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Childhood acute glomerulonephritis in Ibadan Nigeria

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Abstract: *Background:* Acute glomerulonephritis (AGN) is an important cause of mortality and morbidity in children in developing countries while its incidence has declined in the developed world. This study was to document its present impact in our setting.
Aim: To evaluate the sociodemographic features and clinical outcomes in children hospitalized for AGN in the Paediatric Nephrology Unit of the University College Hospital, Ibadan.
Subjects and Methods: This was a descriptive analytical study of children aged 2 to 14 years admitted with AGN between 2007 and 2019. Their sociodemographic data, clinical features, complications and outcome were analysed.
Results: AGN accounted for 116 (8.9%) of new renal cases admitted over the period. Seventy-four (63.8%) were male, mean age (SD) was 8.2 (3.3) years and peak age incidence between 5 and 9 years. Average annual hospital incidence rose from 4-5 new cases to 8-9 new cases/annum with an upsurge to 17 cases in 2019. Highest yearly monthly incidence was between June and December. Forty -five out of 50 (90%) evaluated subjects were in the middle/ low socioeconomic classes. Stage 2 hypertension occurred in 30/50 (60%) with hypertensive crises in 24%. RPGN occurred in 6/50 (12%) of cases accounting for 5 of the 8 dialysed patients and 4 of the 5 deaths. The case fatality rate was 4.3%.
Conclusion: The study showed a progressive increase in the hospital incidence of AGN.RPGN was a major risk factor for death in children with AGN and therefore requires a high index of suspicion and an appropriate early intervention.

Key words: Childhood, Acute Glomerulonephritis (AGN), Post-infectious AGN, Rapidly progressive glomerulonephritis (RPGN), Hypertensive crises, Outcome

Introduction

Acute glomerulonephritis (AGN) is one of the oldest and most common non-suppurative renal disease conditions seen in childhood. It is a common cause of childhood morbidity and mortality in low and middle income countries (LMICs)[1-10] AGN could arise as a primary renal disorder,[11,12] or could be secondary to a systemic disease.[13] Acute post-streptococcal glomerulonephritis (APSGN) is the prototype of AGN but a sub-set of acute post-infectious glomerulonephritis (APIGN) which is mostly caused by group A β -hemolytic streptococcus, but could be caused by any infectious agent (bacteria, virus, fungal, parasite). AGN, APSGN and APIGN many times are used interchangeably.[14] Acute post-streptococcal GN [APSGN] is the most common form of glomerulonephritis (GN) encountered in children and the World Health Organisation (WHO) estimates that 472,000 patients are affected globally with APSGN each year, and results in 5,000 deaths annually.[15] Childhood APIGN has become less prevalent in developed countries because of improved environmental hygiene, socioeconomic status and widespread use of antibiotics.[1] The elderly are presently the most affected group of individuals in developed countries and staphylococcal infection is more commonly incriminated in them.[16] It has indeed been stated that APIGN in high-income countries is predominantly a disease of the adults with risk factors inclusive of diabetes mellitus, malignancy, alcoholism, human immunodeficiency viral (HIV) infection and intravenous drug use.[11,16] The epidemiology of APIGN therefore varies across countries;[1,4,5,8,17,18] and in Nigeria has varied between different geographical locations and may be higher than usually documented as the clinical presentation of APIGN varies from a benign asymptomatic condition (not requiring hospital management) to rapidly progressive glomerulonephritis requiring dialysis.[11] In most parts of the developing world, Nigeria inclusive, paediatric cases of AGN are largely associated with streptococcal infection of the throat and skin, a situation closely linked to low socio-economic status and poor environmental hygiene.[4,8,18] Even though re-

corded mortalities may not be high, life threatening complications do arise if diagnosis is delayed and inaccurate treatment given.[17] This study aimed to describe the sociodemographic variables and clinical outcomes of APIGN among children seen at the University College Hospital (UCH), Ibadan, Nigeria. It was hoped that the findings will inform appropriate recommendation of preventive strategies to health policy makers.

