



EFFECTS OF CONGENITAL ANOMALIES OF THE KIDNEY AND URINARY TRACT ON BLOOD PRESSURE AND RENAL FUNCTION IN CHILDREN: A HOSPITAL-BASED OBSERVATIONAL STUDY

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Abstract

Background: Congenital Anomalies of the Kidney and Urinary Tract (CAKUT) represent a heterogeneous group of developmental disorders involving the kidneys and urinary tract and are among the leading causes of chronic kidney disease (CKD) in children. Reduced nephron number, urinary tract obstruction, vesicoureteral reflux, and renal dysplasia predispose affected children to hypertension, proteinuria, and progressive renal dysfunction. Early identification of these complications is essential for preventing long-term renal and cardiovascular morbidity. Aim of the study to analyze the effects of congenital abnormalities of the kidneys and urinary tract on blood pressure and serum creatinine levels in children diagnosed with CAKUT.

Methods: A hospital-based observational study was conducted in the Department of Pediatrics at a tertiary care teaching hospital between April 2024 and September 2025. Children aged 1–15 years diagnosed with CAKUT were enrolled. Clinical evaluation included blood pressure assessment, serum creatinine estimation, estimated glomerular filtration rate (eGFR), and proteinuria evaluation. Statistical analysis included Chi-square testing, odds ratio estimation, and comparison of means.

Results: A total of 110 children were evaluated. Male predominance was observed (65.45%). Hypertension was present in 9.09% of patients, renal dysfunction in 11.82%, and proteinuria in 13.64%. Children with CAKUT demonstrated significantly higher odds of hypertension, elevated serum creatinine, and proteinuria. Renal dysfunction was present in 80% of hypertensive children compared with only 5% of non-hypertensive children ($p < 0.0001$). Proteinuria showed the strongest association with CAKUT (OR=22.94; $p = 0.003$).

Conclusion: CAKUT is strongly associated with proteinuria, renal dysfunction, and hypertension. Proteinuria appears to be an early marker of renal injury, while hypertension reflects more advanced disease. Regular monitoring of blood pressure, renal function, and urinary protein excretion is essential to improve long-term outcomes.

Keywords: CAKUT, Hypertension, Proteinuria, Serum Creatinine, Chronic Kidney Disease, Children.

Introduction

Congenital Anomalies of the Kidney and Urinary Tract (CAKUT) represent a heterogeneous group of developmental disorders affecting the kidneys, ureters, bladder, and urethra. These anomalies arise during embryogenesis due to disturbances in the complex interactions between the ureteric bud and metanephric mesenchyme, which are essential for normal nephrogenesis and urinary tract development. CAKUT encompasses a broad spectrum of structural abnormalities, including renal agenesis, renal hypoplasia, renal dysplasia, multicystic dysplastic kidney, vesicoureteral reflux (VUR), pelviureteric junction obstruction (PUJO), duplex collecting system, and posterior urethral valves (PUV). Together, these abnormalities account for a substantial proportion of pediatric kidney disorders and are recognized as the leading cause of chronic kidney disease (CKD) and end-stage kidney disease (ESKD) in children worldwide.[1-4]

The prevalence of CAKUT is estimated to be approximately 3–6 cases per 1,000 live births, although the true incidence may vary according to geographic region, genetic background, and accessibility of prenatal diagnostic services.[5] Advances in antenatal ultrasonography have significantly improved the detection of congenital renal anomalies, enabling diagnosis before birth in many cases. Early identification allows for timely postnatal evaluation and intervention, thereby reducing the risk of complications such as urinary tract infections, hypertension, and progressive renal dysfunction.[6]

Normal renal development is a highly regulated process involving reciprocal signaling between the ureteric bud and metanephric mesenchyme. Disruptions in these signaling pathways can impair nephron formation and urinary tract morphogenesis, resulting in a reduced nephron endowment at birth. Experimental and genetic studies have demonstrated that abnormalities in genes regulating renal embryogenesis, including PAX2, HNF1B, EYA1, RET, and GDNF, contribute significantly to the pathogenesis of CAKUT.[7,8] Furthermore, environmental factors such as maternal diabetes, nutritional deficiencies, exposure to teratogenic drugs, and adverse intrauterine conditions may interact with genetic susceptibility to influence renal development.[9]

The clinical manifestations of CAKUT vary considerably depending on the type and severity of the anomaly. Some children remain asymptomatic and are diagnosed incidentally during antenatal or routine imaging studies, whereas others present with recurrent urinary tract infections, poor growth, abdominal masses, voiding dysfunction, hypertension, or impaired renal function.[10] Structural abnormalities associated with urinary tract obstruction or vesicoureteral reflux can predispose affected children to recurrent infections and progressive renal scarring, ultimately resulting in chronic kidney disease. Consequently, long-term monitoring of these patients is essential to detect early signs of renal injury and prevent disease progression.[11]

