Childhood Acute Post-infectious Glomerulonephritis: A Study of Clinical Profile and Outcomes at the University of Abuja Teaching Hospital, Abuja, Nigeria, 2016 to 2021

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ABSTRACT

Background: Acute post-infectious glomerulonephritis (PIGN) is a common cause of childhood morbidity in developing countries, the burden of which reflects low socio-economic conditions and poor environmental and personal hygiene. In Nigeria, temporal and geographical variations exist for the epidemiology of PIGN but more information is needed.

Materials and Methods: A retrospective review of socio-demographic factors and clinical outcomes of childhood PIGN at the University of Abuja Teaching Hospital, Abuja, from January 2016 to December 2021.

Results: Out of 13,823 children, 506 were renal cases, and 55 were PIGN, with PIGN accounting for 10.9 % of renal cases. Among the 50 PIGN studied, 32 were males (64%), with a male-to-female ratio of 1.7: 1 ($p<0.009$). The age range was 4 to 15 years, with a median age of 8 years, and a majority (50%) were of the school-age group (5-10 years) ($p <0.0031$). Affected children were mostly (38, 76%) from low socio-economic households (p=Â 0.0001). The typical clinical features were passage of coke-coloured urine (100%), peripheral oedema (90%) and hypertension (90%); while the commonest laboratory features were haematuria (100%), proteinuria (94%) and decreased complement C3/normal C4 levels (30 of 30 subjects) Acute complications comprised acute kidney injury (56%), congestive heart failure (16%) and urinary tract infections (14%). A majority (92%) were discharged with a low case fatality rate of 4% (2 deaths).

Conclusion: PIGN is common in our setting but with a good clinical outcome. The need to improve the citizenry's socio-economic status and personal and environmental hygiene cannot be over-emphasized.

Keywords: *Acute post-infectious glomerulonephritis, children, clinical features, clinical outcomes, Abuja*

INTRODUCTION

Acute glomerulonephritis (AGN) is an abrupt onset of immune-mediated glomerular injury and inflammation that leads to a decline in glomerular filtration rate, hypertension, oedema, haematuria, and varying degrees of renal insufficiency [1]. AGN could arise as a primary renal disorder or could be secondary to systemic diseases [1]. Acute post-infectious glomerulonephritis (PIGN) is one of the most common causes of AGN in children, with post-streptococcal glomerulonephritis (PSGN) being the archetype of PIGN [1]. However, AGN, PSGN and PIGN have been used interchangeably [2]. Worldwide, the estimated incidence of PIGN is 472,000 cases per year with a majority (77%) in low- and middle-income countries (LMIC) [3]. Whereas improvement in socio-economic status (SES) and personal hygiene,

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and the widespread use of antibiotics have cumulatively reduced the incidence of PSGN in high-income countries [1]; the burden of PSGN remains high in LMIC with an estimated incidence of >200 cases/million population/year [4]. The clinical manifestation of PIGN varies from mild asymptomatic cases to severe acute kidney injury (AKI) requiring dialysis support and/or paediatric intensive care admission [1]. In the tropics, PSGN is mainly caused by skin infection, usually scabies, with a secondary streptococcal infection which contrasts to the temperate area where infection of the upper respiratory tract is the main cause [5]. Intra-familial transmission of scabies in low SES households with the sub-optimal hygienic condition is particularly noted in the tropics [5]. Although the prognosis of PSGN is generally good, with 95% of patients making a full recovery, and with most clinical symptoms resolving spontaneously within 2 – 3 weeks after onset [6]; progression to chronic glomerulonephritis in 1-3 % of paediatric patients, and mortality of up to 7% has been reported in AGN in children [7].

In Nigeria, temporal and geographical variations exist in the epidemiology of AGN, probably reflecting different and changing climatic and socioeconomic conditions [8-17]. Researchers have also subsumed reports on AGN in studies that described the pattern of kidney disorders and AKI [18-23]; while the opportunity for a detailed epidemiological description of AGN was missed.

However, the epidemiology of AGN/PIGN in different settings allows for focused and appropriate preventive measures. Thus, the aims of this study are to describe the socio-demographic and the clinical outcomes of PIGN among children attending the University of Abuja Teaching Hospital (UATH), Abuja, Nigeria, from January 2016 to December 2021.

