Research Article

Echocardiographic Findings in Children with Different Stages of Chronic Kidney Disease

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Abstract: Background: The burden of CKD during childhood extends beyond the early years, as both the underlying disease and its cardiovascular consequences may continue to affect health well into adult life.

Objective: To assess the echocardiogrphic findings in children with chronic kidney disease (CKD).

Materials and Methods: This cross-sectional study was carried out at the Department of Pediatric Medicine, National Institute of Child Health, Karachi, Pakistan, from 3rd September 2025 to 20th February 2026, after obtaining approval from the institution (letter: IERB-57/2023, dated: 21st August, 2025). Children of age bracket 1 month to 18 years with CKD (> 3 months duration) were enrolled by non-probability consecutive sampling. Demographic and clinical data were logged. Two-dimensional echocardiography was performed to assess heart functions. Data were analyzed using SPSS 25.

Result: Among 112 children, the median age was 10.0 years (IQR=6.0-13.0), 68 were males (60.7%), and the median illness duration was 16.0 months (IQR=10.0-24.0). CKD stages 1 to 5 were present in 9 (8.0%), 15 (13.4%), 28 (25.0%), 27 (24.1%), and 33 (29.5%) children. Hypertension was present in 46 (41.1%) and dialysis history in 38 (33.9%) children, both increasing significantly ($p< 0.001$). Overall, 73 (65.2%) had at least one echocardiographic abnormality which increased significantly with CKD severity ($p<0.001$). LVH ($p=0.007$), diastolic dysfunction ($p<0.001$) and systolic dysfunction ($p=0.003$) were present in 49 (43.8%), 36 (32.1%), and 19 (17.0%), respectively, and worsening with increase in CKD severity. Left ventricular ejection fraction declined ($p<0.001$), and wall thickness increased ($p<0.001$) with increase in CKD staging.

Conclusion: Echocardiographic abnormalities were highly prevalent in children with CKD and became more prominent with advancing CKD stage. LVH, diastolic dysfunction, and declining systolic performance formed the dominant pattern with structural and functional changes becoming increasingly apparent with increase in CKD severity.

Keywords: Children, Chronic kidney disease, Echocardiography, Ventricular dysfunction, Ventricular hypertrophy.

INTRODUCTION

Chronic kidney disease (CKD) has become an imperative universal health concern as its incidence and prevalence continue to rise [1, 2]. Regardless of the underlying cause, CKD is a medical ailment marked by a gradual worsening of renal function [3]. In children, CKD has several age-specific consequences, one of the most important being its adverse effect on physical growth and development [4]. The burden of CKD during childhood extends beyond the early years, as both the underlying disease and its cardiovascular consequences may continue to affect health well into adult life [5].

Cardiovascular involvement in children with CKD has been evident in numerous studies. In a local investigation of children undergoing maintenance hemodialysis, left ventricular dysfunction was reported in 31%, left ventricular diastolic dysfunction in 47%, and left ventricular hypertrophy (LVH) in 55% [6]. Findings from Egypt also showed evidence of diastolic impairment in 66% children [7]. Data from Nigeria revealed that 86% of children had at least one cardiovascular complication, and the most frequently reported abnormalities were LVH in 27.6%, left atrial enlargement in 21.6%, a combination of both in 17.2%, and ventricular premature contractions in 6% [8]. In another report by Mulia and colleagues, 92.1% of patients had at least one echocardiographic abnormality, while LVH was present in 16.7% [9].

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Available evidence suggests that children with CKD who develop echocardiographic abnormalities tend to have poorer outcomes than those without such changes [10]. Delayed presentation, limited access to diagnostic services, and financial barriers to treatment may all contribute to this unfavorable prognosis. Understanding the echocardiographic profile of pediatric CKD is therefore important, particularly because the disease often follows a progressive course and carries a heightened risk of early cardiovascular mortality. Although the cardiovascular effects of CKD have been extensively studied in adults, corresponding data in children remain limited in our local population. By examining the cardiac changes across different stages of CKD in children may guide better understanding and earlier intervention to improve outcomes of these children. This study intended to evaluate the echocardiographic findings in children with CKD.

