IJKD KIDNEY DISEASES

Echocardiographic Indices in Pediatric Chronic Kidney Disease

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Keywords. pediatric, chronic kidney disease, echocardiography

Original Paper

Introduction. Cardiovascular disease (CVD) may accompany chronic kidney disease (CKD), resulting in additional complications and increased death rate. This study was performed to evaluate cardiac structure and function and several risk factors in hospitalized CKD children.

Methods. Seventy-four children with CKD were enrolled in this cross-sectional descriptive study. Two-dimensional and M-mode ultrasonography, Doppler flow velocity and Tissue Doppler Imaging (TDI) were used to evaluate cardiac chamber size, left ventricular mass (LVM) and echocardiographic indices of ventricular function.

Results. Advanced stages of CKD showed statistically insignificant increased LVM and LVM indexed to height$^{2.7}$ (LVMI), and mildly reduced diastolic function. Hypertensive patients had an insignificant increase in the incidence of left ventricular hypertrophy (LVH) defined as LVMI greater than 95th percentile for age and sex and LVH2 as LVMI2 more than 95 gr/m^2 for girls and more than 115gr/m^2 for boys older than 8 years. Patients with LVH had lower left ventricular ejection fraction (LVEF) and abnormal right ventricular (RV) function based on the tricuspid valve systolic velocity (TV S′) survey. LVH2 cases, however, revealed decreased LV systolic function according to ejection fraction (EF) and abnormal mitral valve systolic velocity (MV S′).

Conclusion. LVH related to hypertension and mild systolic and diastolic dysfunction were more prevalent in advanced CKD cases, however TDI showed no statistically significant difference in the prevalence of MV S′ and TV S′. We recommend strict blood pressure control and prevention of renal function deterioration as effective tools for cardiac protection in CKD children.

IJKD 2022;16:88-95
www.ijkd.org
DOI: 10.52547/ijkd.6643

INTRODUCTION

According to National Kidney Foundation (NKF), chronic kidney disease (CKD) is defined as kidney damage with structural or functional abnormalities, or glomerular filtration rate (GFR) less than 60 mL/min/ 1.73 m^2, for more than 3 months. Pediatric CKD may be due to congenital, acquired, inherited or metabolic etiologies.[1]

Patients with CKD are generally at increased risk of cardiovascular disease (CVD). In children with CKD, CVD is the most common cause of death as a consequence of cardiomyopathy, accelerated ischemia or valvular heart diseases. It is responsible for 1000 times higher death rate in CKD patients,

among them the lowest risk being reported in transplant recipients. Traditional CVD risk factors, including hypertension, dyslipidemia, impaired glucose metabolism, hyperuricemia and obesity, are more common among CKD patients. Uremic risk factors for CVD, including impaired metabolism of minerals, volume overload, proteinuria, malnutrition, anemia and inflammation also enhance the traditional risk factors progression.[2-7]

Cardiovascular alterations during CKD progression appear to be related to adaptive biochemical and hemodynamic mechanisms. It seems that cardiovascular changes initially lead to improvement in left ventricular and vascular function but ultimately result in decreased ventricular performance and cardiorespiratory fitness.[3,4]

There are two mechanisms for cardiovascular involvement in CKD. The first one is the increased left ventricular mass (LVM), either as concentric left ventricular hypertrophy (LVH) which is mostly thought to be due to hypertension, or eccentric LVH possibly due to cardiac remodeling, secondary to volume and sodium overload, low hemoglobin concentration or induced arteriovenous shunts. The second mechanism is vascular involvement due to underlying atherosclerosis, arteriosclerosis and arterial wall calcification, presenting as increased intima-media thickness and stiffness as well as coronary arterial wall calcification. [4,5]

According to an echocardiographic study on 2273 healthy children , left ventricular mass index more than 40 gr/$m^{2.7}$ in girls and 45 gr/$m^{2.7}$ in boys can be considered as LVH (compatible with > 95th percentile) for children older than 9 years old, however these values show significant variations in younger children and it is recommended to use a table consisting different percentiles for younger age groups.[8]

