Original Article

Acute Poststreptococcal Glomerulonephritis: Experience with 175 Pediatric Patients

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Objective: To evaluate the clinical presentation, risk factors associated with acute poststreptococcal glomerulonephritis (APSGN) complications and outcome of pediatric APSGN.

Materials and Methods: Retrospective medical records were reviewed of all children diagnosed as APSGN at Queen Sirikit National Institute of Child Health (QSNICH) between January 1, 2006 and December 31, 2014.

Results: One hundred seventy-five cases were studied. The mean patient age was 8.6±2.7 years old, with a male to female ratio of 1.4:1. The upper respiratory tract antecedent infections were found in 53.7% of the cases, while antecedent skin infections had a prevalence of 30.9%. Clinical manifestations were edema in 99.4%, hypertension in 95.4%, acute kidney injury (AKI) in 48.6%, gross hematuria in 24.5%, rapidly progressive glomerulonephritis (RPGN) in 7.4%, and hypertensive encephalopathy in 5.7% of the cases. An elevated ASO titer and anti-DNase B were detected in 79.4% and 96.9% of the cases, respectively. Proteinuria was significant risk factors of AKI (adjusted OR 4.48, 95% CI 1.57 to 12.72, p=0.005), while obesity was significantly linked to hypertensive encephalopathy (OR 8.41, 95% CI 1.82 to 38.75, p=0.018). The result of the initial treatment was excellent. However, two cases experienced complete remission post-treatment for three years where one case developed proteinuria and the other presented with proteinuria and deterioration of kidney function.

Conclusion: Pediatric APSGN is more common in males and mostly occurs following sore throat. The most common clinical presentation is edema. Proteinuria is a significant risk factor for AKI, while obesity is significantly related to hypertensive encephalopathy. Short-term clinical outcome is excellent but long-term prognosis is uncertain.

Keywords: Acute poststreptococcal glomerulonephritis, APSGN, Acute kidney injury, AKI, Hypertensive encephalopathy

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Acute poststreptococcal glomerulonephritis (APSGN) is a kidney disease that was discovered in the 18th century. Despite a worldwide reduction in its incidence and severity, pediatric APSGN is still one of the most common kidney problems for children living in developing countries[1]. The classical manifestation of APSGN is the sudden onset of acute nephritis or edema, hematuria, and hypertension, which may be associated with oliguria and azotemia after sore throat and/or skin pyoderma due to a preceding β-hemolytic streptococci group A infection. In rare cases, APSGN presents with nephrotic syndrome, acute kidney injury (AKI), rapidly progressive glomerulonephritis (RPGN), hypertensive encephalopathy, or posterior reversible encephalopathy syndrome (PRES)[2-4]. Current research has shown that the nephritis-associated plasmin receptor (NAPlr) and streptococcal pyrogenic exotoxin (erythrotoxin) B (SPEB), which can be found within the glomerular deposits, are nephritogenic antigens[5].

Kidney biopsy is generally unnecessary to confirm the diagnosis, but is indicated when patients present with atypical clinical features or there is the possibility of a different diagnosis such as normal serum complement, absence of rising in streptozyme titer, nephrotic syndrome, AKI at presentation, or

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in patients who have persistent hypertension, low serum complement, diminished renal function, proteinuria, and hematuria. The typical feature of kidney biopsy in acute phase of the disease, showing under light microscopy, includes endocapillary proliferation without a thickening of the capillary wall. Immunofluorescence mostly reveals diffuse, irregular, and coarsely granular staining of IgG and complement C3. The hallmark pathological findings, under electron microscopy, are sub-epithelial 'humps' that represent sub-epithelial immune complex depositions[6].

Supportive and symptomatic treatment such as restricted salt and fluid intake, diuretics (thiazide and/or loop diuretics), and antihypertensive agents are the first line treatment to control blood pressure and edema. Renal replacement therapy is rarely indicated. Penicillin is required if the infection is still present at the time of diagnosis, which may limit the spread of the β-hemolytic streptococci group A but does not affect the natural history or prevention of the disease[2,3]. The short-term prognosis is excellent in most cases, but the long-term prognosis is still questionable, especially in adults[7].

The objective of the present study was to identify the clinical presentation, risk factors associated with APSGN complications, and outcome of pediatric APSGN.