MATERIALS AND METHODS

The study was a retrospective review of data of children aged 4-15 years with PIGN who received care and treatment at the UATH, Abuja, from January 2016 to December 2021. Ethical consideration followed the Helsinki Declaration of 1975, as revised in 1983. Permission to use the data was obtained from the Research and Ethics Committee of the UATH, Abuja.

Abuja experiences two weather conditions annually [24]. This includes a warm, humid rainy season and a harsh dry season [24]. The dry season spans from November to March [24].

The information abstracted from patients' records for this study included socio-demographic data, the clinical presentations of the PIGN and the month of diagnosis. Other data were the investigations' results of the dipstick urinalysis, urine sediment microscopy, urine culture, culture of the throat swab, spot urine protein: creatinine ratio, plasma albumin and protein, fasting serum triglyceride/cholesterol levels, anti-streptolysin O titre (ASOT), serum complements C3/C4, serum electrolytes, urea and creatinine, full blood count, and renal ultrasound scans. Glomerular filtration rate (eGFR) was also estimated from serum creatinine by Schwartz formula for each patient. Serum IgA levels, antinuclear antibodies (ANA) were usually requested for subjects with suspected Henoch Schonlein Purpura (HSP) and systemic lupus erythematosus respectively. On one occasion, antinuclear cytoplamsic antibody was specifically requested to rule out granulomatous polyangitis (GPA) in an adolescent girl who presented wth historical chronic otitis media, arthritis, nose bleed, frothy blood stained sputum, and acute kidney disease (AKI). Historical review on clinical presentation for all subjects included recent throat or skin infection (up to 6 weeks previously), previous episodes of macroscopic haematuria (IgA, Alport syndrome, membranoproliferative glomerulonephritis (MPGN), SLE, ANCA positive vasculitis), joint pains and swelling (HSP, SLE, ANCA positive vasculitis) and family history of chronic kidney disease (CKD) or deafness (Alport syndrome).

Definitions

The following definitions apply to the study:

The diagnosis of **AGN** was based on the presence of clinical features and relevant laboratory results which comprised sudden onset of haematuria, hypertension, oedema, oliguria, proteinuria and varying degrees of renal insufficiency [17-25].

The diagnosis of **PIGN** was based on the presence of features of AGN and the absence of clinical or laboratory features suggestive of systemic non-infectious conditions such as connective tissue disorders or vasculitides [17].

Rapidly progressive glomerulonephritis (RPGN) "is a type of nephritic syndrome, is a pathologic diagnosis accompanied by extensive glomerular crescent formation (i.e. > 50% of sampled glomeruli contain crescents which can be seen in a biopsy specimen) that, if untreated, progresses to end-stage renal disease over weeks to months"[26] RPGN is a pathologic diagnosis [26].

The diagnosis of **haematuria** on dipstick urinalysis is graded semi-quantitatively as negative, trace, 1+, 2+, 3+, and 4+ [27]. Mild haematuria is a trace on dipstick urinalysis [27].

Hypertension was defined as systolic and or diastolic blood pressure (BP) $\geq$ the 95th centile for age, gender and length using a normogram published in the fourth report of the National High Blood Pressure Education Program [28]. BP less than 90th percentile is normal. BP between the 90th and 95th percentile is prehypertension [28]. In adolescents, BP equal to or exceeding 120/80 mmHg is prehypertension, even if this figure is less than the 90th percentile [28]. Stage I is BP 95th percentile to the 99th percentile plus 5 mmHg [28]. Stage 2 is BP greater than 99th percentile plus 5 mmHg [28]. For this study, Stage I is regarded as a mild BP.

Socioeconomic status (SES) stratification was done using the one proposed by Oyedeji which employs the educational status and occupation of parents [29]. SES was classified into high, medium and low based on the occupation and level of education of the parents.