Unlike adult CKD, LV systolic function is almost frequently preserved in pediatric CKD and in this group, subtle abnormalities, less than that expected, are found in the presence of more severe LVH. Altered midwall shortening of LV myocardium occurs during CKD progression. Impaired ventricular diastolic function due to decreased ventricular compliance is more prevalent in CKD patients under dialysis. Anemia, hyperphosphatemia, increased calcium-phosphorus product and LVH are risk factors for CVD. Some studies suggest factors such as increased levels of fibroblast growth factor 23, secondary hyperparathyroidism and abnormal vitamin D signaling as important mechanisms for development of CVD in CKD patients.[3-5,9]

Changes in circulatory inhibitors of calcification and regulators of calcium and phosphorous homeostasis, uremic arteriopathy and atherosclerotic intimal calcification play important roles in vascular involvement causing stiffness and thickening of the vessel wall.[3,4] According to the mentioned factors mentioned, it makes sense to control traditional CVD risk factors in children, early in the course of CKD and to evaluate cardiac and coronary complications periodically to prevent organ damage.[10]

The initiative for this survey was the presence of limited studies on cardiac involvement in pediatric CKD population. In the current study, echocardiographic indices of a cross sectional a cohort of children with CKD were studied and reported.

MATERIALS AND METHODS

In this cross-sectional descriptive study, all pediatric CKD patients less than 18 years, who had been admitted to the nephrology ward of Mofid children's hospital between April 2016 and August 2019 were studied (census method). Patients with congenital heart disease, coronary artery disease, rheumatic valvular heart disease, primary myocardial disease and those who had a history of malignancy were excluded from the study. Ultimately, 74 patients met the inclusion criteria. Written consents were obtained from the parents. Demographic and laboratory data were collected and recorded. Body mass index (BMI) was calculated as weight (kg) / height (m^2). Body surface area (BSA) was calculated using Mosteller equation: √weight (Kg) x height (cm) /3600. Systolic and diastolic blood pressures were measured using an aneroid sphygmomanometer at forearm position, applying mean value of three measurements.

Blood pressure (BP) was categorized based on the clinical practice guideline on childhood hypertension, issued by the American Academy of Pediatrics (AAP) in 2017 as normal for B*P* < 90th percentile for age, sex, and height or < 120 / < 80 mm Hg for adolescents ≥ 13 years old; elevated for B*P* ≥ 90th percentile and < 95th percentile for age, sex, and height; or 120 to 129 / < 80 mm Hg

for adolescents ≥ 13 years. old and hypertension for B*P* > 95th percentile for age, sex, and height or ≥ 130 / 80 mmHg for adolescents ≥ 13 years old.

Complete blood counts, kidney function tests, blood glucose levels and serum electrolytes were measured by Sysmex KX-21N Hematology Analyzer-Sysmex Corporation-Japan, Prestige 24i, Biolis 12i, 24i, 50i Autoanalyzer-Diamond Diagnostics-USA and Automatic electrolyte analyzer XI-921 - Caretium Medical Instruments- China, respectively. Standard quality control protocols were implemented in the laboratory.

GFR was calculated using Schwartz' equation: K x H / Cr where the constant K is 0.45 for children and adolescent girls and 0.7 for adolescent boys, H refers to height and Cr refers to serum creatinine. Patients with CKD were classified according to NKF classification based on GFR levels as stage 1 (≥ 90 mL/min/ 1.73 m^2), stage 2 (60 to 89 mL/ min/ 1.73 m^2), stage 3 (30 to 59 mL/min/ 1.73 m^2), stage 4 (15 to 29 mL/min/ 1.73 m^2) and stage 5 (< 15 mL/min/ 1.73 m^2 or on dialysis),

Cardiac examination was performed with echocardiography by a single academic pediatric cardiologist using Samsung HS70A ultrasound machine- Samsung Company- South Korea. Patients were placed at rest in the left lateral decubitus position. Images were obtained using standard subcostal four chamber, apical four chamber, parasternal long axis and short axis and suprasternal long axis views, applying PE 2-4 and PA 3-8B phased array probes. Sedation (Chloral hydrate 50 to 75 mg/kg, orally) was prescribed as needed.