Materials and Methods

Study population

The present study undertook a retrospective case review of APSGN who were admitted to Queen Sirikit National Institute of Child Health (QSNICH) between January 1, 2006 and December 31, 2014. All patients were under 15 years of age. The inclusion criteria were 1) acute nephritis, 2) positive evidence of recent streptococcal infection [i.e., elevated antistreptolysin O (ASO) titer and/or anti-deoxyribonuclease B (anti-DNase B) titer], 3) serum complement C3 less than 0.9 g/L. Hypertension was diagnosed in the event that the patient's average blood pressure was higher than the ninety-fifth percentile for the patient's sex, age, and height, or greater than 130/80 mmHg for adolescents older than 13 years[8]. Hypertensive encephalopathy was defined as acute severe hypertension, leading to clinical symptoms of encephalopathy, including seizures, severe headache, visual disturbance, focal neurological deficits, and/or characteristic radiological findings[9]. AKI was defined using the pediatric-modified RIFLE and/or the acute kidney injury network (AKIN) criteria[10,11]. Obesity was defined using the WHO Child Growth Standards on length/height, weight, and age[12]. In contrast, patients were excluded from the study if they had a previous history of hypertension, acute and/or chronic nephritis, and cardiac disease. Demographic data, sex, age, body weight, clinical manifestations, serum creatinine (Cr), blood urea nitrogen (BUN), albumin, complement, ASO titer, anti-DNase B titer, and urine protein to Cr ratio for each patient were recorded. The study was approved by the Ethics Committee of QSNICH.

Statistical analysis

Data were expressed as percentage and mean ± standard deviation (SD). Statistical analyses were conducted using the Chi-square test or Fisher's exact test in Stata version 12.1. All p-values presented as two-tailed (p-value less than 0.05) were considered statistically significant. Multivariate logistic regression analysis was calculated by step wise technique. Risk factors of AKI and hypertensive encephalopathy were presented by odds ratio (OR) and ninety-five percent confidence interval (CI).

Results

One hundred seventy-five pediatric patients were included in the study. Most were boys (male-to-female ratio of 1.4:1). Their ages ranged from 2 to 14 years (mean 8.6±2.7 years) and 87.4% of the cases occurred between 4 and 14 years of age. Eleven patients were obese. Upper respiratory tract infections were the most common antecedent infections, occurring in 53.7% of the cases studied. The most common clinical manifestations were edema, which was observed in 99.4%, while other significant manifestation was hypertension, seen in 95.4% of the cases. The most common complication in the present study was AKI, occurring in 48.6% of the cases, followed by RPGN and hypertensive encephalopathy. A summary of the demographic data, clinical manifestations and complications is provided in Table 1. Urinalysis revealed that every patient in the study presented with hematuria. Proteinuria was seen in 76% of the cases, in contrast to a 16% incidence rate in nephrotic range proteinuria. Serologic findings showed reduction in serum complement C3 occurred in all cases, while low level of serum complement C4 was noted in 17.4% of the cases. The rising titers of ASO and anti-DNaseB were positive in 79.4% and 96.9% of the cases studied, respectively. Laboratory findings are shown in Table 2.

The clinical result of the patients in the present study was excellent. None required renal replacement therapy and no mortality or progression to chronic kidney disease (CKD) occurred after initial treatment.

Table 1. Demographic data and clinical manifestations of APSGN (total 175 cases)

Characteristics	n (%)
Sex	
Male	102 (58.3)
Female	73 (41.7)
Age (years), Range	2 to 14
Nutritional status	
Obesity	11 (6.3)
Preceding infection	
Upper respiratory tract infection	94 (53.7)
Skin infection	54 (30.9)
Upper respiratory tract and skin infection	20 (11.4)
No infection	7 (4.00)
Clinical manifestations	
Edema	174 (99.4)
Hypertension	167 (95.4)
Gross hematuria	43 (24.5)
Cough	22 (12.6)
Headache	17 (9.7)
Dyspnea	16 (9.1)
Nausea and/or vomiting	12 (6.9)
Convulsion	10 (5.7)
Blurred vision	3 (1.7)
Complications	
Acute kidney injury	85 (48.6)
Rapidly progressive glomerulonephritis	13 (7.4)
Hypertensive encephalopathy	10 (5.7)
Respiratory failure	3 (1.7)
Congestive heart failure	2 (1.1)
Pulmonary edema	2 (1.1)