Materials and methods

This was a cross-sectional descriptive study of children aged between 2 and 14 years who were admitted into the Paediatric Nephrology Unit (PNU) of the University College Hospital, (UCH) Ibadan with features of AGN. The diagnosis of AGN was based on the presence of clinical features and relevant laboratory results which comprised sudden onset of features of glomerular injury viz haematuria, hypertension, oedema, oliguria, proteinuria and varying degrees of renal insufficiency.[19] The diagnosis of acute post-infectious glomerulonephritis was based on the presence of features of acute glomerulonephritis, and the absence of clinical or laboratory features suggestive of systemic non-infectious conditions such as connective tissue disorders or vasculitides. Other renal diagnoses were based on the KDIGO recommendations.[20] Hypertension was defined and staged according to the Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents 2017.[21] The diagnosis of RPGN in this study was based on an acute presentation with a nephritic picture and rapid decline in glomerular filtration rate over days to weeks[2] in a child whose anthropometry and renal ultrasound features did not suggest a chronic illness, with or without renal biopsy evidence.

The following data which included age, sex, date of admission, presenting complaints, parental educational level and occupation were obtained. Each child had a detailed physical examination and the findings were documented. All the children were followed up till discharge or in-hospital death, need for dialysis and the outcomes were recorded. Urinalysis, urine microscopy and culture; serum urea, electrolyte and creatinine, and serum albumin, full blood count, serum lipids, Anti – streptolysin O (ASO) titre, spot urine protein creatinine ratio were determine and abdominal ultrasonography was performed. In addition, all the children had screening for human immunodeficiency virus (HIV) infection, Hepatitis B and C infection and malaria. All had their haemoglobin genotype determined by electrophoresis. Serum complements C3 and C4, anti-deoxyribonuclease B titres and anti-neutrophil cytoplasmic antibody (ANCA) were not routine but done to rule out systemic disorders when necessary because of costs.

The socio-economic classification and clinic-laboratory analyses were performed on the last 50 patients seen between 2015 and 2019. The social class stratification of the children's parents was determined using the method by Olusanya *et al.*[23] Renal biopsies were not routinely done. Data entry and statistical analysis were performed using IBM Statistical Package for Social Sciences (SPSS) ™ version 22.0 for Windows. Simple statistics of proportions and percentages were employed in the analysis with continuous data represented as mean or mode as appropriate while categorical data were presented as percentages.

Results

A total of 116 children were confirmed to have acute glomerulonephritis between January 2007 and December 2019, a 13-year period, out of a total of 1,303 children admitted newly with primary renal disorders. Of the 116 children, 74 (63.8%) were male, with a male to female ratio of 1.8:1. The mean age (SD) of these children was 8.2±3.3 years. The peak age incidence was between 5 to 9 years (Table1) with the youngest aged 2 years and the oldest 14 years old.

With regards to socioeconomic class, 90% were in the middle and low socioeconomic classes. Table 1 shows the sociodemographic characteristic of the children with AGN

Table 1: Socio-demographic characteristics of children with APIGN

Variables(Number analyzed)	Frequencies (%)
Age (116)	
1 - < 5	17(14.7)
5 - < 10	58(50.0)
10 - 15	41(35.3)
Gender (116)	
Male	74(63.8)
Female	42(36.2)
Socio-economic class (50)	
Class I	Nil
Class II	5 (10)
Class III	13 (26)
Class IV	16 (32)
Class V	16 (32)

AGN accounted for 8.9% of new childhood renal admissions with an annual hospital incidence of 8-9 new cases per annum but with an upsurge to 12-17 between 2017 and 2019 (Fig 1). The lowest monthly incidence was in the months of March and April and the highest between June and December each year (Fig. 2). The annual incidence is shown in Figure 1 while the monthly/seasonal incidence is shown in Figure 2. The clinico-laboratory parameters evaluated in 50 patients are as shown in Table 2. The major complaints were body swelling (oedema) in 92%, fever (34%), coca-coloured urine (34%), reduced urine output (oliguria/anuria) in 28%, cough (29%) pallor (26%) and others as shown in Table 2. Hypertension was present in 86% of the patients, 26% being stage 1 and 60% stage 2 hypertension (Table 2). A nephrotic nephritic picture was seen in 14 out of 50 patients (28%) with 6/50 (12%) having rapidly progressive glomerulonephritis (RPGN). Table 3 shows the complications of AGN seen in the cohort with hypertensive crises in 24%, stage III acute kidney injury (AKI) without a rapidly progressive picture in 3 (6%) and uraemic