LVM was measured via two-dimensional directed M-mode echocardiography using the Devereux formula: 0.8 x 1.4 x ((LVEDD + IVST + PWT) ^{3} - LVEDD$^{3)}$) + 0.6; where LVEDD refers to left ventricular end-diastolic diameter, IVST refers to diastolic interventricular septal thickness and PWT refers to diastolic left ventricular posterior wall thickness. LVM was indexed to height$^{2.7}$ (LVMI) and body surface area (LVMI2). Left ventricular hypertrophy (LVH) was considered as LVMI greater than 95th percentile for age and sex[8] or LVMI2 more than 95 g/m^2 for girls and more than 115 g/m^2 for boys older than 8 years.[11]

Doppler flow velocity and Tissue Doppler Imaging (TDI) were used to evaluate ventricular function. Pulsed Doppler evaluation was performed with the cursor positioned between the tips of mitral valve (MV) leaflets to define trans-mitral early (E) and late (A) diastolic peak flow velocities. Study of TDI of the MV annulus with the cursor at the lateral and septal positions and tricuspid valve (TV) annulus with the cursor at the lateral position was applied to determine myocardial velocities at peak systole (S′), early diastole (E′) and late diastole (A′) on apical four chamber view.

Left ventricular ejection fraction (LVEF) was evaluated using biplane technique by machine. LVEF and MVS′ were used to evaluate LV systolic function and E/A, E′/A′ and E/E′ ratios for LV diastolic function. Left ventricular myocardial performance index (LV MPI), a non-geometric index, was calculated based on the equation: "ICT+IRT/ ET" where ICT is the isovolumetric contraction time, IRT is the isovolumetric relaxation time and ET is the ejection time; this equation is used to evaluate global ventricular function using pulsed Doppler signals across the aortic and mitral valves. The TVS′ recorded during TDI and tricuspid annular plane systolic excursion (TAPSE) using M-mode evaluation, with the cursor positioned at lateral TV annulus in apical four chamber view, were analyzed for RV function evaluation. All data were measured during three cardiac cycles and the mean values were considered for analysis.

Age-specific reference values were used for describing these parameters and classification of ventricular dysfunction.[12] The data was entered in Microsoft excel sheet. Data were presented as means and standard deviations for continuous variables and as percentages for categorical variables. Continuous variables were analyzed using Student's T test and categorical variables were analyzed using Pearson's chi-square. The adjustment of the confounders and comparisons of the changes between the study groups were performed using repeated measurements by analysis of variance (ANOVA) models. Statistical significance was set at a *P* level less than .05. Data analysis was performed using IBM SPSS 24.

RESULTS

In this study, 74 pediatric CKD patients including 33 (44.6%) girls and 41 (55.4%) boys were examined. Mean age was 10.15 ± 4.08 years (1-17 years). One CKD patient (1.4%) was at stage 2, 11 (14.9%) were at stage 3, 16 (21.6%) were at stage 4 and 46 (62.2%) were at stage 5. Stages 2 and 3 were considered

as mild and stages 4 and 5 as advanced disease in our study. Accordingly, among the studied patients, 12 (16.2%) were in mild and 62 (83.8%) were at advanced stages. There was no significant difference in demographic findings between the two groups (Table 1). Patients at advanced stages of CKD, demonstrated significantly increased LVM, LVMI and LVMI2 (Table 2).

Twenty-seven (36.5%) patients revealed to have normal blood pressure, 19 (25.7%) had elevated blood pressure and 21 (28.4%) were hypertensive. Systolic and diastolic blood pressures were significantly higher among advanced CKD cases ($P < .05$ and $< .001$) (Table 1 and 3).

Laboratory findings are summarized in Table 1. Serum calcium and sodium levels were lower and phosphorus and calcium- phosphorus product levels were significantly higher among advanced CKD patients ($P < .05$). Uric acid, alkaline phosphatase, albumin and hemoglobin levels were not different between the two groups.

