients. In the present study, urine ACR had not a significant relationship with LVH (P-value = 0.534), but in another study which was done in Cardiovascular Research Centre, Faculty of Medicine, University of Glasgow, UK Department of Cardiology by McQuarrie et al. investigated twenty-six patients the result was showed PCR was significant correlated with LVMI (P = 0.001), and level of urinary protein excretion is independently and significantly associated with left ventricular mass in patients with CKD<sup>(35)</sup>. This difference could be related to that McQuarrie and his friends used cardiac MRI to detect LVH, and the age their patient was older compared to the age in this study and they had a smaller number of patients.

Obesity is thought to be an independent risk factor of LVH, and heart disorders in obesity include structural adaptation with LVH and functional abnormalities<sup>(32)(36)</sup>. Kotsis et al. found day time pulse pressure and obesity are predictors of LVH in individuals with normal blood pressure<sup>(37)</sup>. In obese hypertensive patients, metabolic syndrome is a risk factor for LVH independently of age and systolic blood pressure and effects of metabolic syndrome on LVH are mainly related to degree of abdominal obesity<sup>(38)</sup>. In the present study, we have found BMI had some kind of relationship with LVH (P-value=0.045), and this result is near agreement with Nitta K et al. study<sup>(32)</sup> reported a body mass index BMI was independently associated with LVH in CKD patients in CKD-JAC study (P-value=0.000) by multivariate logistic regression analysis. This difference related to mean age group, sex and BP.

In this study, we have shown that serum phosphate and serum calcium associated with LVH in CKD patients and the prevalence of hyperphosphatemia and hypocalcemia were higher in those CKD patients with LVH (P =0.001, P =0.003 respectively), and this result is similar to other study<sup>(39)(40)</sup>.

In conclusion, there was a high prevalence of LVH in the study of CKD patients and it was increased with

progressively decline renal function. There was a significant association between systolic and diastolic BP, intact PTH, hemoglobin, BMI, and minerals with LVH in CKD patients.