encephalopathy in one (2%). Urinary tract infection was found in 10%. Tell-tale features of previous skin sepsis were found in 14/50 (28%) patients, while in pharyngo-tonsillitis was found in 9/50 (18%). Other infectious agents identified in some of the patients were Hepatitis B in 3 patients; mumps, varicella and probable diphtheria, a case each. Seven patients had haemodialysis and one patient had peritoneal dialysis, consequently 8 (16%) had RRT and RPGN was the indication in 5 patients. The total duration of hospital stay ranged between 1 and 74 days, with a median of 8 days. Children who required dialysis had longer duration of stay expectedly.One hundred and eleven (95.7%) of these patients were discharged while 5 died with a case fatality rate of 4.3%. Four of the 5 deaths were due to RPGN and the 5th death was due to a not rapidly considered progressive severe AKI. In effect, all 5 deaths were associated with severe AKI.

Table 2: Analysis of clinical features in children with AGN (50)

Clinical Features	Frequencies	Percentages (%)
Presenting complaints		
Body swelling (Oedema)	46	92
Fever	17	34
Coca-coloured urine	17	34
Cough	14	28
Oliguria/anuria	14	28
Skin infection	14	28
Pallor	13	26
Sore throat	9	18
Abdominal pain	9	18
Breathlessness	8	16
Vomiting	8	16
Seizure	7	14
Headaches	7	14
Herbal remedies	6	12
Others (Diarrhoea, hesitancy, Jaundice etc)	6	12
Blood Pressure Pattern		
Normal	7	14
Stage I	13	26
Stage II	30	60

Fig 1: AGN admission in years

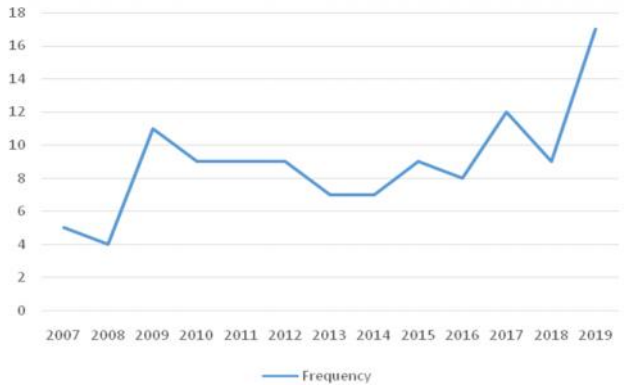


Fig 2: Shows the monthly/seasonal incidence

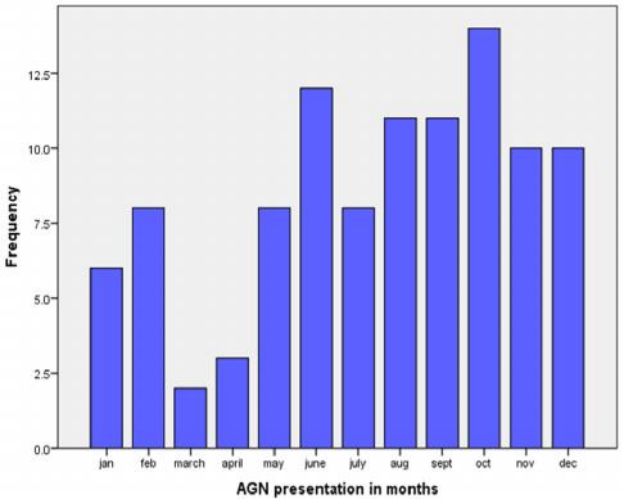


Table 3: Laboratory and Ultrasound Features in children with AGN (50)

