Chronic Kidney Disease in Children at the University of Abuja Teaching Hospital, Abuja, Nigeria (2017-2020)

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ABSTRACT

Background: Childhood chronic kidney disease (CKD) is increasing globally, and it is associated with significant morbidity and mortality. This article describes the incidence, pattern, and outcomes of childhood CKD at the University of Abuja Teaching Hospital (UATH), Abuja, Nigeria.

Materials and Methods: This was a retrospective review of children aged 1 month to 16 years seen at the Department of Paediatrics, UATH, from January 2017 to December 2020.

Results: Ninety-four cases of CKD were seen with an incidence rate of 36 per million-child population per year. The age range was between 1 month and 16 years, with a mean age of 7.7 ± 4.1 years, and a majority (41, 43.6%) was between 5 and 10 years. The commonest causes of CKD were nephrotic syndrome (NS) (58, 61.7) and posterior urethral valve (PUV) (14, 14.8%). There was a male predominance (71/94, 75.5%) which was not statistically significant (p=0.157). Eleven (11.7%) subjects in end-stage kidney disease (ESKD) had chronic intermittent haemodialysis over a mean duration of 2 months before cardiovascular-related mortality ensued. Most of the mortality was from steroid-resistant idiopathic NS (5/11, 45.5%) and PUV (2/11, 18.2%). A child with idiopathic steroid-resistant NS who had living unrelated renal transplantation is surviving.

Conclusion: The incidence of childhood CKD is high, and it is commoner among school-age children. The commonest CKD was NS and PUV. Financial constraints limit the accessibility of kidney replacement therapy (KRT) among those with ESKD. The need for preventive nephrology and public-funded KRT cannot be over-emphasized.

Keywords: *Childhood, chronic kidney disease, Abuja, Nigeria*

INTRODUCTION

Chronic kidney disease (CKD) is the presence of kidney damage (structural or functional abnormality involving pathological, laboratory, or imaging findings) for ≥ 3 months or a glomerular filtration rate (GFR) < 60 ml/min/1.73 m2 for ≥ 3 months [1]. This definition does not preclude children less than 2 years whose GFR is still increasing and/or children born with major congenital anomalies of the kidney and urinary tract (CAKUT) who are less than 3 months old [1,2]. Although children in earlier stages of CKD are asymptomatic and may remain undiagnosed, the progression of CKD to end-stage kidney disease (ESKD) is accompanied by low quality of life, and high mortality and morbidity from cardiovascular diseases and infection [2,3]. The epidemiology of childhood CKD is affected by lack of national renal registries, but aetiology of CKD varies with age,

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genetics, race, and geographical locations [2]. While CAKUT and hereditary nephropathies are responsible for about two-thirds of all cases of CKD in the United States and Europe [4-6];- chronic glomerulonephritis (CGN) predominates in India, Southeast Asia, Latin America, Caribbean area and sub-Saharan Africa [7-14].

In Nigeria, there is a dearth of data on CKD in children. The few available hospital-based reports on CKD put the estimated incidence at 1.6-11 per million-child population, with the leading causes being nephrotic syndrome (NS), CGN, and posterior urethral valve (PUV) [15-18]. Undoubtedly, these hospital-based studies might have underestimated the actual burden of CKD in the community [19].

Nevertheless, the burden of childhood CKD is increasing globally and in Nigeria, and it is fraught with many challenges including late presentation, misdiagnosis, under-reporting, and unaffordability and inaccessibility of kidney replacement therapy (KRT) [15-18]. Payment for haemodialysis is still out-of-pocket with Nigeria's national health insurance scheme (NHIS) catering for only 6 sessions of acute haemodialysis [20].

Thus, a better understanding of the epidemiology of childhood CKD in Nigeria is important. This will increase the index of suspicion that allows for a precise and early diagnosis of CKD [19,20]. In addition, preventive nephrology is cheaper in preventing or reversing the clinical course of CKD progression and prognosis [20].