MATERIALS AND METHODS

This cross-sectional study was executed at the Department of Pediatric Medicine, National Institute of Child Health (NICH), Karachi, Pakistan, from 3rd September 2025 to 20th February 2026, after obtaining approval from the institution (letter: IERB-57/2023, dated: 21st August, 2025). A sample size of 112 was computed via OpenEpi calculator, with 92.1% proportion of echocardiographic abnormalities in children with CKD [9], at a confidence level of 95% and a margin of error of 5%. Children of age bracket 1 month to 18 years, presented with any stage of CKD and had disease duration of more than 3 months were studied. The exclusion criteria were known cases of any kind of congenital cardiac abnormality or malignancy. Children unable to undergo echocardiographic evaluation due to any reason were also excluded. CKD was classified as per KDIGO 2024 criteria [11]. Eligible patients were consecutively enrolled using a non-probability sampling approach. Written informed consents were sought from parents/care givers of all study participants.

During enrollment, detailed demographic information, including age, gender, and weight, and relevant clinical history regarding primary disease, cause of CKD, duration of CKD, history of dialysis, and details of any comorbidities or any concurrent treatments were recorded for each child. Necessary laboratory investigations were performed as per standard institutional protocols. A two-dimensional echocardiography was conducted, and left ventricular ejection fraction (LVEF), diastolic function, and the presence of LVH were evaluated. LV dysfunction was labeled on the basis of an LVEF <50% [12]. LV diastolic dysfunction was described on the basis of the E/A ratio, calculated using Doppler velocity measurements. E/A ratio <0.75 or >1.8 will be labeled as LV diastolic dysfunction [13]. LVH was labeled as intraventricular thickness or LV posterior wall thickness ≥12 mm [14]. Serum creatnine was measured kinetic Jaffe method. Estimated Glomerular Filtration Rate (eGFR) was estimated employing the updated bedside Schwartz equation with the constant adjusted for non-IDMS traceable Jaffe method for pediatric patients: eGFR (mL/min/1.73 m^2) = 0.55 × height (cm) / serum creatinine (mg/dL) [15]. This methodology ensures accurate staging of CKD in accordance with local laboratory practices where enzymatic methods are not standard. All the necessary data were documented on the standardized proforma developed for this study.

STATISTICAL ANALYSIS

Data were analyzed using IBM-SPSS Statistics, version 25.0. To evaluate continuous variables distribution, Shapiro Wilk test was implemented. Medians and interquartiles (IQR) were used to describe continuous variables, with deviations from normality (i.e., age and weight). Numbers and percentages were used descriptively for categorical data. One-way ANOVA was performed to assess differences between CKD stages in normally distributed data and Kruskal-Wallis test for skewed data. Associations between categorical variables were studied using chi-square and Fisher's exact tests as appropriate. Statistical significance was established with a P-value <0.05. Tukey was used to perform multiple comparisons following ANOVA for significant group differences, while pairwise Mann-Whitney U tests with Bonferroni adjustment were employed following Kruskal-Wallis testing due to multiple comparisons.

RESULT

In a total of 112 children, the median age was 10.0 years (6.0-13.0), and 68 (60.7%) were boys. The median duration of illness was 16.0 months (10.0-24.0). Stage-1 CKD was present in 9 (8.0%) children, stage-2 in 15 (13.4%), stage-3 in 28 (25.0%), stage-4 in 27 (24.1%), and stage-5 in 33 (29.5%) children. Congenital anomalies of the kidney and urinary tract were recorded in 38 (33.9%) children, glomerular disease in 31 (27.7%), hereditary nephropathies in 17 (15.2%), reflux nephropathy or obstructive uropathy in 14 (12.5%), and other or undetermined causes in 12 (10.7%) children. Hypertension