Mean LVEF and mean LV MPI were 67.86 ± 10.97% and 0.51 ± 0.20, respectively. This means that LVEF was normal in most patients although 12 (16.7%) patients showed reduced LVEF. LV MPI was within abnormal limits in 51(72.9%) patients, most of

Table 2. Echocardiographic Quantitative Indices in Relation to CKD Severity

Variable	Mild CKD (Stage 2 to 3)	Advanced CKD (Stage 4 to 5)	*P*
Age, y	8.50 ± 4.21	10.47 ± 4.01	> .05
Height, cm	123.66 ± 23.94	130.53 ± 22.18	> .05
Weight, kg	26.12 ± 13.45	30.05 ± 12.87	> .05
BSA, m^2	0.93 ± 0.32	1.03 ± 0.29	> .05
BMI, kg/m^2	16.10 ± 4.99	16.95 ± 4.06	> .05
Systolic BP, mmHg	104.04 ± 10.81	119.71 ± 18.42	.001
Diastolic BP (mmHg	64.77 ± 7.31	77.83 ± 15.44	< .001
Sodium, meq/L	137.60 ± 2.17	135.09 ± 4.83	.012
Calcium, mg/dL	9.14 ± 0.79	8.43 ± 1.46	.028
Phosphorus, mg/dL	4.53 ± 1.06	6.08 ± 1.89	.001
Calcium- phosphorus Product, mg/dL^2	41.45 ± 10.22	52.44 ± 18.18	.010
Alkaline Phosphatase, IU/L	551.35 ± 310.80	651.65 ± 726	> .05
Albumin, g/dL	3.78 ± 0.75	3.73 ± 0.69	> .05
Hemoglobin, g/dL	9.46 ± 2.64	8.67 ± 2.71	> .05
Uric Acid, mg/dL	6.56 ± 2.62	6.46 ± 2.40	> .05

Abbreviations: *LVM,* left ventricular mass; *LVMI,* left ventricular mass indexed to height;$^{2.7}$ *LVMI2,* left ventricular mass indexed to BSA; *LVEF,* left ventricular ejection fraction; *LV MPI,* left ventricular myocardial performance index; *MV E/A*trans, mitral peak early to late diastolic flow velocity; *MV E'/A',* TDI of mitral valve peak early to late diastolic flow velocity*; MV E/E',* trans-mitral peak early to TDI of mitral valve peak early velocity*; lateral MV S'* TDI, survey of the MV annulus with the cursor at the lateral position; *septal MV S'* TDI, survey of the MV annulus with the cursor at the septal position; *TV S'* TDI, survey of the TV annulus; *TAPSE,* tricuspid annular plane systolic excursion.

Table 3. Blood Pressure and Qualiotative Echocardiographic Variables Related to CKD Stages

Variable	Mild CKD (Stage 2 to 3)	Advanced CKD (Stage 4 to 5)	*P*
LVM, g	66.20 ± 39.92	112.90 ± 77.66	.004
LVMI, $g/m^{2.7}$	35.47 ± 17.83	59.43 ± 51.49	.006
LVMI2, g/m^2	69.30 ± 38.47	110.79 ± 80.11	.009
LV MPI	0.47 ± 0.11	0.52 ± 0.21	> .05
MV E/A	1.58 ± 0.40	1.35 ± 0.45	> .05
Lateral MV E'/A'	2.00 ± 0.73	1.66 ± 0.86	> .05
Septal MV E'/A'	1.64 ± 0.78	1.27 ± 0.41	> .05
MV E/E'	5.34 ± 1.62	6.35 ± 2.55	> .05
Lateral MV S', m/s	9.53 ± 1.28	9.83 ± 2.06	> .05
Septal MV S', m/s	7.99 ± 1.32	8.55 ± 1.56	> .05
TV S', m/s	12.29 ± 2.69	13.74 ± 2.44	> .05
TAPSE, cm	1.97 ± 0.44	2.007 ± 0.38	> .05

Abbreviations: *BP,* blood pressure; *LVH*, left ventricular hypertrophy; *LVEF,* left ventricular ejection fraction; *lateral MV S'* TDI, survey of the MV annulus with the cursor at the lateral position; *septal MV S'* TDI, survey of the MV annulus with the cursor at the septal position; *LV MPI,* left ventricular myocardial performance index; *TAPSE,* tricuspid annular plane systolic excursion; *TV S'* TDI, survey of the TV annulus. Pearson's chi-square was used for comparisons.