Considering the foregoing, this study reports the burden and causes of CKD among children aged 1 month to 16 years attending the University of Abuja Teaching Hospital (UATH), Abuja, Nigeria, from January 2017 to December 2020.

MATERIALS AND METHODS

Ethical consideration followed the Helsinki Declaration of 1975, as revised in 1983. Permission was obtained from the Research and Ethics Committee of the UATH, Abuja. All cases of CKD diagnosed among children aged 1 month to 16 years from January 2017 to December 2020 at the Department of Paediatrics, UATH, were included in the study.

The following operational diagnosis of CKD applies:

CKD refers to kidney damage or estimated glomerular filtration rate (eGFR by Schwartz formula) of < 60 ml/min/1.73 m^2 for ≥ 3 months. Kidney damage is pathologic abnormalities or markers of damage on urine, blood, or imaging tests [1]. This also includes ultrasound demonstration of shrunken kidneys [21]. Nomenclatures regarding CKD include normal to increased GFR G1 (GFR ≥ 90), mildly reduced GFR G2 (60-89), moderately reduced GFR G3a (45-59), moderately reduced GFR G3b (30-44), severely reduced GFR G4 (15-29), kidney failure G5 (ESKD) < 15 or treated by dialysis, CKD G5 without kidney replacement therapy, CKD G5 D (treated with dialysis), CKD G1-G5 after transplantation [22].

NS is defined as the presence of oedema, massive proteinuria of spot urine protein creatinine ratio ≥ 200 mg/mmol, hypoalbuminaemia (serum albumin ≤ 25 g/l), and hypercholesterolemia (serum cholesterol > 5.2 mmol/l) [23].

Secondary NS refers to aetiology that is not intrinsic to the kidney and which includes systemic lupus erythematosus (lupus nephritis), human immunodeficiency virus (HIV), hepatitis B, hepatitis C, and sickle cell anaemia (haemoglobin SS) [24].

When NS is not congenital, infantile, or secondary, it is said to be idiopathic NS (INS) [24].

CGN markers were hypertension, azotemia, reduced eGFR, red blood cell cast, proteinuria, hematuria, and symmetrically shrunken kidneys on ultrasound [25]. The term CGN referred to either NS or non-nephrotic CGN [26].

PUV is diagnosed with micturating cystourethrogram when there are dilatation and elongation of the posterior urethra with a linear radiolucent band corresponding to the valve [27].

Multicystic dysplastic kidney (MCDK) is diagnosed with ultrasound (USS) findings of a lobulated renal contour with multiple internal cysts of varying sizes and shapes; the renal parenchyma is usually fibrous and echogenic [28]. The cysts cluster and are non-communicating. It is usually confirmed as a non-excretory kidney on intravenous urography (IVU) [28].

Pelvic-ureteric junction obstruction (PUJO) refers to a dilated renal pelvis with a collapsed proximal ureter diagnosed with USS and IVU [29].

A duplex kidney is the kidney unit containing 2 pyelocaliceal systems that are accompanied by a single ureter or with double ureters confirmed with

IVU demonstration of two pyelocaliceal systems with double ureters [30].

A renal ectopia is described by an abnormal location of kidneys outside the flank region (L1-L3 vertebral levels) [30]. It includes both ectopic location of kidneys and abnormal fusion of whole or part of the kidneys diagnosed with USS confirming the ectopic kidney in the pelvic region [30].

A simple renal cyst is a pocket of fluid that originates from the surface of the kidney and is contained by a thin wall with the USS features of a simple cyst being an anechoic, thin imperceptible cystic wall, with no internal septations or debris [31].

Outcome measures of the CKD could be (a) death; (b) discharge against medical advice or loss to follow up before surgical intervention; (c) referral to other health facilities; (d) on follow-up at the paediatric nephrology clinic.