was present in 46 (41.1%) children, diabetes mellitus in 4 (3.6%), and a history of dialysis in 38 (33.9%) children. Age differed significantly across CKD stages, with median age rising from 6.0 (4.0-8.0) years in stage-1 to 12.0 (9.0-15.0) years in stage 5 (p=0.006) with significant difference of stage-I with stage-2, 4 and 5 as shown in as shown in post-hoc analysis. The median weight increased across CKD stages, from 15.0 (10.0 to 21.0) kg in stage-1 to 29.0 (20.0-40.0) kg in stage-5 (p=0.011). The median duration of illness showed a stepwise increase with worsening CKD stage, from 9.0 (6.0 to 12.0) months in stage-1 to 22.0 (16.0-32.0) months in stage-5 (p<0.001). Hypertension was significantly more frequent with advancing disease stage and was documented in 1 (11.1%) child in stage-1, 2 (13.3%) in stage-2, 5 (17.9%) in stage-3, 13 (48.1%) in stage-4, and 25 (75.8%) in stage-5 (p<0.001). History of dialysis increased from no child in stage-1 to 24 (72.7%) in stage-5 (p<0.001). Table **1** is showing demographic and clinical characteristics of patients with respect to CKD stages.

Table 1. Characteristics of Patients across CKD Stages (N=112).

Characteristics	Overall (N=112)	Stage-1 (n=9)	Stage-2 (n=15)	Stage-3 (n=28)	Stage-4 (n=27)	Stage-5 (n=33)	p value
Age (years), median (IQR)	10.0 (6.0-13.0)	6.0 (4.0-8.0)[a]	7.0 (5.0-10.0)[a,b]	9.0 (6.0-12.0)[b,c]	11.0 (8.0-14.0)[b,c]	12.0 (9.0-15.0)[c]	0.006
Male sex, n(%)	68 (60.7%)	5 (55.6%)	8 (53.3%)	17 (60.7%)	17 (63.0%)	21 (63.6%)	0.961
Weight (kg), median (IQR)	22.5 (14.0-33.0)	15.0 (10.0-21.0)[a]	18.0 (12.0-24.0)[a,b]	21.0 (14.0-29.0)[b,c]	25.0 (17.0-36.0)[c,d]	29.0 (20.0-40.0)[d]	0.011
Duration of illness in months, median (IQR)	16.0 (10.0-24.0)	9.0 (60-12.0)[a]	11.0 (8.0-15.0)[a,b]	14.0 (10.0-20.0)[b,c]	18.0 (12.0-26.0)[c,d]	22.0 (16.0-32.0)[d]	<0.001
Hypertension, n(%)	46 (41.1%)	1 (11.1%)	2 (13.3%)	5 (17.9%)	13 (48.1%)	25 (75.8%)	<0.001
Diabetes mellitus, n(%)	4 (3.6%)	0 (0%)	0 (0%)	1 (3.6%)	1 (3.7%)	2 (6.1%)	0.648
History of dialysis, n(%)	38 (33.9%)	0 (0%)	1 (6.7%)	4 (14.3%)	9 (33.3%)	24 (72.7%)	<0.001

Note: Different superscript letters indicate statistically significant differences between groups on post hoc pairwise comparison, whereas groups sharing at least one letter are not significantly different.

Table 2. Laboratory Profile according to CKD Stage (N=112).

Parameters	Overall (N=112)	Stage-1 (n=9)	Stage-2 (n=15)	Stage-3 (n=28)	Stage-4 (n=27)	Stage-5 (n=33)	P-value
Haemoglobin(g/dL, mean ± SD	9.4±1.8	11.4±1.2[a]	10.8±1.1[a,b]	9.8±1.4[b]	8.9±1.5[b]	8.3±1.4[b]	<0.001
Leukocyte count (10^9/L), mean ± SD	8.9±2.7	7.8±1.9	8.1±2.1	8.6±2.4	9.3±2.8	9.8±3.1	0.091
Platelet count (10^9/L), mean ± SD	288.0±86.4	301.2±72.5	294.8±79.6	289.4±82.8	281.7±87.1	276.3±95.4	0.764
Serum creatinine (mg/dL), median (IQR)	3.5 (1.9-5.8)	0.8 (0.7-0.9)[a]	1.2 (1.0-1.4)[a,b]	2.3 (1.8-2.9)[b,c]	4.4 (3.8-5.2)[c,d]	6.5 (5.3-8.1)[d]	<0.001
Serum urea (mg/dL), median (IQR)	76.0 (48.0-112.0)	28.0 (22.-36.0)[a]	39.0 (31.0-48.0)[a]	61.0 (49.0-76.0)[b]	90.0 (74.0-108.0)[c]	122.0 (101.0-146.0)[d]	<0.001
Serum albumin (g/dL), mean ± SD	3.4±0.6	3.9±0.4[a]	3.7±0.5[a,b]	3.5±0.5[a,b]	3.3±0.6[a,b]	3.0±0.6[b]	<0.001
Serum uric acid (mg/dL)	6.3 (5.2-7.8)	4.7 (4.2-5.3)[a]	5.1 (4.5-5.8)[a]	5.9 (5.1-6.8)[a,b]	6.9 (5.9-8.0)[b]	7.7 (6.5-9.1)[b]	<0.001
Serum sodium (mmol/L), mean ± SD	136.1±4.2	138.4±2.9[a]	137.8±3.1[a]	136.7±3.6[a,b]	135.1±4.4[b, c]	134.5±4.8[b]	0.031