AKI and its severity were as described by pRIFLE criteria using urine output and serum creatinine estimation measured by Jaffe's method [30]. Although AKI was diagnosed at hospital admission, the maximum serum creatinine level reached in each patient on or before post-admission Day 7 was used for the final AKI severity as per Injury or Failure. Estimation of glomerular filtration rate (eGFR) was by Schwartz's formula and adequacy of urinary output was based on the body weight [30]. AKI/risk was defined as a decrease of at least 25% in eGFR and/or a urine output of <0.5 mL/kg/h for >8 h [31]. The injury was a decrease of at least 50% in eGFR and/or a urine output of <0.5 mL/kg/h for 16 h [31]. Failure was a decrease of at least 75% in eGFR or eGFR <35 mL/min/ 1.73 m2 and/or urine output <0.3 mL/kg/h for 24 h or if the child was anuric for 12 h [31]. It is important to emphasize that this is a description of children with PIGN coming to UATH for the first time, therefore, their baseline serum creatinine was unknown, and a baseline GFR of 120 mL/min/1.73 m2 was assumed [18, 31].

Management of PIGN

Management was often supportive as PIGN usually resolves spontaneously [32]. Hospital admissions were necessary for those with AKI, fluid overload/ congestive heart failure, hypertension or electrolyte imbalance [32]. No added salt was observed [32]. Close monitoring for fluid input and urine output were done and patients with oliguria had fluid restricted to replacement of insensible losses (400 ml/m^2/day) plus previous days' urine output, while those with fluid overload/hypertension/oedema had frusemide to induce a negative fluid balance. Hypertension in euvolemic patients was treated with nifedipine or amlodipine. Those who developed AKI and/or nephrotic syndrome were treated accordingly as previously described [33,34]. Oral phenoxymethyl penicillin was given to prevent the horizontal spread of nephritogenic strains of group A streptococcus to household members and those in close contact [34].

Statistical analysis

A simple description of the clinico-sociodemographic features of the PIGN was done. Subjects' characteristics were summarized using medians (QQ plot showed that subjects' age in years and the duration of admission in days were not normally distributed). The proportions of the categorical variables were compared using Chi-Square for age groups and gender, and Fisher's exact test for SES. A P-value less than 0.05 was considered significant. Statistical analysis was done with the SPSS version 20 (IBM SPSS Statistics for Windows, version 20, IBM Corp., Armonk, N.Y., USA).

RESULTS

Over 6 years, from 2016 to 2021, a total of 13,823 children were seen, of which 506 (3.6%) were renal cases, and 55 were PIGN cases. Post infectious glomerulonephritis accounted for 0.4% of paediatric cases, and 10.9 % of renal cases. The clinical details of 5 subjects were missing, and the studied cases were limited to 50. No subject had features of vasculitides or Alport syndrome. **Table 1** describes the sociodemographic characteristics of 50 subjects

with PIGN. The 50 patients with PIGN comprised 32 males (64%) and 18 females (36%) with a male-to-female ratio of 1.7: 1 (p=0.009). The age range of the patients with PIGN was 4 to 15 years, with a median age of 8 years, and a majority (25, 50%) within the school-age group (5-10 years), p=0.0031. PIGN was diagnosed in 48 (96%) children from low and medium socioeconomic class (SEC) and was also significantly more among subjects in low SEC (38, 76%) compared to those in medium and high SEC (p=<0.0001). Other characteristics were as shown in Table 1.

Table 1: Sociodemographic characteristics of subjects with acute post-infectious glomerulonephritis

Characteristics	Number	%
Age groups in years[μ]		
< 5	9	18
5-10	25	50
>10	16	32
Gender §		
Male	32	64
Female	18	36
Socioeconomic status [γ]		
Low	38	76
Medium	10	20
High	2	4
Ethnicity		
Fulani	11	22
Hausa	10	20
Gbagi	10	20
Yoruba	5	10
Ibo	5	10
Others		
Bassa	4	8
Ebira	3	6
Tiv	2	4
Age range in years	4-15	-
Median age in years	8	-
Interquartile range in years	7	-
Range of duration of admission in days	5-21	-
Median duration of admission in days(IQR)	12 (5)	-

μ*Chi-square test 11.58, p=0.0031;* ***§*** *Chi-square test 6.76, p=0.009;* [γ]*Fisher's exact test 64.32, p=> 0.0001,*