Table 3. Blood Pressure and Qualiotative Echocardiographic Variables Related to CKD Stages

Variable	Mild CKD (Stage 2 to 3)	Advanced CKD (Stage 4 to 5)	*P*
Blood Pressure			
Normal BP	9 (75%)	18 (32.7%)	
Elevated BP	3 (25%)	16 (29.1%)	0.011
Hypertension	0 (0%)	21 (38.2%)	
LVH			
No	10 (83.3%)	34 (54.8%)	> .05
Yes	2 (16.7%)	28 (45.2%)	
LVH2			
No	6 (100%)	29 (67.4%)	> .05
Yes	0 (0%)	14 (32.6%)	
LVEF			
Decreased	2 (16.7%)	10 (16.7%)	> .05
Normal	10 (83.3%)	50 (83.3%)	
Lateral MV S'			
Decreased	4 (36.4%)	25 (42.4%)	
Normal	3 (27.3%)	16 (27.1%)	> .05
Increased	4 (36.4%)	18 (30.5/%)	
Septal MV S'			
Decreased	3 (30%)	16 (27.1%)	
Normal	3 (30%)	13 (22%)	> .05
Increased	4 (40%)	30 (50.8%)	
LV MPI			
Normal	3 (25%)	16 (27.6%)	> .05
Increased	9 (75%)	42 (72.4%)	
LV Diastolic function			
Impaired	0 (0%)	16 (26.7%)	0.047
Normal	11 (100%)	44 (73.3%)	
TAPSE			
Decreased	2 (16.7%)	9 (15%)	> .05
Normal	10 (83.3%)	51 (85%)	
TV S'			
Decreased	8 (67.7%)	25 (42.4%)	
Normal	1 (8.3%)	9 (15.3%)	> .05
Increased	3 (25%)	25 (42.4%)	

Abbreviations: *BP*, blood pressure; *LVH*, left ventricular hypertrophy; *LVEF*, left ventricular ejection fraction; *lateral MV S'* TDI, survey of the MV annulus with the cursor at the lateral position; *septal MV S'* TDI, survey of the MV annulus with the cursor at the septal position; *LV MPI*, left ventricular myocardial performance index; *TAPSE*, tricuspid annular plane systolic excursion; *TV S'* TDI, survey of the TV annulus.
Pearson's chi-square was used for comparisons.

whom had advanced CKD (82.4%), however this finding was not statistically significant ($P > .05$). Diastolic function was mildly reduced in 16 (21.6%) patients, all in advanced stages of CKD, and was normal in the rest of the patients, with a statistically significant difference ($P < .05$) (Table 3).

Sodium, potassium, calcium and phosphorus levels did not show any correlation with echocardiographic cardiac function indices (data not shown).

Hypertensive patients revealed lower albumin (3.39 ± 0.68 vs. 3.9 ± 0.64, $P < .05$) and hemoglobin levels (7.71 ± 2.29 vs. 9.35 ± 2.88, $P < .001$), and higher LVM (125.8 ± 93.4 vs. 90.8 ± 58.5, $P > .05$), LVMI (77.2 ± 1.28 vs. 44.6 ± 32.3, $P < .05$), LVMI2 (132.3 ± 102.4 vs. 90.3 ± 62, $P < .05$) than patients with normal and elevated blood pressure. Significant LVH was detected in 13 (61.9 vs. 37.23%) and non-significant LVH (regarding to our LVH2 definition) was detected in 5 (41.7 vs. 18.73%) hypertensive patients compared to normal and elevated blood pressure group ($P < .05$ and $P > .05$). No significant differences in LVEDD, LVEF and diastolic function were found between hypertensive patients and the others.

LVH was detected in 30 (40.5%) patients, 28 (93.3%) of whom were in advanced stages, although the difference between prevalence of LVH in mild and advanced CKD was not statistically significant ($P > .05$) (Table 3). Among patients with LVH, 13 (50%) were hypertensive and 9 (34.6%) had elevated BP, ($P < .05$) (Table 3). In patients with LVH, 8 (26.7%) demonstrated decreased and 22 (73.3%) normal LVEF ($P > .05$); 22 (77.9%) showed abnormal and 7 (24.1%) normal TV S' ($P < .001$), although LV MPI and diastolic function showed no significant difference. LVH was also related to lower hemoglobin levels (9.74 ± 2.9 vs. 7.38 ± 1.57, $P < .001$).