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Serum potassium (mmol/L), median (IQR)	4.7 (4.2-5.2)	4.1 (3.9-4.3)[a]	4.3 (4.0-4.6)[a,b]	4.5 (4.2-4.9)[a,b]	4.9 (4.5-5.3)[b]	5.2 (4.8-5.8)[b]	<0.001
Serum magnesium (mg/dL), median (IQR)	2.0 (1.8-2.2)	1.8 (1.7-1.9)[a]	1.9 (1.8-2.0)[a,b]	2.0 (1.9-2.1)[a,b]	2.1 (1.9-2.3)[a,b]	2.2 (2.0-2.4)[b]	<0.001
Estimated GFR (mL/min/1.73 m^2), median (IQR)	26.0 (14.0-49.0)	97.0 (92.0-103.0)[a]	72.0 (66.0-79.0)[b]	45.0 (38.0-52.0)[c]	22.0 (19.0-26.0)[d]	12.0 (10.0-14.0)[e]	<0.001

Note: Different superscript letters indicate statistically significant differences between groups on post hoc pairwise comparison, whereas groups sharing at least one letter are not significantly different.

The mean haemoglobin (p<0.001) decreased, and median serum creatinine increased with increase in CKD staging (p<0.001). The median serum urea increased from 28.0 (22.0-36.0) mg/dL to 122.0 (101.0-146.0) mg/dL from stage-1 to stage-5 CKD (p<0.001), respectively. The mean serum albumin fell significantly across CKD stages (p<0.001). The median serum uric acid rose from 4.7 (4.2-5.3) mg/dL in stage-1 to 7.7 (6.5-9.1) mg/dL in stage-5 (p<0.001). The mean serum sodium (p=0.031), serum potassium (p<0.001), and serum magnesium (p<0.001) varied significantly across CKD stages. The median estimated glomerular filtration rate decreased significantly with increase in CKD staging (p<0.001). Table **2** is showing comparison of laboratory profile according to CKD stages.

Overall, 73 (65.2%) children had at least one echocardiographic abnormality. LVH was recorded in 49 (43.8%) children, left ventricular diastolic dysfunction in 36 (32.1%), and left ventricular systolic dysfunction in 19 (17.0%). Pericardial effusion was present in 11 (9.8%) children, chamber dilatation in 8 (7.1%), and more than one echocardiographic abnormality in 28 (25.0%) children. The identification of at least one echocardiographic abnormality increased significantly with the rise in CKD staging (p<0.001). LVH rose from 1 (11.1%) in stage-1 to 21 (63.6%) in stage-5 (p=0.007), while left ventricular diastolic dysfunction increased consistently with worsenig of CKD staging (p<0.001). Left ventricular systolic dysfunction was confined to stages 3 to 5 and affected 2 (7.1%), 5 (18.5%), and 12 (36.4%) children, respectively (p=0.003). Pericardial effusion became more frequent with advancing CKD stage (p=0.021), whereas chamber dilatation did not differ significantly across CKD stages (p=0.109). More than one echocardiographic abnormality was recorded in 1 (6.7%) child in CKD stage-2, 4 (14.3%) in stage-3, 8 (29.6%) in stage-4, and 15 (45.5%) in stage-5 (p=0.001). The mean LVEF decreased consistently with increase in CKD staging (from 64.2±4.8% in stage-1 to 51.9±8.1% in stage 5, p<0.001), and the mean left ventricular wall thickness also showed a significant rise with the increase in CKD severity (p<0.001). Table **3** is showing details of echocardiographic findings across CKD stages.