Laboratory results and Abdominal USS	Frequencies (%)
Serum urea(mg/dl)	
<45	29 (58)
>45	21 (42)
>100	10(20)
Serum Creatinine(mg/dl)	
<1.2	35 (70)
>1.2	15 (30)
Haematuria	50 (100)
Proteinuria (Urinalysis)	
≤ 2+	36 (72)
≥ 3+	14 (28)
Haematocrit	
PCV > 30%	32(60)
PCV< 30%	13(26)
PCV < 25%	4 (8)
PCV <20%	1(2)
Total WBC (>11,000/cmm)	19(38)
Platelet count <100,000/cmm	3 (6)
>100,000/cmm	47(94)
Abdominal Ultrasound Results(45)	
Normal kidneys	15 (33.3)
Grade 1 Parenchymal disease	23 (51.1)
Grade II Parenchymal disease	4 (8.9)
Grade III Parenchymal disease	2 (4.4)
Non-specific	1 (2.2)
Renomegaly	1(2.2)
Hepatosplenomegaly	25 (55.6)

Table 4: Complications and outcome

Complications	Frequencies (%)
Rapid progressive glomerulonephritis (RPGN)	6 (12)
Stage III AKI without RPGN	3 (6)
Hypertensive encephalopathy	4 (8)
Heart failure/pulmonary oedema	5 (10)
Hypertensive urgencies	3 (6)
Urinary tract infection (Associated)	5 (10)
Duration of stay (days)	
Mean ± SD	12 ±11
Mode	7
Outcome (116)	
Discharge	111 (95.7)
Death	5 (4.3)

Table 5: Comparison of Epidemiological Data from Previous studies with the Present

Authors & Centre	Years of study (duration)	No. studied	Peak Age (yrs)	Mean ± SD (yrs)	Gender ratio (M:F)	Annual inci-dence	% in low social class	Peak mths of the year	Pyoderma Vs Pharyngitis related	Case fatality (%)
Aikhionbare & Abdurrahman[27] Zaria NW Nigeria 1984	1978-1982 (4years)	202	76% 4-10yrs	-	1.35:1	50	-	June-Dec	Pyoderma>> Pharyngi-tis	1.5
Okafor HU et al [28] Enugu SE Nigeria 1995	1985-1991 (7years)	216	5-8	-	1.3:1	31	-	July & Dec./ January	Pyoderma> Pharyngeal	1.4
Ibadin &Abiodun[3] Benin, S Nigeria2003	1996-2000 (5years)	63	3	6.6±4.3 M;7.6±3.6F	44.4/56.6 %	9-17	90.5	June to August	Pyoderma> Pharyngeal	3.2
Etuk et al Calabar[4] SS Nigeria 2009	1997-2006 (10yrs	76	Modal age-5yrs	7.2±4.3 M 6.5±3.2F	1.4:1	5-8	82	2Peaks May-July & Oct-Jan	Pyoderma> Tonsillitis	5
Anochie et al[5] Port Harcourt SS Nige-ria 2009	2006-2008 (3yrs)	31	5-10	-	1.1:1	15.5	77.4	October to February	33.4/66.6% Pharyngi-tis>Pyoder ma	9.7
Ibeneme et al[9] Umuahia S East Nigeria 2019	2011-2017 (6yrs)	19	Peak-5-10yrs-Mode - 6yrs;	6.85 ±3.48M	2:1	3	52.6	2Peaks May-July &Octo/Jan	Pyoderma> Pharyngitis	5.3
Bhalla et al[16] India 2019	Recruited over 1yr	50/48	Range :4-14yrs	8.7	1.72:1	-	90	July-January	Pharyngi-tis>>Pyoderma	Nil
Gebreyesus et al[36] Ethopia 2018	2013-2015	334	5-9	8.6	60.5/39.5 %=1.5:1	-	77.2% rural areas	Both sea-sons	Pharyngitis > Pyoder ma	6
Sharmin et al[35] Bangladesh 2020	2014/2015 (6mth)	60	7-9	-	58.3/41.7 % =1.4:1	60 in 6 mths	83.3	-	Pyoderma> Pharyngitis	1.7
Asinobi, Ademola & Nwankwo Present study	2007-2019	116	5-9	8.2±3.3	1.8:1	8-9	64	June to Decemb.	Pyoderma >pharyngitis	4.3

Fig 1: Cellular crescent in a child with rapidly progressive glomerulonephritis compared with normal glomerulus upper half. T: Glomerular tuft White arrow: Cellular crescent