Statistical analysis

The study was summarized by simple descriptive analysis. The clinical features (gender characteristics, the aetiology of CKD, Stages of CKD, and clinical outcomes) were expressed in bar charts and frequency and percentages tables. A Chi-square test was used to test the age group distribution of the CKD cases. A Fisher exact test was applied to test the association between CKD cases and gender. The prevalence of CKD was calculated based on the counts per all the paediatric diagnoses and expressed as percentages. The incidence rate was expressed as per million-child population per year (pmcp/year). The population of children less than 19 years in FCT, Abuja was 651, 310 according to the available 2006 Nigeria's Population Census Commission. 32 *P* value < 0.05 was regarded as being statistically significant.

RESULTS

A total of 10,071 children were seen, out of which 324 were renal cases (a prevalence of renal case of 3.2%), and 94 were CKD (prevalence of CKD of 0.93% per paediatric cases and 29 % of renal cases). The incident rate per million-child population per year (pmcp)/year was 36.1. The CKD cases decreased significantly from 46.5% in 2018 to 14.9% in 2019 (p=0.001) but with a significant rise to 27.8% in 2020 (p=0.009). **Table 1.**

The 94 CKD cases comprised 71 males (75.5%) and 23 females (24.5%) with a male-to-female ratio of 3.1: 1 but this was not statistically significant (p=0.157). The age range was between 1 month and 16 years, with a mean age of 7.7 ± 4.1 years, and a majority 41 (43.6%) was between 5 and 10 years of age. **Figure 1.**

Concerning the pattern of the CKD, CGN constituted the majority (61/94, 64.9%) including 58 (95.1%) NS, and 3 (4.9%) non-nephrotic CGN. Of the 58 NS, 51 (87.9%) were Idiopathic NS and 7 (12.1%) were secondary NS comprising two with HIV infection and one each of hepatitis B, hepatitis C, sickle cell anaemia (haemoglobin SS), lupus nephritis, and congenital NS. Idiopathic NS accounted for 87.9% and infection-related secondary NS for 6.9% (4/58). The 3 non-nephrotic CGN included 2 idiopathic cases and one with hepatitis B infection.

CAKUT made up 35.1% (33/94) of the CKD cases, of which 14 were PUV, 7 PUJO, 5 MCDK, 4 ectopic kidneys, 2 left duplex kidneys and 1 with a right simple renal cyst. In summary, the common causes of CKD were NS (58, 61.7), PUV (14, 14.8%), and PUJO (7, 7.4%). There was no statistically significant gender predilection in the distribution of the CKD cases (p values =0.405 for chronic glomerulonephritis and p= 0.837 for CAKUT). **Table 2.**

Table 3 depicts the CKD Stages. The majority (36/94, 38.3%) were in GFR G2 and the least was in G3a (5/94, 5.3%). Eighty-three with CKD G1-G4 were without kidney replacement therapy. Eleven (11.7%) subjects presented in Stage 5 and had chronic intermittent haemodialysis (CIH). The duration of CIH was from 1 month to 6 months, with an average duration of 2 months before death from cardiovascular complications. The case fatality rate in the CKD Stage 5 was 90.9% (10/11). Only one (idiopathic NS) of the 11 Stage 5 cases who had renal transplantation was alive at the time of this study.

Table 4 shows the outcomes of CKD. Most of the mortality was accounted for by steroid-resistant idiopathic NS (5/11, 45.5%) and PUV (2/11, 18.2%). Mortality was also reported from one each of secondary NS including HIV, lupus nephritis and congenital NS. Thirty-seven idiopathic NS were still on follow-up. All the 3 non-nephrotic CGN were still on follow-up. Regarding the outcomes of the CAKUT, of the 14 with PUV, 10 had surgical valve ablation (Mohan's valvulotome ablation), 2 of whom died from uraemia and sepsis after the surgery, 2 were lost to follow-up before surgery could be done, and 4 of the