One of the most important long-term consequences of CAKUT is chronic kidney disease. Congenital abnormalities of renal structure often result in a reduced number of functioning nephrons. According to the hyperfiltration hypothesis proposed by Brenner and colleagues, nephron deficiency leads to compensatory hypertrophy and hyperfiltration of the remaining nephrons. Although this adaptation initially maintains renal function, persistent hyperfiltration causes glomerular hypertension, proteinuria, and progressive nephron loss over time.[12] Reduced nephron endowment has also been linked to an increased risk of hypertension and cardiovascular disease later in life.[13] Therefore, children with CAKUT require ongoing surveillance for early markers of renal injury.

Hypertension is increasingly recognized as a significant complication in children with congenital renal anomalies. The kidneys play a central role in blood pressure regulation through sodium and water

balance, regulation of extracellular fluid volume, and activation of the renin–angiotensin–aldosterone system (RAAS). Structural abnormalities of the kidneys may disrupt these physiological mechanisms, leading to persistent elevation of blood pressure.[3] Several studies have reported a higher prevalence of hypertension among children with CAKUT compared with the general pediatric population.[14] Hypertension may remain clinically silent for prolonged periods and is frequently identified only during routine clinical assessment. Persistent hypertension accelerates renal damage by increasing intraglomerular pressure and promoting nephron loss, thereby creating a vicious cycle that contributes to progressive CKD.[15]

Proteinuria is another important marker of renal injury in children with CAKUT. Increased urinary protein excretion reflects impairment of the glomerular filtration barrier and often precedes measurable reductions in glomerular filtration rate (GFR). Persistent proteinuria has been identified as a strong predictor of CKD progression and adverse renal outcomes.[16] In pediatric nephrology practice, the urine protein-creatinine ratio is widely used as a reliable and convenient method for assessing proteinuria, particularly because collection of 24-hour urine samples is often difficult in children. Monitoring proteinuria provides valuable information regarding disease activity and progression and assists clinicians in identifying patients who may benefit from renoprotective interventions.[17]

Serum creatinine remains one of the most commonly used biomarkers for evaluating renal function in children. Elevated serum creatinine levels indicate reduced glomerular filtration and are often associated with significant renal impairment. However, serum creatinine alone may not adequately reflect early renal injury; therefore, it is commonly interpreted in conjunction with estimated glomerular filtration rate (eGFR) and proteinuria.[18] Assessment of these parameters is particularly important in children with CAKUT because early renal dysfunction may progress silently before becoming clinically evident.

Despite increasing awareness regarding CAKUT, data concerning the association between hypertension, proteinuria, and renal dysfunction in Indian children remain limited. Most available studies have focused primarily on structural abnormalities and surgical outcomes, while fewer investigations have evaluated the relationship between blood pressure abnormalities and markers of renal injury. Understanding these associations is essential for developing effective monitoring strategies and improving long-term outcomes. Therefore, the present study was undertaken to analyze the effects of congenital abnormalities of the kidneys and urinary tract on blood pressure and serum creatinine levels and to evaluate the correlation between hypertension, renal dysfunction, and proteinuria in children diagnosed with CAKUT.

Materials and Methods

This hospital-based observational study was conducted in the Department of Pediatrics at a tertiary care teaching hospital over an 18-month period from April 2024 to September 2025.

Children aged 1–15 years diagnosed with CAKUT based on ultrasonographic findings were included. Written informed consent was obtained from parents or guardians. Children whose guardians declined participation were excluded.

A detailed clinical assessment was performed for each participant. Demographic characteristics, clinical findings, and imaging reports were recorded. Blood pressure was measured using an appropriately sized pediatric cuff according to standard pediatric recommendations. Serum creatinine, eGFR, and urine protein measurements were obtained.

The primary objective was to determine the association of hypertension with CAKUT. Secondary objectives included assessment of serum creatinine abnormalities and evaluation of the relationship among hypertension, renal dysfunction, and proteinuria.

Statistical analysis was performed using appropriate tests. Continuous variables were expressed as mean $\pm$ standard deviation. Chi-square tests were used to evaluate categorical associations. Odds ratios with 95% confidence intervals were calculated. A p-value less than 0.05 was considered statistically significant.

Results

Demographic Characteristics

A total of 110 children with CAKUT were included. The age distribution ranged from 1 to 15 years. Most patients were diagnosed during late childhood and early adolescence. The highest frequency was observed in the 13–15-year age group (24.55%), followed by the 7–9-year age group (20.91%). Male predominance was evident, with 72 males (65.45%) and 38 females (34.55%), producing a male-to-female ratio of approximately 1.9:1.