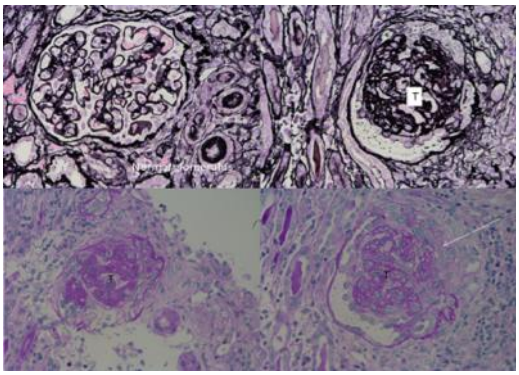
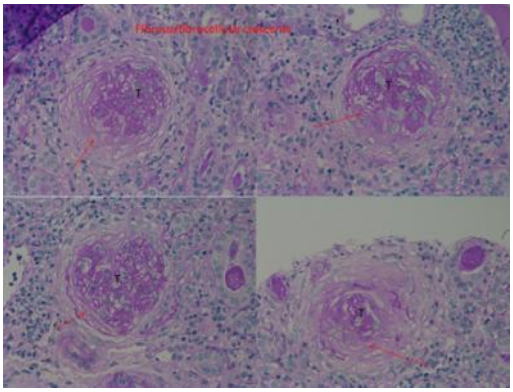


Fig 2: Fibrous / Firocellular crescent in a child with rapidly progressive glomerulonephritis T: Glomerular tuft
Red arrows: Fibrous/ fibrocellular crescents



Discussion

This study showed an average annual hospital incidence of 8 to 9 new cases per annum over a 13 year period but with an upsurge in the last 3 years of the study, almost doubling in 2019. The pattern of childhood renal disorders was first studied in Ibadan by Hendrickse and Gilles[23] over 50 years ago who documented 22 cases over a 5-year period giving an average of 4-5 cases per annum and that was the picture at the beginning of the present study in 2007 and 2008 (fig. 1). The average annual incidence found in this study is similar to the finding of 8 and 10 per annum by Ladapo et al from Lagos (2012)[24] and Olowu from Ile-Ife (2002)[25] respectively, both from Southwest Nigeria as Ibadan but less than 15.5 per annum by Anochie and Eke[5] and much less than the 50 cases/annum and 30 cases/annum reported from Zaria in the northern part of Nigeria[26] and from Enugu, Southeast Nigeria respectively.[27] It should however be noted that the last two referred studies were carried out over 2 decades ago.[26,27] There appears to have been a slow but steady rise in the incidence of AGN in Ibadan in the past 11years which may be a reflection of the present state of worsening poverty in our nation. There is also a possibility that poor living conditions might have been compounded by the effects of global climate change which manifests with extremes of weather encouraging staying indoors, overcrowding

and possible spread of infections.

Acute post-infectious AGN accounted for 8.9% of childhood renal admissions in this study and did not vary much from the previous findings of 11.4% and 11.6% from Ibadan[23,28] 11.4% from Port-Harcourt[29], 10% from Lagos[25] and Abuja[30] but much less than the figures reported from Zaria (38.1%), Jos (37.7%), Enugu (31.9%) and Kano (24.8%)[31,6,32,33]. The findings from Benin (20%) and Gusau (24%) are in between,[34,10] These data seem to suggest that children from the northern part of Nigeria are at greater risk for AGN and this most probably may due to climatic and socioeconomic factors.

The association of AGN in this study with low socioeconomic class was in keeping with studies from the developing world.[4,17,18,35-37] Approximately 90% of children with AGN in the present study belonged to the middle and low socioeconomic class and this did not differ much from the study by Etuk et al,[4] where 93.8% of their patients with AGN were of the middle and low socioeconomic classes. The average purchasing power and housing of Nigerians in these social classes have been very inadequate in the last two decades hence the call for an increase in the minimum wage of Nigerian workers. An increase from eighteen thousand Naira (N18,000 ≈ $50) to thirty thousand Naira (N30,000) per month which is approximately $83 per month has been accepted, but not fully implemented. This amount is still considered inadequate as it would be nearly impossible for a family of four to six individuals to live comfortably with that amount in present times. It is therefore not surprising that children in our environment commonly suffer from diseases associated with low socio-economic conditions such as APIGN.