## REFERENCES

1. Amoako YA, Laryea DO, Bedu-Addo G, Nkum BC, Plange-Rhule J. Left ventricular hypertrophy among chronic kidney disease patients in Ghana. *Pan Afr Med J*. 2017;28:79.
2. De Roij Van Zuijdewijn CLM, Hansildaar R, Bots ML, Blankestijn PJ, Van Den Dorpel MA, Grooteman MPC, et al. Eccentric Left Ventricular Hypertrophy and Sudden Death in Patients with End-Stage Kidney Disease. *Am J Nephrol*. 2015;42(2):126–33.
3. Muntner P, He J, Astor BC, Folsom AR, Coresh J. Traditional and Nontraditional Risk Factors Predict Coronary Heart Disease in Chronic Kidney Disease: Results from the Atherosclerosis Risk in Communities Study. *J Am Soc Nephrol* [Internet]. 2005 Feb [cited 2019 Mar 18];16(2):529–38. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/15625072>
4. Parfrey PS, Foley RN, Harnett JD, Kent GM, Murray DC, Barre PE. Outcome and risk factors for left ventricular disorders in chronic uraemia. *Nephrol Dial Transplant* [Internet]. 1996 Jul [cited 2019 Mar 18];11(7):1277–85. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/8672023>
5. Glasscock RJ, Pecoits-Filho R, Barberato SH. Left ventricular mass in chronic kidney disease and ESRD. *Clin J Am Soc Nephrol* [Internet]. 2009 Dec 1 [cited 2019 Mar 18];4 Suppl 1(Supplement 1):S79-91. Available from: <http://cjasn.asnjournals.org/cgi/doi/10.2215/CJN.04860709>
6. Amann K, Rychlík I, Miltenberger-Milteny G, Ritz E. Left ventricular hypertrophy in renal failure. *Kidney Int Suppl* [Internet]. 1998 Dec [cited 2019 Mar 18];68:S78-85. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/9839289>
7. Cioffi G, Tarantini L, Frizzi R, Stefenelli C, Russo TE, Selmi A, et al. Chronic kidney disease elicits excessive increase in left ventricular mass growth in patients at increased risk for cardiovascular events. *J Hypertens* [Internet]. 2011 Mar [cited 2019 Mar 18];29(3):565–73. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/21150636>
8. Pecoits-Filho R, Barberato SH. Echocardiography in Chronic Kidney Disease: Diagnostic and Prognostic Implications. *Nephron Clin Pract* [Internet]. 2010 [cited 2019 Mar 18];114(4):c242-7. Available from: <https://www.karger.com/Article/FullText/276575>
9. Tikellis G, Arnett DK, Skelton TN, Taylor HW, Klein R, Couper DJ, et al. Retinal arteriolar narrowing and left ventricular hypertrophy in African Americans. the Atherosclerosis Risk in Communities (ARIC) study. *Am J Hypertens* [Internet]. 2008 Mar 1 [cited 2019 Mar 18];21(3):352–9. Available from: <https://academic.oup.com/ajh/article-lookup/doi/10.1038/ajh.2007.57>
10. Hogg RJ, Furth S, Lemley K V, Portman R, Schwartz GJ, Coresh J, et al. National Kidney Foundation's Kidney Disease Outcomes Quality Initiative clinical practice guidelines for chronic kidney disease in children and adolescents: evaluation, classification, and stratification. *Pediatrics* [Internet]. 2003 Jun [cited 2019 Mar 18];111(6 Pt 1):1416–21. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/12777562>
11. Levey AS, Coresh J, Greene T, Stevens LA, Zhang YL, Hendriksen S, et al. Using standardized serum creatinine values in the modification of diet in renal disease study equation for estimating glomerular filtration rate. *Ann Intern Med* [Internet]. 2006 Aug 15 [cited 2019 Mar 18];145(4):247–54. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/16908915>
12. Post WS, Blumenthal RS, Weiss JL, Levine DM, Thiemann DR, Gerstenblith G, et al. Spot urinary albumin-creatinine ratio predicts left ventricular hypertrophy in young hypertensive African-American men. *Am J Hypertens* [Internet]. 2000 Nov [cited 2019 Mar 18];13(11):1168–72. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/11078176>
13. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr* [Internet]. 2005 Dec [cited 2019 Mar 18];18(12):1440–63. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S0894731705009831>
14. Eghan BA, Amoako-Atta K, Kankam CA, Nsiah-Asare A. Survival pattern of hemodialysis patients in Kumasi, Ghana: a summary of forty patients initiated on hemodialysis at a new hemodialysis unit. *Hemodial Int* [Internet]. 2009 Oct [cited 2019 Mar 18];13(4):467–71. Available from: <http://doi.wiley.com/10.1111/j.1542-4758.2009.00379.x>
15. Abdulkader RCRM, Burdmann EA, Lebrão ML, Duarte YAO, Zanetta DMT. Aging and decreased glomerular filtration rate: An elderly population-based study. Ashton N, editor. *PLoS One* [Internet]. 2017 Dec 19 [cited 2019 Mar 18];12(12):e0189935. Available from: <https://dx.plos.org/10.1371/journal.pone.0189935>