Table 1: Prevalence of Hypertension, Renal Dysfunction and Proteinuria among Children with CAKUT (n = 110)

Parameter	Frequency (n)	Percentage (%)
Hypertension	10	9.09
Renal Dysfunction	13	11.82
Proteinuria	15	13.64

Blood Pressure Profile: Hypertension was identified in 10 children (9.09%). Although the overall prevalence was relatively low, hypertensive children exhibited significantly worse renal parameters than normotensive children.

Renal Dysfunction: Renal dysfunction was present in 13 children (11.82%). Elevated serum creatinine levels were significantly more common among children with CAKUT.

Proteinuria: Proteinuria was observed in 15 children. Among CAKUT patients, 28% had proteinuria compared with only 1.67% in children without CAKUT.

Table 2: Association Between Hypertension and Renal Dysfunction

Renal Dysfunction	Hypertensive (n=10)	Normotensive (n=100)	Chi-Square test	P - Value
Present	8 (80.0%)	5 (5.0%)	49.07	< 0.0001
Absent	2 (20.0%)	95 (95.0%)		

Renal dysfunction was identified in 80% of hypertensive children compared with only 5% of non-hypertensive children. This difference was highly significant (Chi-square=49.07; $p < 0.0001$). These findings suggest that hypertension may be an important marker of underlying renal impairment in children with CAKUT.

Table 3: Association of CAKUT with Renal Dysfunction and Proteinuria

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Raised Serum Creatinine	5.38	1.84–15.72	0.002*
Proteinuria	22.94	2.89–182.00	0.003*

The odds of raised serum creatinine were 5.38 times higher in children with CAKUT compared with controls, indicating a strong association between congenital renal anomalies and impaired renal

function.

The odds ratio for proteinuria was 22.94 (95% CI: 2.89–182; $p=0.003$), demonstrating a very strong association.

Discussion

The present study evaluated the relationship between CAKUT, hypertension, renal dysfunction, and proteinuria in pediatric patients. The findings highlight the substantial burden of renal complications associated with congenital renal anomalies.

Male predominance observed in this study is consistent with previous reports by Kommareddy et al. and Kumar et al., who reported a higher frequency of CAKUT among boys, partly due to male-specific conditions such as posterior urethral valves.[19,6]

Hypertension was present in 9.09% of the study population. Although lower than some published reports, the prevalence remains clinically important. Taha et al. reported that hypertension is a frequent complication in children with CAKUT and is strongly associated with progression of chronic kidney disease.[14] Similar observations were described by Flynn et al., who emphasized the importance of early blood pressure monitoring in children with renal abnormalities.[3]

The present study demonstrated a strong association between elevated serum creatinine and CAKUT. Children with congenital renal anomalies had more than fivefold higher odds of renal dysfunction. These findings support previous studies indicating that reduced nephron endowment and compensatory hyperfiltration contribute to progressive loss of renal function.[17,13]

Proteinuria emerged as the strongest marker of renal injury in this study. Children with CAKUT had nearly 23 times higher odds of proteinuria compared with controls. Similar observations have been reported by Westland et al. and Warady et al., who identified proteinuria as an important predictor of CKD progression.[15,20] Persistent proteinuria reflects glomerular injury and often precedes measurable decline in eGFR.

A particularly important observation was the close relationship between hypertension and renal dysfunction. Eighty percent of hypertensive children demonstrated renal impairment compared with only 5% of normotensive children. This finding aligns with the work of Taha et al., who found a significantly greater burden of CKD among hypertensive children with CAKUT.[14] The results support the concept that hypertension and renal dysfunction form a vicious cycle, each accelerating the progression of the other.

The pathophysiological basis for these findings can be explained by nephron deficiency and hyperfiltration injury. Brenner et al. proposed that reduced nephron number increases intraglomerular pressure, leading to proteinuria, progressive sclerosis, and hypertension. This mechanism is particularly relevant in children with renal hypoplasia, dysplasia, and solitary functioning kidneys.[21] The findings also reinforce evidence from the Chronic Kidney Disease in Children (CKiD) cohort, which identified proteinuria, hypertension, and impaired renal function as major predictors of disease progression.[20] Early identification of these risk factors provides opportunities for timely intervention and nephroprotective therapy.

The study has important clinical implications. Children with CAKUT require long-term surveillance even when asymptomatic. Regular monitoring of blood pressure, serum creatinine, eGFR, and urinary protein excretion can facilitate early detection of disease progression and improve outcomes.

Conclusion

Congenital Anomalies of the Kidney and Urinary Tract are strongly associated with proteinuria, renal

dysfunction, and hypertension in children. Proteinuria appears to represent the earliest indicator of renal injury, whereas hypertension reflects more advanced renal involvement. Elevated serum creatinine further confirms the substantial burden of renal dysfunction among affected children.