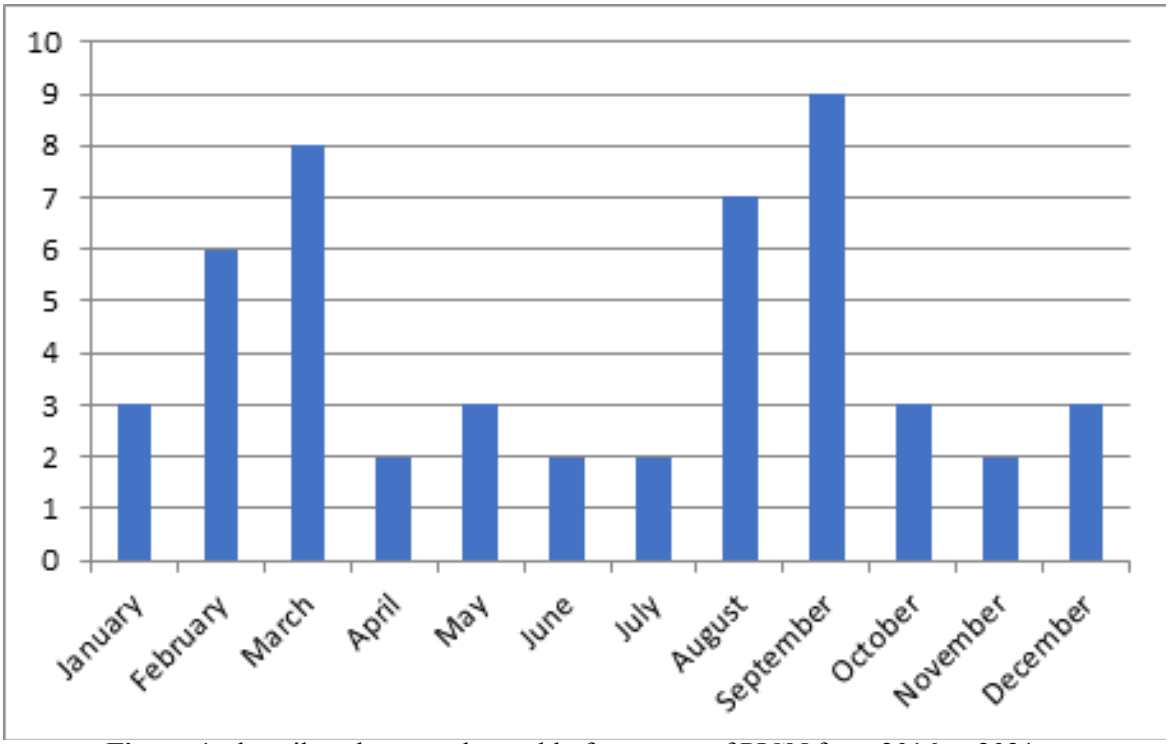

Figure 1: describes the annual monthly frequency of PIGN from 2016 to 2021.

Table 2 depicts the trends of the PIGN from 2016 to 2021. The PIGN cases seen increased progressively from 5 in 2016 to 15 in 2021 with an annual average of 9 cases.

Table 3 shows the clinical and laboratory features of PIGN at presentation. Passage of coke-coloured urine was seen in all subjects (100%); this was followed by oedema in 45 (90%), and hypertension in 43 (86%). Pyoderma was seen in 32 (64%) and throat infection in 18 (36%). The classical low C4 levels were seen in all the 30 (100%) subjects who were able to do the test. Haematuria (50, 100%) and proteinuria (47, 94%) were other prominent laboratory features. Red cell casts were seen in 19 (38%) subjects only. The adolescent girl who had a suspicion of granulomatous polyangitis (GPA) because she presented with historical chronic otitis media, arthritis, nose bleed, frothy blood stained sputum, and acute kidney disease (AKI) had c-ANCA negative result from a private laboratory thereby, excluding the diagnosis of GPA. In addition, her nose bleed and the frothy blood-stained sputum later resolved with haemodialysis. *Streptococcus pyogenes* was the commonest organism isolated from 30 (60%) subjects with 18 (36%) from the throat, 6 (12%) from pyoderma and 6 (12%) from the blood. *Staphylococcus aureus* was isolated from 10 (20%) subjects (20%) with 7 (14%) from the throat and 3(6%) from pyoderma. *Salmonella typhi* was cultured from the blood of 8 (16%) subjects. Others were Hepatitis B (6%), Hepatitis C (6%) and HIV (4%). Other clinical features were as highlighted in Table 3.