According to our LVH2 definition in children older than 8 years, (14 [100%]) patients with LVH2 were at advanced stages ($P > .05$); 5 (45.5%) were hypertensive and 4 (36.4%) had elevated BP with no statistical significance ($P > .05$), while 5 (35.7%) showed decreased and 9 (64.3%) normal LVEF ($P < .05$), 14 (100%) had abnormal lateral MV S' ($P < .05$) , 11(78.57%) had abnormal and 3(21.42%) normal septal MV S' ($P < .05$). LV MPI and diastolic function showed no significant difference.

Based on TDI, 19 (25.7%) patients had normal lateral MV S', while it was reduced in 29 (39.2%) and increased in 22 (29.7%) of patients. Also 16 (21.6%) patients had normal septal MV S', while it was decreased in 19 patients (25.7%) and increased in 34 (45.9%) of patients. TV S' was normal in 10 (13.5%) patients, while it was decreased in 33(44.6%) and increased in 28 (37.8%) of patients. There was no statistically significant relation between TDI and CKD stages.

DISCUSSION

In this study, 74 CKD patients (33 girls and 41 boys) with mean age of 10.15 ± 4.08 were evaluated. Increased LVM, LVMI and LVMI2 and significant increase in systolic and diastolic blood pressures were seen in advanced CKDs. Tissue Doppler indices (lateral MV S′, septal MV S′ and TV S′) were not different significantly between mild and advanced CKD patients. Also, though the patients with LVH were mostly at advanced stages of CKD, the findings were not significantly different. Evidence of hypertension, lower Hb levels, significantly higher LV MPI and abnormal tricuspid valve S′ were also noted in LVH patients.

Pediatric CKD patients are susceptible to CVD and its consequent morbidity and mortality.[2-6] Most CVD related deaths in CKD patients are due to cardiac arrest, arrhythmias, congestive heart failure and cerebrovascular disease. Unlike adults, in pediatric population cardiac arrhythmias and sudden death are thought to be due to cardiomyopathies, although alteration in ionic milieu may play an additional role. Symptomatic atherosclerosis and diabetes mellitus are rare presentations in pediatric CKD population.[2-5,13]

LVH, left ventricular dysfunction, increased carotid intimal-medial thickness and coronary calcification are considered as markers of cardiovascular involvement and prognostic predictors in CKD patients. However, each of these modifications may initially assist cardiovascular operation in these patients. LVH, as the most common alteration, is believed to reduce wall stress and preserve cardiac function temporarily. LVH occurs early in the course of CKD, develops during its progression, may worsen during dialysis and is usually decreased after transplantation. Several factors, including chronic hypertension, altered vascular tone, increased sympathetic activity, low hemoglobin concentrations and increased PTH levels, are the most suggested contributors in LVH. Histological changes of atherosclerosis also commence early in pediatric CKD patients.[3-6,13-16]

Wong CJ in a CKiD (CKD in Children) study evaluated 586 children aged 1 to 16 years with CKD for cardiovascular complications. After one-year follow-up, 62 (17%) of 366 children showed LVH (11% eccentric and 6% concentric). Presence of hypertension in this study was a good predictor of LVH with odds ratio of 4.3.[17] In the present study however, concentric LVH was more prevalent in advanced CKD cases and was related with hypertension. Also, some degrees of systolic dysfunction according to LVEF and abnormal TV S′ were observed.

In the study by Matteucci *et al.*, which was performed with the cooperation of Effect of Strict Blood Pressure Control and ACE Inhibition on the Progression of CRF in Pediatric Patients (Escape) trial group, 156 children aged between 3 to 18 years with CKD stages 2 to 4 were compared with 133 healthy matched (by age and gender) children. LVH was more common in male patients ($P < .05$) and concentric and eccentric LVH were observed in 12.1% and 21% of patients, respectively. LVH probability was higher in male patients (OR = 2.62) and patients with a higher BMI (OR = 1.56). Lower levels of hemoglobin, glomerular filtration rate (GFR) and age were correlated with LV mass index. LV thickness and cardiac geometry were correlated with serum albumin and CR$P > 10$ mg/dL, respectively.[18] In our study increased LVM, LVMI, LVMI2 and significant increase in systolic and diastolic blood pressures were noted in patients with advanced CKD. Lower Hb levels, significantly higher LV MPI and abnormal tricuspid valve S′, were also noted in patients with LVH.