10 that had surgery were still on follow up for bladder dysfunction and kidney insufficiency. Of the 7 with PUJO, 6 had dismembered pyeloplasty (1 died after pyeloplasty from sepsis/uraemia), 1 patient was lost to follow-up before surgical correction, and 3 were on follow-up. All 5 cases of MCDK were non-excreting on IVU, and all had nephroureterectomy because of recurrent febrile UTI from severe VUR. The 5 were on follow-up as an acquired solitary kidney. The 4 patients with ectopic kidneys are on follow up including one who had nephrectomy for associated severe cystic hydronephrosis. The 2 with duplex kidneys had pyelorrhaphy and ureteroneocystostomy and were on follow-up. The child with a simple renal cyst was on follow-up in the clinic. In total, 24 subjects (72.7%) had surgery, death was reported in 3 cases (9.1%)-2 from PUV (66.7%) and 1 from PUJO(33.3%), 3 cases (9.1%) also were lost to follow-up before surgical intervention, while 20 subjects (60.6%) were on follow up in the clinic. In total, 13 (13.8%) CKD cases were lost to follow-up. **Table 4.**

The comparison of the present study with other studies in Nigeria was as shown in **Table 5.**

Table 1: Trend of cases during the study period

Year	Total paediatric admissions	Renal cases	Prevalence of renal cases(%)	Chronic kidney disease	Prevalence of chronic kidney disease per pediatric cases (%)	Prevalence of chronic kidney disease per renal cases (%)	P	P*
2017	2417	62	2.6	21	0.86	33.9	-	-
2018	2421	71	2.9	33	1.36	46.5	0.447	0.106
2019	3034	101	3.3	15	0.49	14.9	0.420	0.001
2020	2199	90	4.1	25	1.14	27.8	0.161	0.009
2017-2020	10, 071	324	3.2	94	0.93	29.0	-	-

P*= for renal cases,* ***P**** *=for chronic kidney diseases*

Table 2: Pattern of chronic kidney disease

Diagnosis*	Male	Female	Frequency (%)
1. Chronic glomerulonephritis			
A. Nephrotic syndrome			
Idiopathic nephrotic syndrome	40	11	51 (54.2)
Human immunodeficiency virus positive	1	1	2 (2.1)
Hepatitis B virus positive	1	-	1 (1.1)
Hepatitis C virus positive	-	1	1 (1.1)
Systemic lupus erythematosus	-	1	1 (1.1)
Sickle cell anaemia (Haemoglobin SS)	1	-	1 (1.1)
Congenital nephrotic syndrome	1	-	1 (1.1)
B. Non-Nephrotic syndrome			
Idiopathic	1	1	2 (2. 1)
Hepatitis B positive	1	-	1 (1.1)
2. Congenital anomalies of the kidneys and urinary tract			
Posterior urethral valve	14	-	14 (14.8)
Pelvic-ureteric junction obstruction	5	2	7 (7.3)
Multicystic dysplastic kidney	2	3	5 (5.3)
Ectopic kidney	3	1	4 (4.3)
Left duplex kidneys	1	1	2 (2.1)
Right simple renal cyst	-	1	1(1.1)
Total	71	23	94 (100)

**=Further description is in the text; For chronic glomerulonephritis, Fisher's exact test=0.637, P=0.405; For congenital anomalies of the kidneys and urinary tract, Fisher's exact test=1.648, p=0.837.*

Table 3: Staging of the chronic kidney disease

CKD Stage	Estimated glomerular filtration rate	Number (%)
Normal to increased GFR; G1	≥90	22 (23.4)
Mildly reduced GFR; G2	60-89	36 (38.3)
Moderately reduced GFR; G3a	45-59	5 (5.3)
Moderately reduced GFR; G3b	30-44	10 (10.6)
Severely reduced GFR; G4	15-29	10 (10.6)
Kidney failure (ESKD); G5*	<15	11 (11.7)

**11 had chronic intermittent haemodialysis, 1 of which later had renal transplantation; 83 CKD G1-G4 without kidney replacement therapy*