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With regards to seasonal variation of the disease, the present study found an annual peak incidence between June and December and the lowest incidence in March and April of the year which differed slightly from the finding of Ugwu *et al*[8] who reported a peak between October and January. Etuk *et al*,[4] however, recorded two annual peak periods, May-July and October-January translating to peaks in the middle of both dry and rainy seasons of the year. Okafor and Okoro,[28] Ibeneme *et al*[9] also observed dual annual peaks and from almost all Nigerian studies the later third of each year had increased incidence. The above findings, the present study inclusive, suggest that the disease may be more common in the Southern part of Nigeria during the rainy season and early harmattan period when people may be more indoors.

This peak age incidence of between 5 and 9 years found in this study is in keeping with reports from most centres in Nigeria[8, 9] and from outside Nigeria.[38] but differed from the report by Olowu[26] whose patients were majorly below 6 years of age with a peak age incidence of 3 years. Ibadin and Abiodun[3] from South-south Nigeria in 2003 and Bhalla *et al*.[17] from India in a more recent article also reported a lower peak age incidence

The male preponderance in this study is similar to finding from most other studies conducted in Nigeria[27,4,5,28,38,9,30] and outside Nigeria.[16,35,36] [36-] Specifically, the male to female ratio of 1.8:1 is similar to findings of studies conducted in Asaba, and Umuahia, Nigeria[8,9] and in India.[17]

As regards antecedent infection in sporadic APSGN, upper respiratory tract infection, (pharyngitis and tonsillitis), is more common in winter and spring in temperate areas, whereas skin infections are commonly found to precede APSGN in the more tropical and subtropical areas, with a peak incidence during summer and autumn.[39] In the present study, a small proportion could recall preceding infections but pyoderma associated AGN was more prevalent. There is variable data from centres in Nigeria (Table 5) but pyoderma-associated AGN appears to occur more commonly in Nigeria at the present. Concerning the infectious agents incriminated in APIGN, the APSGN was by far the most common in the present study and in most childhood studies.[14] The other organisms encountered in this study were Hepatitis B, and the very uncommon mumps, varicella and diphtheria. Hepatitis B has been associated with AGN even in Nigeria.[27] The prevalence of Hepatitis B associated AGN was 6% in this study which is much less than 41% documented by Aikhionbare and Abdurraham[27] in the 1980s. It is noteworthy that in India, Gunasekaran et al [40] found majorly pyoderma-related AGN in 80.6% of cases but with a few cases of the unusual agents namely varicella, measles and mumps some of which were seen in this study. The afore-mentioned infections being vaccine-preventable, are declining in their prevalence and further decline is expected in the future.

This study has corroborated the fact that a Nigerian child with AGN is more likely to present acutely with generalised oedema of varying degrees accompanied by hypertension and proteinuria.[3,4,27,29] The findings from this study were quite similar to the findings of Etuk *et al*.[4] oedema for instance (92% versus 98.5%) and hypertension (86% versus 86.8%) were quite prominent features in these two studies. Ibadin and Abiodun,[3] Bhalla *et al*,[17] and Sharmin[35] also documented hypertension prevalence of 80% and above but these were higher than the range of 45% -68% recorded in other centres within Nigeria,[8,9,27,28,29] and much higher than 25.3% reported by Kuem *et al*[37] from Korea. It is possible that the different cut off values for the definition of hypertension would have been responsible for lower values in the past. It is notable that even though 60% of the children had Stage 2 hypertension and 24% had hypertensive crises, none died from hypertensive crises.

Another feature that requires a high index of suspicion is rapidly progressive glomerulonephritis (RPGN) which may complicate AGN and contribute to severe morbidity and mortality. RPGN was first recognized by Ellis[41] in 1942 and further defined by Heptinstall[42] in 1974. It was first described as an unusual form of acute glomerulonephritis (AGN) which progressed to renal failure, in contrast to the typical course of rapid resolution. A clinical feature which seemed to separate RPGN from AGN

was a period of prolonged oliguria and its renal histology is characterized by crescent formation with or without endocapillary proliferation of cells. In 1980, Cunningham et al[43] described their experience with a cohort of 13 patients with RPGN, 7 of whom had APSGN, during a period of trial of anticoagulants and anti-platelets drugs. The posited that antiplatelet therapy appeared to have improved survival and the severity of crescent formation, not the presumable aetiology, appeared to be a reliable prognosticator.