Hung and colleagues detected higher levels of serum phosphorus and serum calcium and phosphorus products, lower levels of serum albumin and hematocrit, higher trans-mitral E and A waves, E/E′ and worsened LV diastolic dysfunction with progression of CKD in an adult population. They also demonstrated that concentric and eccentric LVH affects systolic and diastolic functions and that impaired LV relaxation is related to CKD worsening.[19] In our study however, significantly higher levels of serum phosphorus and serum calcium phosphorus product and significantly lower levels of serum calcium and sodium were detected in more patients with more advanced CKD, Systolic function impairment was not significantly different between patients with mild vs. advanced CKD, however patients with advanced CKDs showed significantly higher rate of diastolic dysfunction.

Tesdemir and colleagues demonstrated lower E/A and increased left atrial dimension in CKD patients as compared with healthy controls.[20] Our study data also suggested decreased E/A in 21.6% of patients, all of them in advanced stages.

Rao and colleagues evaluated cardiac function in

250 adult CKD patients. They described increasing prevalence of LV systolic and diastolic dysfunction with progression of CKD, while systolic dysfunction was noted in 16.7% of our cases, with no significant difference between mild and advanced CKD stages; however diastolic dysfunction was only detected among cases in advanced stages.[21]

Laddha and colleagues showed increased incidence of LVH and systolic and diastolic dysfunction in adult hypertensive end stage renal disease patients.[9] Although LVH was more prevalent in our hypertensive patients and in advanced stages, this difference was not statistically significant. Also, most patients in the present study had normal EF, but their trans-mitral systolic velocities were abnormal, and this was not significantly related to CKD severity. Diastolic function was mildly impaired in advanced stages of CKD. Hypertensive CKDs demonstrated increased LVMI, LVMI2. Children with LVH had abnormal TV S′.

Rinat and colleagues evaluated traditional and non-traditional risk factors in 70 CKD children. They showed evidence of CVD at early stages of CKD according to underlying risk factors, and consequently emphasized on strict blood pressure and hemoglobin monitoring in these patients.[22] In our study, electrolyte imbalance and hypertension were more prevalent in patients with advanced CKD. Also, hypertensive patients showed increased incidence of LVH.

In another study on 69 post-transplantation CKD patients compared to 33 healthy children, DoVal and colleagues demonstrated that duration of dialysis, hypertension, anemia and dyslipidemia were the risk factors for increasing LVM and IMT, but only systolic blood pressure was related to outcome [(23)]. Hypertension and anemia became more severe during CKD progression in our patients.

Lindblad and colleagues reported development of LV diastolic dysfunction according to E′/A′ and E/E′ ratios even after transplantation and emphasized on applying TDI survey for LV functional study and close blood pressure monitoring.[24] Our study revealed diastolic dysfunction as decreased E′/A′ level in advanced CKD, but E/E′ did not show any significant difference between patients with mild and advanced CKD.

CONCLUSION

This study revealed that concentric LVH was more prevalent in advanced CKD cases and this was correlated with hypertension. Our CKD patients showed some degrees of systolic dysfunction based on LVEF and abnormal TV S′. Diastolic function was also impaired in advanced stages of CKD, but this impairment had no significant relation with hypertension. We also found no statistically significant difference in lateral MV S′, septal MV S′, TV S′ based on TDI. It seems that prevention of CKD progression and restrictive control of hypertension could be effective in management of concentric and eccentric LVH.

STUDY LIMITATIONS

According to the age range of the studied patients (children younger than 18 years in general), possible cardiac complications may appear in later life, which we were not able to follow them.

CONFLICT OF INTEREST

The authors declare no conflict of interests.

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Received October 2021
Revised December 2021
Accepted February 2022