Table 4: Outcome of the chronic kidney disease

Diagnosis*	Discharge and on follow-up	Discharge and lost to follow-up	Surgical intervention	Surgical intervention and on follow-up	Left against medical advice/lost to follow-up before surgical intervention	Died
Chronic glomerulonephritis						
A. Nephrotic syndrome						
Idiopathic nephrotic syndrome	37	9	-	-	-	5
Human immunodeficiency virus positive	-	1	-	-		1
Hepatitis B viruspositive	1	-	-	-	-	-
Hepatitis C virus positive	1	-	-	-	-	-
Systemic lupus erythematosus	-	-	-	-	-	1
Sickle cell anaemia (Haemoglobin SS)	1	-	-	-	-	-
Congenital nephrotic syndrome	-	-	-	-	-	**1**
B. Non-Nephroticsyndrome						
Idiopathic	2	-	-	-	-	-
Hepatitis B virus positive	1	-	-	-	-	-
Congenital anomalies of the kidneys and urinary tract						
Posterior urethral valve	-	-	10	4	2	2
Pelvic-ureteric junction obstruction	-	-	6	3	1	1
Multicystic dysplastic kidney	-	-	5	5	-	-
Ectopic kidney	4	-	1	1	-	-
Left duplex kidney	2	-	2	2	-	-
Right simple renal cyst	1	-	-	-	-	-
Total (%)	50 (53.2)	10 (10.6)	24 (25.5)	15 (15.9)	3(3.2)	11(11.7)

**=Further description is in the text*

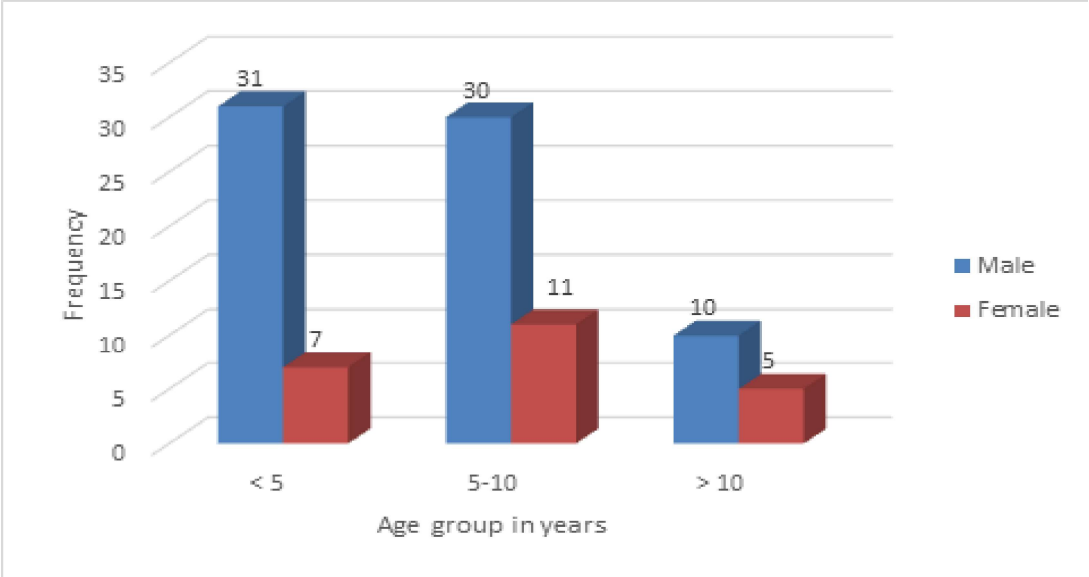

Figure 1: Legend. Age group distributions of the chronic kidney disease cases
Chi-square: 2.000, p-value: 0.157