Table 4 shows the complications and outcomes among the 50 PIGN cases. AKI was noted in 28 (56%) subjects; 4 (14.3%) with Risk, 6 (21.4%) with Injury, and 18 (64.3%) in Failure categories. Three (6%) subjects with increasing proteinuria ultimately evolved into nephrotic syndrome. One (2%) subject with features in keeping with RPGN also developed AKI rapidly. Haemodialysis was provided for 19 (38%) children including 18 of the AKI (in Failure) and the child with suspected RPGN. Regarding outcomes of management, 46 (92%) were discharged, 2 (4%) left against medical advice, and two children died from AKI (a case fatality rate of 4%), one of whom was the suspected RPGN. At three months of follow-up, 10 (10/46, 21.7%) had already been lost to follow-up. Among those in follow-up, the acute glomerulonephritis seemed to have resolved in 80.5% (29/36), while 19.4% (7/36) still had mild haematuria and mild hypertension.

Table 2. Trend of post-infectious glomerulonephritis during the study period

Year	Total paediatric admissions	Renal cases	Prevalence (%)	Annual prevalence of renalcases per 1000 children	Acute post-infectious glomerulonephritis (PIGN)	Prevalence of PIGN per paediatric cases (%)	Prevalence of PIGN per renal cases (%)	Annual prevalence of PIGNcases per 1000 children
2016	1252	67	5.4	54	5	0.39	7.5	75
2017	2417	62	2.6	26	5	0.21	8.1	81
2018	2421	71	2.9	29	7	0.29	9.9	99
2019	3034	101	3.3	33	10	0.33	9.9	99
2020	2199	90	4.1	41	13	0.59	14.4	144
2021	2500	115	4.6	46	15	0.60	13.0	130
2016-2021	13,823	506	3.6	36	55	0.39	10.9	109

Table 3: Clinical and laboratory features of post-infectious glomerulonephritis at presentation

Clinical features*	**Frequency in decreasing order (%)**
Passage of coke-coloured urine	50 (100)
Peripheral oedema	45 (90)
Hypertension	43 (86)
Pallor	40 (80)
Pyoderma	32 (64)
Fever	30 (60)
Diarrhoea	25 (50)
Oligoanuria	23 (46)
Headache	20 (40)
Throat infection	18 (36)
Abdominal pain	15 (30)
Vomiting	10 (20)
Cough/Breathlessness	8 (16)
Malaise	7 (14)
Lethargy	7 (14)
Convulsion	4 (8)
Loss of consciousness	4 (8)
Laboratory parameters	
Depressed C3/Normal C4 (done in 30 subjects)	30(100)
Haematuria-(dipstick)	50(100)
Proteinuria (dipstick)	47 (94)
Anaemia	35 (70)
Urinary red blood cell casts	19 (38)
Leucocyturia (dipstick)	7 (21)
Renal ultrasound scans (50)	
Normal	5 (10)
Grade I parenchymal disease	15 (30)
Grade II parenchymal disease	25 (50)
Grade III parenchymal disease	5 (10)

**= Multiple responses possible; *Streptococcus pyogenes (Elevated ASO titre, > 200 Todds units) (30, 60%), Staphylococcus aureus (10, 20%), Salmonella typhi (8, 16%), Hepatitis B (3, 6%), Hepatitis C(3, 6%), Human immunodeficiency virus (2, 4%)*

Table 4: Complications and outcomes of post-infectious glomerulonephritis

Features	Number	%
Complications		
Acute kidney injury	28	56
Congestive heart failure/hypervolaemia	8	16
Urinary tract infection	7	14
Hypertensive encephalopathy	4	8
Nephrotic syndrome	3	6
?Rapidly progressive glomerulonephritis	1	2
Outcome		
Discharged	46	92
Left against medical advice (LAMA)	2	4
Death while on admission	2	4
Loss to follow-up at 3 months	10	21.7
Resolution of acute nephritis (proteinuria, haematuria, hypertension, and normal eGFR) among 36 patients at 3 months	29	80.5
Persistent mild haematuria and mild hypertension at 3 months of follow-up	7	19.4