16. Aktas Yilmaz B, Mete T, Dincer I, Kutlay S, Sengül S, Keven K, et al. Predictors of Left Ventricular Hypertrophy in Patients with Chronic Kidney Disease. *Ren Fail* [Internet]. 2007 Jan 7 [cited 2019 Mar 18];29(3):303-7. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/17497444>
17. Ha SK, Park HS, Kim SJ, Park CH, Kim DS, Kim HS. Prevalence and patterns of left ventricular hypertrophy in patients with predialysis chronic renal failure. *J Korean Med Sci* [Internet]. 1998 Oct [cited 2019 Mar 18];13(5):488. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/9811177>
18. Ali T, Idrees MK, Shoukat, Akhtar SF. Left ventricular hypertrophy among predialysis chronic kidney disease patients: Sindh institute of urology and transplantation experience. *Saudi J Kidney Dis Transpl* [Internet]. 2017 [cited 2019 Mar 18];28(6):1375-80. Available from: <http://www.sjkdt.org/text.asp?2017/28/6/1375/220856>
19. Glassock RJ, Pecoits-Filho R, Barbareto S. Increased Left Ventricular Mass in Chronic Kidney Disease and End-Stage Renal Disease: What Are the Implications? *Dial Transplant* [Internet]. 2010 Jan 1 [cited 2019 Mar 18];39(1):16-9. Available from: <http://doi.wiley.com/10.1002/dat.20391>
20. Sambhi RS, Gaur AK, Hotchandani R, Aggarwal KK, Kaur S, Gupta M, et al. Patterns of left ventricular hypertrophy in chronic kidney disease: an echocardiographic evaluation. *Indian Heart J* [Internet]. [cited 2019 Mar 18];63(3):259-68. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/22734347>
21. Levin A, Singer J, Thompson CR, Ross H, Lewis M. Prevalent left ventricular hypertrophy in the predialysis population: identifying opportunities for intervention. *Am J Kidney Dis* [Internet]. 1996 Mar [cited 2019 Mar 18];27(3):347-54. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/8604703>
22. Grabysa R, Wańkiewicz Z. Echocardiographic markers of left ventricular dysfunction among men with uncontrolled hypertension and stage 3 chronic kidney disease. *Med Sci Monit* [Internet]. 2013 Oct 9 [cited 2019 Mar 18];19:838-45. Available from: <http://www.medscimonit.com/abstract/index/idArt/889586>
23. Shlipak MG, Lash JP, Yang W, Teal V, Keane M, Cappola T, et al. Symptoms characteristic of heart failure among CKD patients without diagnosed heart failure. *J Card Fail* [Internet]. 2011 Jan [cited 2019 Mar 18];17(1):17-23. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S1071916410010882>
24. Devereux RB, Roman MJ, Liu JE, Welty TK, Lee ET, Rodeheffer R, et al. Congestive heart failure despite normal left ventricular systolic function in a population-based sample: the Strong Heart Study. *Am J Cardiol* [Internet]. 2000 Nov 15 [cited 2019 Mar 18];86(10):1090-6. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/11074205>
25. Asp AM, Wallquist C, Rickenlund A, Hylander B, Jacobson SH, Caidahl K, et al. Cardiac remodelling and functional alterations in mild-to-moderate renal dysfunction: comparison with healthy subjects. *Clin Physiol Funct Imaging* [Internet]. 2015 May [cited 2019 Mar 18];35(3):223-30. Available from: <http://doi.wiley.com/10.1111/cpf.12154>
26. Silberberg JS, Rahal DP, Patton DR, Sniderman AD. Role of anemia in the pathogenesis of left ventricular hypertrophy in end-stage renal disease. *Am J Cardiol* [Internet]. 1989 Jul 15 [cited 2019 Mar 18];64(3):222-4. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/2525871>
27. Weiner DE, Tighiouart H, Vlagopoulos PT, Griffith JL, Salem DN, Levey AS, et al. Effects of Anemia and Left Ventricular Hypertrophy on Cardiovascular Disease in Patients with Chronic Kidney Disease. *J Am Soc Nephrol* [Internet]. 2005 Jun [cited 2019 Mar 18];16(6):1803-10. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/15857925>
28. Foley RN, Parfrey PS, Harnett JD, Kent GM, Martin CJ, Murray DC, et al. Clinical and echocardiographic disease in patients starting end-stage renal disease therapy. *Kidney Int* [Internet]. 1995 Jan [cited 2019 Mar 18];47(1):186-92. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/7731145>
29. Devereux RB, Pickering TG, Alderman MH, Chien S, Borer JS, Laragh JH. Left ventricular hypertrophy in hypertension. Prevalence and relationship to pathophysiologic variables. *Hypertens (Dallas, Tex 1979)* [Internet]. 1987 Feb [cited 2019 Mar 18];9(2 Pt 2):II53-60. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/2879790>
30. Parfrey P s., Griffiths SM, Harnett JD, Taylor R, King A, Hand J, et al. Outcome of Congestive Heart Failure, Dilated Cardiomyopathy, Hypertrophic Hyperkinetic Disease, and Ischemic Heart Disease in Dialysis Patients. *Am J Nephrol* [Internet]. 1990 [cited 2019 Mar 18];10(3):213-21. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/2143353>
31. Locatelli F, Bommer J, London GM, Martín-Malo A, Wanner C, Yaqoob M, et al. Cardiovascular disease determinants in chronic renal failure: clinical approach and treatment. *Nephrol Dial Transplant* [Internet]. 2001 Mar [cited 2019 Mar 18];16(3):459-68. Available from:

## *Left Ventricular Hypertrophy in Chronic Kidney Disease in Sulaimani City*

<http://www.ncbi.nlm.nih.gov/pubmed/11239016>

32. Nitta K, Iimuro S, Imai E, Matsuo S, Makino H, Akizawa T, et al. Correction to: Risk factors for increased left ventricular hypertrophy in patients with chronic kidney disease: findings from the CKD-JAC study. *Clin Exp Nephrol* [Internet]. 2019 Jan 9 [cited 2019 Mar 18];23(1):99-99. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/30094596>
33. Randon RB, Rohde LE, Comerlato L, Ribeiro JP, Manfro RC. The role of secondary hyperparathyroidism in left ventricular hypertrophy of patients under chronic hemodialysis. *Brazilian J Med Biol Res = Rev Bras Pesqui medicas e Biol* [Internet]. 2005 Sep [cited 2019 Mar 18];38(9):1409-16. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/16138225>
34. Dell'omo G, Giorgi D, Di Bello V, Mariani M, Pedrinelli R. Blood pressure independent association of microalbuminuria and left ventricular hypertrophy in hypertensive men. *J Intern Med* [Internet]. 2003 Jul [cited 2019 Mar 18];254(1):76-84. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/12823644>
35. McQuarrie EP, Patel RK, Mark PB, Delles C, Connell J, Dargie HJ, et al. Association between proteinuria and left ventricular mass index: a cardiac MRI study in patients with chronic kidney disease. *Nephrol Dial Transplant* [Internet]. 2011 Mar 1 [cited 2019 Mar 18];26(3):933-8. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/20624770>
36. Alpert MA. Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. *Am J Med Sci* [Internet]. 2001 Apr [cited 2019 Mar 18];321(4):225-36. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/11307864>
37. Kotsis V, Stabouli S, Toumanidis S, Tsivgoulis G, Rizos Z, Trakateli C, et al. Obesity and daytime pulse pressure are predictors of left ventricular hypertrophy in true normotensive individuals. *J Hypertens* [Internet]. 2010 May [cited 2019 Mar 18];28(5):1065-73. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/20411600>
38. Guerra F, Mancinelli L, Angelini L, Fortunati M, Rappelli A, Dessì-Fulgheri P, et al. The Association of Left Ventricular Hypertrophy with Metabolic Syndrome is Dependent on Body Mass Index in Hypertensive Overweight or Obese Patients. *Federici M, editor. PLoS One* [Internet]. 2011 Jan 31 [cited 2019 Mar 18];6(1):e16630. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/21304952>
39. Chue CD, Edwards NC, Moody WE, Steeds RP, Townend JN, Ferro CJ. Serum phosphate is associated with left ventricular mass in patients with chronic kidney disease: a cardiac magnetic resonance study. *Heart* [Internet]. 2012 Feb 1 [cited 2019 Mar 18];98(3):219-24. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/22021416>
40. Di Lullo L, Gorini A, Russo D, Santoboni A, Ronco C. Left Ventricular Hypertrophy in Chronic Kidney Disease Patients: From Pathophysiology to Treatment. *Cardiorenal Med* [Internet]. 2015 Oct [cited 2019 Mar 18];5(4):254-66. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/26648942>