DISCUSSION

The incidence rate of 36 pmcp/year in this hospital-based study was higher than the 1.7-11 pmcp/year earlier reported in Nigeria [15-18], probably agreeing with the increasing global burden of CKD [18]. Furthermore, in similar settings in Nigeria including Benin [15], Enugu [16], Port Harcourt [17], and Ile-Ife [18], the common causes of CKD in children were CGN and PUV, the findings similar to this study. CGN was also the commonest cause of childhood CKD in other developing countries including India, Southeast Asia, and South America [7-11]. Bhimma *et al*. in KwaZulu-Natal, South Africa [13], and Ali el-TM *et al*. in Sudan [14] also reported CGN to be the commonest cause of childhood CKD. However, in the United States and Europe, CAKUT and hereditary nephropathies are responsible for about two-thirds of all cases of CKD [4-6]. Causes of CKD tend to vary with age, genetics, race, and geographical location [2]. Specifically, the high prevalence of bacterial, viral, and parasitic infections may explain the predominance of CGN in sub-Saharan Africa and developing countries [16,18]. However, most of the CGN in this study were from Idiopathic NS, with infection-related secondary NS accounting for 6.9%. The success of childhood immunization campaigns may be leading to the reduction of infection-related glomerulonephritis [18]. In this study, the case of secondary NS from lupus nephritis is worthy of note as Olowu *et al*. in Ile-Ife, Nigeria, also reported lupus nephritis as the third commonest cause of CKD in their series [18].

Although a male predominance of CKD was found in this study and those of others [17,33] this male predominance could be attributed to the fact that PUV which occurs only in male children was also the major cause of CKD in this study.

In this study, most of the CKD cases were in the school-age group of 5 to 10 years (41, 43.6%) and in CKD Stage G2 (36, 38.3%), unfortunately, genetic testing that could detect more than 200 causative genes of the common aetiologic categories of CKD (CAKUT, steroid-resistant NS, CGN, and ciliopathies) in children less than 25 years [34], is not available in Nigeria for early screening of CKD among at-risk children. In addition, the contribution of APOL1 genetic mutation (noted for idiopathic focal segmental glomerulosclerosis CKD) is still not known among Nigerian children [35]. Contrariwise, in European countries, the diagnoses and treatment of CKD are commonly made during infancy [6]. The need for a deliberate prenatal and neonatal screening for CAKUT in Nigeria for early diagnosis and management cannot be overemphasized [18].

The management of CKD in children in our setting is still fraught with many challenges. The high

case fatality rate among those in ESKD requiring renal replacement therapy (RRT) in this study was related to financial constraints which prevented patients from assessing chronic intermittent haemodialysis (CIH) and renal transplantation (RT). The mean duration of time over which dialysis could be afforded was 2 months as payment was by out-of-pocket expenses. Arogundade in Ile-Ife Nigeria had also reported that only 5% of adults needing dialysis could afford it for more than 3 months [36]. Financial constraint was also the limiting factor against dialysis in other paediatric studies in Nigeria [15-18]. Sadly, Nigeria's national health insurance scheme (NHIS) caters for only 6 sessions of acute haemodialysis and the coverage is less than 5% of the population [20]. Financial challenges are also the reason for the loss of follow-up in this study and those of others [15,16]. The import of this reality suggests that preventive nephrology would be more beneficial to children in poor resource countries like Nigeria. The NHIS can be empowered to provide funds for CIH and RT among Nigerian children. Continuing medical education remains invaluable in increasing the diagnostic capacity of health workers at the primary and secondary levels of health care. A robust referral system to nephrologists at tertiary health facilities should also be pursued. This is especially true for CAKUT whose long-term prognosis may depend on early diagnosis and surgical intervention.

Education on renal protective measures should be publicized as a general measure.

Limitations of study

The pattern and the incidence of CKD described in this hospital-based retrospective study may not reflect the burden of CKD in this setting. True incidence might also have been underestimated because the incidence was based on the 2006 population of children in Abuja. We are also mindful of the unavailability of electron and immunofluorescence microscopy which limited the exact diagnosis in two of the post-infectious chronic glomerulonephritis.

CONCLUSION

This study indicates that CKD is commoner among school-age children and the commonest CKD was due to chronic glomerulonephritis (NS) and CAKUT (PUV). Financial constraints limit the accessibility of KRT among those with ESKD. The need for preventive nephrology and public-funded RRT cannot be over-underscored.

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