eGFR=estimated glomerular filtration rate

DISCUSSION

We reviewed 50 cases of PIGN seen in our hospital over a 6-year period. There was an average of 9 cases per annum with an increasing number of cases from 5 in 2016 to 15 in 2021. Almost all patients are from low to medium socioeconomic class. The commonest cause was *Streptococous pyogenes*. The commonest modes of presentation were passage of coke-coloured urine, peripheral oedema and hypertension. Common complications were AKI, congestive heart failure and urinary tract infection. Haemodialysis was carried out in 19 subjects (19%). Most patients survived till discharge. A few patients (19.4%) had persisting hypertension and haematuria at 3 months post – discharge. Patients with persisting hypertension and haematuria 3 months-post-discharge were specifically on further closer follow-up in the nephrology clinic to monitor for a potential complication of chronic glomerulonephritis and CKD. In the same setting, we had earlier reported 13 cases of AGN over a 4 -year period, from 2013 and 2016, with an annual average of 3 cases [22].

The increasing number of cases of PIGN seen over 6 years in this study would suggest that there is no lessening of poverty over the period of study in our setting. Arguably, households have continued to live in poor housing conditions, with overcrowding and poor personal and environmental hygiene, which tended to sustain familial and communal spread of infections that caused PIGN. Apparently, a meagre increase in the minimum monthly wage of Nigerian workers from eighteen thousand Naira (N18, 000 ≈ $43) to thirty thousand Naira (N30,000 ≈ 72) over the last decade could not have translated to alleviation of poverty by no means.

Acute PIGN was responsible for 10.9% renal admissions in this study which was higher than the 8.9% reported in Ibadan [17]. However, in similar studies done elsewhere in Nigeria, the 10.9% in this study was lower than the 11.4% from Port-Harcourt [35] and much lower than the 38.1% from Zaria [19], 37.7% from Jos [20], 31.9% from Enugu [36], 24.8% from Kano [23], 20% from Benin [37] and 24% from Gausau [21]. We argue that the different burden of PIGN in different cities in Nigeria reflects different health seeking behaviours for infectious diseases as well as the different prevalent climatic and socioeconomic conditions. In actual fact, and unlike in high-income countries where improvement in socioeconomic conditions and personal hygiene have reduced the incidence of PSGN[1]; this study and other Nigerian studies [13,17], and those in India [38] and Ethiopia [39] have shown that patients with PIGN were mostly from households with low and medium

socioeconomic conditions. This study revealed that 96 % of patients with PIGN were from low or medium SEC. The finding that 96% of children with PIGN were from low or medium SEC compares to the 90% of children with PIGN in the study of Asinobi *et al* [17] in Ibadan, southwest Nigeria and the 93.8% in the study of Etuk *et al* [13] in Calabar, south-south Nigeria where most childhood sufferers of AGN belonged to the middle and low socioeconomic classes. Ibadin and Abiodun [11] in Benin, south-south Nigeria also reported that 90% of the patients with AGN came from low socio-economic class families. As argued previously, overcrowding, poor ventilation, poor personal hygiene that favour the horizontal transmission of nephritogenic strains of *Streptococcal* skin and scabies infection, delayed treatment of streptococcal pharyngitis are prerequisites that are sustained by low socioeconomic status [1,5,11,40].

In this study, the annual surge of PIGN noticed during the peak of the dry season in January-March and during the wettest months of August-September probably corresponds to the respective shortage of water that favour skin infections and the cold weather that favour pharyngitis. Etuk *et al* [13] also reported two annual peak periods in May-July and October-January representing peaks in the middle of dry and rainy seasons of the year. Similar dual annual peaks were reported by Okafor *et al* [10] in Enugu, southeast Nigeria and Ibeneme *et al* [16] in Umuahia, also in southeast Nigeria. However, Asinobi *et al* [17] reported the annual peak incidence of PIGN between June and December and the lowest incidence in March and April.

Regarding the epidemiology, as it relates to the age of occurrence of PIGN, this study found that children within the school-age group of 5-10 were significantly more at risk of PIGN at a median age of 8 years. This finding is similar to the peak age incidence of between 5 and 9 years reported by Asinobi *et al* [17] and > 5 years to 10 years by Ugwu [15] in Oghara, southern Nigeria and that of 5-10 years by Ibeneme *et al* [16]. Our study, however, differed from the observation of Olowu [12] who reported that the majority of AGN occurred in children below 6 years of age with a peak age incidence of 3 years. Ibadin and Abiodun [11] also reported a peak age incidence of 3 years. Although PIGN can occur at any age including infancy, most cases occur in school-aged children from 5-15 years [7], the age group which also accounts for the greatest overall number of *Streptococcal* pharyngitis [41].