RPGN was the most challenging complication noted in our study and was seen in 12% of cases but accounted for 5 of the 8 children that had dialysis and was responsible for 4 of the 5 deaths recorded in this study. The diagnosis of RPGN was based on an acute presentation with a nephrotic nephritic picture and rapid decline in glomerular filtration rate in a matter of days or weeks in a child whose anthropometry and renal ultrasound did not suggest a chronic or systemic illness, with or without renal histology evidence. Renal biopsies could only be done on two of the cases with suspected RPGN and the biopsies demonstrated glomerular crescent formation (figure 3). Most of the children with RPGN arrived late and there was an additional challenge of non-accessibility and high cost of intravenous methylprednisolone in the earlier years of the study. The outcome with regards to mortality has improved in recent years. Ayoob and Schwaderer[44] described their experience with 17 children with APSGN that developed RPGN and some other atypical features. They opined that different strains and virulence factors may be the cause of more severe disease seen in the past and suggested that PSGN should be still be considered in cases of severe nephritis even when the presentation is consistent with other types of nephritis. They suggested that detailed investigations including determination of M serotype of organisms, serology tests, serum complements, renal histology in severe PSGN may be helpful in determining if a severe clinical course is based on the infecting organism or underlying patient characteristics. RPGN has not been significantly highlighted in the studies from Nigeria and there is need for a high index of suspicion for it. An early diagnosis of RPGN and appropriate intervention with pulse corticosteroids, with or without other immunosuppressive therapies and renal replacement therapy, could make a difference.

The other common complications seen in this study were in keeping with the findings of most studies but in varying proportions. AKI with pulmonary oedema occurred in 32% of patients studied by Bhalla et al[17] compared with 10% in this study. The prevalence of Hypertensive encephalopathy of 8% was seen in the present study and was similar to the findings by Bhalla et al[17] and Ibeneme *at al.*[9] The prevalence of urinary tract infection in the present study was much less common than encountered by Ibadin and Abiodun,[3] and Ibeneme *et al.*[9]

With regards to outcome in these subjects, the duration of stay ranged between one day and 74 days in this study with a mean duration of 12 days and a mode of 7 days. The duration was longer in those requiring haemodialysis expectedly. The mean duration was in keeping with findings from other workers.[8,27] The prognosis is usually good unless associated with severe AKI and crescentic glomerulonephritis in which case the outcome could be relatively poor unless treatment is early and adequate. Immunosuppressive therapy is not required in simple acute proliferative glomerulonephritis but is essential in modifying the outcome of RPGN (crescentic glomerulonephritis). It is however to be noted that most of the patients from the highlighted studies survived the acute kidney injury, the hypertensive crises and pulmonary oedema because they were managed by paediatric nephrologists, had access to immunosuppressive and renal replacement therapy. Children who do not have the privilege of such services in low income settings would die and the cause of death may never be known.

APIGN is still a challenge in many African and Asian countries, Nigeria inclusive. It is the most frequent renal pathology in children in Nepal,[45] the third and fourth most frequently seen in Ethopia (12.2%)[46] and Cote D'Ivoire (12.9%)[47.] The variable picture in Nigeria has been highlighted. Late presentation is of major concern as stated by Sylla *et al*[48] in Mali. Improvement in the standard of living with good environmental hygiene and easy access to good healthcare will reduce this preventable disease as has been demonstrated in the industrialized nations.

Conclusion

This study has shown an increasing prevalence of AGN in our centre which may be associated with the nation's economic downturn and increasing poverty among the populace. Acute post-streptococcal glomerulonephritis was the most common type of AGN encountered in this study but in a very few cases, AGN was associated with Hepatitis B, Mumps, Varicella and probable Diphtheria. Even though AGN is generally associated with good prognosis if diagnosed early and appropriate treatment given, the outcome could be poor when it is complicated by RPGN. There should therefore be a high index of suspicion for RPGN as institution of dialysis and immunosuppressives at an early stage may avert death or chronic kidney disease.

Limitations

Estimation of anti-Streptolysin O (ASO) titre and serum C3 and C4 which were supposed to be part of the routine investigations of our patients with AGN were not carried out in all patients.

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