The strong association between hypertension and impaired renal function emphasizes the need for routine blood pressure monitoring in pediatric CAKUT patients. Early identification of proteinuria and renal dysfunction may allow timely interventions that slow progression toward chronic kidney disease and end-stage renal disease.

Long-term multidisciplinary follow-up is essential to improve renal and cardiovascular outcomes in children with CAKUT.

References

1. Harambat J, van Stralen KJ, Kim JJ, Tizard EJ. Epidemiology of chronic kidney disease in children. *Pediatr Nephrol*. 2012;27(3):363-373.
2. Stonebrook E, Hoff M, Spencer JD. Congenital anomalies of the kidney and urinary tract: clinical review. *Curr Treat Options Pediatr*. 2019;5(3):223-235.
3. Flynn JT, Kaelber DC, Baker-Smith CM, et al. Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents. *Pediatrics*. 2017;140(3):e20171904.
4. Walawender L, Szczepanska M, Szczepanski W. Congenital anomalies of the kidney and urinary tract: current concepts. *Children (Basel)*. 2023;10(4):682.
5. Sanna-Cherchi S, Ravani P, Corbani V, et al. Renal outcome in patients with congenital anomalies of the kidney and urinary tract. *Kidney Int*. 2009;76(5):528-533.
6. Kumar BH, Suresh B, Reddy PR. Clinical profile of congenital anomalies of kidney and urinary tract in children. *Int J Contemp Pediatr*. 2019;6(4):1560-1566.
7. Ichikawa I, Kuwayama F, Pope JC, Stephens FD, Miyazaki Y. Paradigm shift from classic anatomic theories to contemporary cellular and molecular understanding of CAKUT. *Kidney Int*. 2002;61(3):889-898.
8. Nicolaou N, Renkema KY, Bongers EMHF, Giles RH, Knoers NVAM. Genetic, environmental and epigenetic factors involved in CAKUT. *Nat Rev Nephrol*. 2015;11(12):720-731.
9. Ishiwa S, Sanna-Cherchi S. Genetic basis of congenital anomalies of the kidney and urinary tract. *Pediatr Nephrol*. 2019;34(9):1503-1515.
10. Rodriguez MM. Congenital anomalies of the kidney and urinary tract (CAKUT). *Fetal Pediatr Pathol*. 2014;33(5-6):293-320.
11. Soliman NA, Ali RI, Ghobrial EE, Habib EI, Ziada AM. Pattern of congenital anomalies of the kidney and urinary tract in Egyptian children. *Saudi J Kidney Dis Transpl*. 2015;26(2):413-418.
12. Brenner BM, Garcia DL, Anderson S. Glomeruli and blood pressure: less of one, more the other? *Am J Hypertens*. 1988;1(4):335-347.
13. Charlton JR, Baldelomar EJ, Hyatt DM, Bennett KM. Nephron number and its determinants. *Pediatr Nephrol*. 2021;36(4):797-807.
14. Taha K, Catapang M, Becknell B, Matsell DG. Hypertension in children with congenital anomalies of the kidney and urinary tract. *Pediatr Nephrol*. 2024;39(4):1185-1192.
15. Westland R, Schreuder MF, Bökenkamp A, et al. Renal injury in children with a solitary functioning kidney. *Nephrol Dial Transplant*. 2011;26(5):1533-1541.
16. Warady BA, Abraham AG, Schwartz GJ, et al. Predictors of rapid progression of CKD in children. *JAMA Pediatr*. 2015;169(11):1015-1022.
17. van Biljon G, Meintjes CJ, Becker PJ, Karusseit VOL. Risk factors for progression of chronic kidney disease in children. *Nephrology*. 2023;28(5):276-282.
18. Mong Hiep TT, Ismaili K, Collart F, et al. Clinical characteristics and outcomes of children with chronic kidney disease. *Pediatr Nephrol*. 2010;25(5):935-940.
19. Kommareddy A, Vagha K, Vagha JD, et al. A Study of Clinical Profile of Congenital

- Anomalies of Kidney and Urinary Tract (CAKUT) in a Tertiary Care Center. *Cureus*. 2024 Jul 3;16(7):e63766.
20. Warady BA, Abraham AG, Schwartz GJ, et al. Predictors of Rapid Progression of Glomerular and Nonglomerular Kidney Disease in Children and Adolescents: The Chronic Kidney Disease in Children (CKiD) Cohort. *Am J Kidney Dis*. 2015 Jun;65(6):878-88.
 21. Brenner BM, Garcia DL, Anderson S. Glomeruli and blood pressure. Less of one, more the other? *Am J Hypertens*. 1988 Oct;1(4 Pt 1):335-47.