This study also supports a male predilection to PIGN. Similar findings have been reported by other authors in Nigeria [8-17] and elsewhere [38,39]. However, Ibadin and Abiodun [11] as well as Jiya *et al* [42] in Sokoto have reported female preponderance of PIGN in their studies. Generally, the reason for male preponderance in PIGN is unknown [7].

About preceding infections, this study attributed skin infections (64%) to be most responsible for the PIGN cases. This finding is in keeping with other Nigerian studies [8,10,11,13,16,17] that reported pyoderma to be commoner than pharyngitis/tonsillitis. It also agreed with an earlier assertion that pyoderma-associated PIGN is commoner in the tropics and subtropics [5]. Contrariwise, Anochie *et al* [14] in Port Harcourt, south-south Nigeria, Bhalla *et al* [38] in North India and Gebreyesus *et al* [39] in North Ethiopia found pharyngitis-associated APIGN to be more than those from pyoderma. Expectedly, *Streptococcus pyogenes* was responsible for most (60%) PIGN cases in this study. Other infectious agents were *Staphylococcus aureus* (20%), *Salmonella typhi* (16%), Hepatitis B (6%), Hepatitis C (6%) and HIV (4%). This finding of APSGN predominance agrees with that of Asinobi *et al* [17] and by far in most childhood PIGN as epidemiologically described [2]. Asinobi *et al* [17] also identified Hepatitis B (6%), mumps (2%), varicella (2%) and diphtheria (2%) to be the non-streptococcal infectious agents responsible for PIGN in their study. However, in the 1980s, Aikhionbare and Abdurraham [8] in Zaria northern Nigeria reported Hepatitis B to be responsible for 41% of PIGN cases. Undoubtedly, the role of vaccination against identified infectious diseases as an indirect control measure of PIGN cannot be over-emphasized [17].

This study revealed the commonest clinical features of PIGN to include passage of coke-coloured urine (100%), haematuria (100%), depressed C3/ normal C4 complement levels (100%), proteinuria (94%), oedema (90%), hypertension (86%), anaemia (70%), oligo-anuria (46%) and red blood cell casts in 38% (low incidence probably reflecting the fact that the urine samples examined were not fresh). This study also identified AKI (56%), congestive heart failure (16%), UTI (14%), NS (6%) and a suspected RPGN (2%) as the acute complications of PIGN. These clinical features and the acute complications

are explainable consequent to the understanding of the pathophysiology of PIGN; whereby, glomerular injury follows glomerular deposition or in situ formation of immune complexes [1,7]. Both nephritis-associated plasmin receptor (NAPlr) and streptococcal pyrogenic exotoxin B (SPeB) from *Streptococcus pyogenes* activate the alternative complement pathway resulting in low serum complement levels [1,7]. The disruption of the glomerular filtration barriers is accompanied by varying degrees of haematuria/anaemia, leucocyturia, proteinuria, and active urine sediment with red blood cells and red blood cell casts [1,7]. The decreased glomerular filtration rate and the avid distal nephron salt and water retention result in varying degrees of intravascular volume expansion, oedema, oliguria and systemic hypertension [1,7]. Unexpectedly, the clinical features and the acute complications of PIGN noticed in this study were similar but vary differently in burden from other studies [8-17,37]. For examples, Asinobi *et al* [17] reported oedema (92%), hypertension (86%), oliguria (28%) and pallor (26%) as clinical features and documented RPGN (12%), heart failure/ pulmonary oedema (10%), UTI (10%) and hypertensive encephalopathy (8%) as complications of AGN. Etuk *et al* [13] found haematuria (100%), proteinuria (100%), oedema (98.5%), hypertension (86.8%), oliguria (83.4%) and granular casts (47.1%) and reported AKI (5.9%), hypertensive encephalopathy (4.4%) and congestive heart failure (2.9%) as acute complications. Ibadin and Abiodun [11] documented oedema (93.7%), hypertension (82.5%), oliguria (47.6%) and pulmonary oedema (39.7%) and reported complications to include congestive heart failure (39.7%), UTI (20.6%), AKI (12.7%) and hypertensive encephalopathy (4.8%). The typical features of oedema and hypertension were also the commonest presentation of Jiya *et al* [42] and Ibeneme *et al* [16]. We have identified AKI as the commonest complication of PIGN in this study. However, mortality from it was minimal as haemodialysis support was provided as needed. This may not be the case in all settings as an inability to provide dialysis support has been adduced to a higher case fatality among AGN with AKI in other centres [11,42].