Table 3. Echocardiographic Findings across CKD Stages (N=112).

Variable	Overall (N=112)	Stage-1 (n=9)	Stage-2 (n=15)	Stage-3 (n=28)	Stage-4 (n=27)	Stage-5 (n=33)	p value
At least one echocardiographic abnormality, n(%)	73 (65.2%)	2 (22.2%)	5 (33.3%)	16 (57.1%)	21 (77.8%)	29 (87.9%)	<0.001
Left ventricular hypertrophy, n(%)	49 (43.8%)	1 (11.1%)	3 (20.0%)	10 (35.7%)	14 (51.9%)	21 (63.6%)	0.007
Left ventricular diastolic dysfunction, n(%)	36 (32.1%)	-	1 (6.7%)	6 (21.4%)	10 (37.0%)	19 (57.6%)	<0.001
Left ventricular systolic dysfunction, n(%)	19 (17.0%)	-	-	2 (7.1%)	5 (18.5%)	12 (36.4%)	0.003
Pericardial effusion, n(%)	11 (9.8%)	-	-	1 (3.6%)	3 (11.1%)	7 (21.2%)	0.021
Chamber dilatation, n(%)	8 (7.1%)	-	-	1 (3.6%)	2 (7.4%)	5 (15.2%)	0.109
More than one abnormality, n(%)	28 (25.0%)	-	1 (6.7%)	4 (14.3%)	8 (29.6%)	15 (45.5%)	0.001

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Left ventricular ejection fraction (%), mean ± SD	57.8±8.6	64.2±4.8[a]	62.1±5.0[a]	59.4±5.9[a,b]	55.8±7.2[b]	51.9±8.1[c]	<0.001
E/A ratio, mean ± SD	1.28±0.44	1.21±0.16	1.24±0.18	1.27±0.24	1.34±0.29	1.39±0.35	0.084
Left ventricular wall thickness (mm), mean ± SD	9.8±2.6	8.0±0.9[a]	8.5±1.0[a]	9.1±1.4[a]	10.3±1.8[a]	11.7±2.2[b]	<0.001

Note: Different superscript letters indicate statistically significant differences between groups on post hoc pairwise comparison, whereas groups sharing at least one letter are not significantly different.

DISCUSSION

This study observed a high burden of echo cardiac abnormalities in children with CKD as nearly two-thirds had at least one echocardiographic abnormality and the burden rose steadily with worsening CKD stage. These findings may indicate that cardiac remodelling and functional impairment could be beginning early in pediatric CKD, and becoming more evident as kidney dysfunction progresses.

The overall frequency of at least one echocardiographic abnormality in this cohort was high and the stage wise gradient was clinically important, rising from 22.2% in stage-1 to 87.9% in stage-5. In the local study from Lahore in 75 children on maintenance hemodialysis, echocardiographic abnormalities were frequent, with left ventricular hypertrophy in 69.3%, pericardial effusion in 30.7%, and systolic dysfunction in 64% [16]. The higher frequencies in that dialysis only cohort could be expected because it represented a narrower and more advanced population than the present study which included children across all CKD stages. The same stage linked pattern has been described in the 4C program where a recent review summarizing those data reported a prevalence of LVH of 11% in CKD-3a and 48% in CKD-5 [17]. These findings supports the view that worsening kidney dysfunction is strongly linked to progressive cardiac injury in childhood [18].