Notably, the tendency for increased susceptibility to UTI in children with AGN deserves further exploration as UTI was also reported in 26.8% and 47.4% of AGN series by Jiya *et al* [42] and Ibeneme *et al* [16].

Outside Nigeria, Bhalla *et al* [38] in north India reported hypertension of 80%, AKI (40.4%), pulmonary oedema (32%) and hypertensive encephalopathy (12%) as features of AGN in their series.

While this study and those of others [8-17,38,42] may represent the typical acute nephritic syndrome of PIGN, it is important to note that atypical presentation with sub-clinical disease or acute illness related to hypertension or oedema in the absence of overtly abnormal urine sediment is possible [1]. We also suspect atypical presentation in a child who presented with a nephritic-nephrotic feature, and who deteriorated with a rapid decline in GFR to 15 ml/ min/m2 within 72 hours (with no history, anthropometry and renal ultrasound suggestive of CKD or systemic illness) to be a case of RPGN. The child died after a session of haemodialysis and before a renal biopsy could be done. However, we are unsure if the child would have survived if pulse corticosteroid was given earlier at presentation. Asinobi *et al* [17] had earlier reported a prevalence of RPGN of 12% with the RPGN cases accounting for 62.5% of children that had dialysis and also responsible for 80% of mortality in their series. Asinobi *et al* [17] also confirmed glomerular crescent formation on renal biopsies in 2 of the 6 (33.3%) children with RPGN. The pathogenesis that could explain the worse clinical course of RPGN has been linked to the possibilities of different nephritogenic strains and virulence factors of streptococcus [17]. Undoubtedly, a high index of suspicion for early diagnosis of RPGN is warranted as prompt treatment with pulse corticosteroids, with or without other immuno suppressive therapies and renal replacement therapy could reduce the morbidly and mortality from it [17].

Generally, the clinical course of PIGN in this study was benign. Most (92%) cases were discharged after successful hospital admission which spanned 5-21 days, with a median of 12 days. The case fatality rate (CFR) was also low with 2 deaths (4%) arising from AKI-related complications including the child with suspected RPGN. Similarly, low case fatality rates of between 1.4% and 9.7% were reported by other authors [10,8,11,17,13,16,14] in Nigeria and a case fatality rate of 6% in Ethiopia

[41]. While the CFR of 4% in this study was higher than the respective 1.4%, 1.5%, and 3.2% in Enugu [10], Zaria [8] and Benin [11]; it was lower than the respective 4.3%, 5%, 5.3% and 9.7% in Ibadan [17], Calabar [13], Umuahia [16] and Port Harcourt [14]. In this study, although the follow-up period reported was short at 3 months, a majority(80.5%) had already resolved the acute nephritic syndrome with 19.4% still having mild haematuria and mild hypertension.

CONCLUSION

This study would sum to indicate that PIGN is common in our setting but with a good clinical outcome. It is a disease of school-age children who lived in low socioeconomic households**.** It has annual double peak prevalence. The need to improve citizenry`s socio-economic status and personal and environmental hygiene and the prompt treatment of streptococcal infections cannot be over-emphasized.

Limitation of study

This study is limited by its retrospective design. We might have under-reported the true incidence of PIGN in our setting as milder and transient cases may not have presented to UATH. While the records of 5 children were missing, we could not also determine the C3/C4 levels in some other 20 children. In addition, we missed the opportunity of renal biopsy in the child with suspected RPGN. Furthermore, the follow-up period of 3 months was short and loss to follow-up was already at 21.7%, thus, precluding the knowledge of the outcomes of PIGN in this group.

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Authors' contribution

Anigilaje EA participated in the conceptualization and design of the study. Angilaje EA and Elike CM participated in data abstraction, data interpretation and in drafting and critical revision for important intellectual content. Both authors approved the final version of the manuscript to be published

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