LVH was the commonest echocardiographic abnormality in this study. This is higher than the 20-30% prevalence reported in pediatric patients of CKD of stage 2-4 in recent literature, yet lower than the values reported in maintenance dialysis populations where prevalence can rise up to 85% [19, 20]. A cardiovascular magnetic resonance study in 124 children with CKD found cardiac remodelling in 35.5% and showed that left ventricular mass index and wall thickness were higher in advanced CKD stages [18]. A recent review of hypertension and cardiovascular risk in pediatric CKD also highlighted the strong stage dependence of LVH and pointed out that blood pressure plays a central role in its development [17]. The somewhat higher burden of hypertrophy in this study may reflect the relatively large proportion of children in stages 4 and 5, the 41.1% prevalence of hypertension, and the clear burden of anemia and dialysis exposure. This matters because LVH in children with CKD is not a benign imaging finding, and may be marking sustained cardiac stress and may precede later heart failure, arrhythmia, and adverse cardiovascular outcomes [17, 21].

Diastolic dysfunction was the second major finding and showed a clear rise with CKD stage. Results of another study indicate that ambulatory blood pressure measurements provided a more accurate method to identify children with CKD at risk of having subclinical cardiac dysfunction, which emphasizes the relationship between elevated blood pressure burden and early diastolic dysfunction [22]. Recent pediatric CKD data also stated that diastolic dysfunction, or heart failure with preserved ejection fraction appears to be more common than overt systolic failure [17]. Left ventricular systolic dysfunction in this study was confined to stages 3 to 5 (17.0% overall), with a progressive rise to 36.4% in stage-5. This burden is lower than the 64% reported in the Pakistani hemodialysis cohort, likely because the present study included earlier CKD stages as well, and not only children receiving maintenance dialysis [16]. A recent analysis from the 4C and HOT KID studies reported a significant reduction in 1st phase ejection fraction among 321 children with CKD compared with 63 controls and found a positive correlation between eGFR and early systolic function [23]. The decreasing trend in conventional LVEF in the present study may indicate that by advanced CKD, clinically meaningful systolic compromise is already evident in a substantial subset of children [24-26].

The increase in left ventricular wall thickness across CKD stages in this cohort provides structural support for the functional findings. A cardiac remodelling study reported that left ventricular wall thickness was higher in advanced CKD and linked these changes to declining kidney function [18]. This agreement is important because it suggests that the echocardiographic phenotype in pediatric CKD is internally coherent with struc-

tural remodelling and functional impairment progressing together rather than as isolated abnormalities. The finding that pericardial effusion became more frequent in later stages also fits with advanced uraemia and fluid imbalance. The lower rate of pericardial effusion in the present cohort compared with the 30.7% reported in the Lahore dialysis study likely reflects inclusion of earlier stage disease and a broader clinical spectrum.

The cross-sectional design of this study did not allow temporal inference regarding when echocardiographic abnormalities developed or whether they progressed with changes in blood pressure control, dialysis exposure, or renal replacement therapy. The study was conducted at a single tertiary center, which may have increased the proportion of advanced CKD and referred complicated cases.

CONCLUSION

Echocardiographic abnormalities were frequent in children with CKD and became more prominent with advancing CKD stage. LVH, diastolic dysfunction, and declining systolic performance formed the dominant pattern with structural and functional changes becoming increasingly apparent with increase in CKD severity.

AUTHORS' CONTRIBUTION

Maheen Khan: Conceptualized, Study design, Methodology, Data analysis and interpretation, Writing Draft and Final approval, final proof to be published.

Mashal Khan: Study design, Methodology, Data analysis and interpretation, Critical review and revision the manuscript and Final approval, final proof to be published.

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Declared none.

ETHICAL DECLARATIONS

Data Availability Statement

Data will be available from the corresponding author upon a reasonable request.

Ethical Approval

This study was approved by the Ethical Review Committee of National Institute of Child Health (NICH), Karachi, Pakistan, (letter: IERB-57/2023, dated: 21st August, 2025).

Consent to Participate

Written consent was sought from parents prior to study commencement.

Consent for Publication

All authors provide consent to publish the work.

Conflict of Interest

Declared none.

Competing Interest/Funding

Declared none.

Use of AI-Assisted Technologies

The authors used ChatGpt to assist with language editing and proof reading. All intellectual work has been done by the authors.

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