APSGN=acute poststreptococcal glomerulonephritis

Table 2. Laboratory findings in APSGN

Laboratory	n* (%)
Hematuria (RBC ≥5/HPF)	175/175 (100)
Elevated ASO titer	127/160 (79.4)
Elevated Anti-DNaseB titer	154/159 (96.9)
Low serum complement C3	175/175 (100)
Low serum complement C4	28/161 (17.4)
Proteinuria	133/175 (76.0)
Nephrotic range proteinuria	28/175 (16.0)
Serum albumin	
>3.5 g/dL	50/164 (30.5)
3.5 to 2.5 g/dL	113/164 (68.9)
<2.5 g/dL	1/164 (0.6)

APSGN=acute poststreptococcal glomerulonephritis; RBC= red blood cells; ASO=antistreptolysin O

* Positive finding/total number of specimens

All of them had complete resolution of gross hematuria in the first week post-diagnosis and 92% of the cases also had hypertension improved within the same time frame. The mean time to resolution of gross hematuria, microscopic hematuria, and proteinuria was 1±1.2 weeks, 2.6±2.3 months, and 1.8±1.6 months, respectively. No cases experienced recurrence of APSGN within the period of the study.

However, two cases presented with abnormal proteinuria after complete resolution. The first case was a previously healthy 10-year-old boy who had no proteinuria for three months after diagnosis. Nevertheless, he developed proteinuria after three years post-resolution. His kidney function was still normal and kidney biopsy was unremarkable. The second case was a 9-year-old boy with a history of posterior urethral valve S/P valve ablation since six-month of age who had normal blood pressure, normal kidney function, and no proteinuria pre-APSGN diagnosis. His abnormal proteinuria resolved one month after diagnosis. He maintained a normal kidney function without hematuria, proteinuria, edema, or hypertension for three years. Later, he developed proteinuria and a slow deterioration of his kidney function.

An analysis of the risk factors of AKI in APSGN patients by the Chi-square test revealed that the incidence of gross hematuria and proteinuria were significantly higher among the cases with AKI than among those without AKI (OR 2.15, 95% CI 1.06 to 4.36, p=0.032 and OR 4.87, 95% CI 1.74 to 13.6, p=0.001, respectively). Risk factors of AKI in APSGN are shown in Table 3. While using multivariate logistic regression analysis, which are shown in Table 4, proteinuria was the only significant factor associated with AKI (adjusted OR 4.48, 95% CI 1.57 to 12.72, p=0.005). In addition, the risk factors of hypertensive encephalopathy in APSGN after analysis by the Fisher's exact test determined that the major cause seemed to be obesity (OR 8.41, 95% CI 1.82 to 38.75, p=0.018). The risk factors of hypertensive encephalopathy compared with non-hypertensive

Table 3. Risk factors of AKI in APSGN

	AKI (n=85) n (%)	Non-AKI (n=90) n (%)	OR	95% CI	p-value
Sex				0.91 to 3.07	0.094
Male	55 (53.9)	47 (46.1)	1.67		
Female	30 (41.1)	43 (58.9)	1.00		
Obesity			3.01	0.77 to 11.76	0.098
Yes	8 (72.7)	3 (27.3)			
No	77 (47.0)	87 (53.0)			
Upper respiratory tract infection			1.30	0.69 to 2.43	0.404
Yes	58 (50.9)	56 (49.1)			
No	27 (44.3)	34 (55.7)			
Skin infection			1.33	0.73 to 2.43	0.349
Yes	39 (52.7)	35 (47.3)			
No	46 (45.5)	55 (54.5)			
Hypertension			1.55	0.57 to 4.20	0.386
Yes	78 (49.7)	79 (50.3)			
No	7 (38.9)	11 (61.1)			
Gross hematuria			2.15	1.06 to 4.36	0.032*
Yes	27 (62.8)	16 (37.2)			
No	58 (43.9)	74 (56.1)			
Proteinuria			4.87	1.74 to 13.60	0.001*
Yes	80 (53.7)	69 (46.3)			
No	5 (19.2)	21 (80.8)			

APSGN=acute poststreptococcal glomerulonephritis; AKI=acute kidney injury; OR=odds ratio; CI=confidence interval

* p<0.05 were statistically significant

Table 4. Multiple logistic regression of risk factors associated with AKI in APSGN

	Adjusted OR	95% CI	p-value
Sex	1.70	0.88 to 3.26	0.109
Obesity	2.54	0.62 to 10.41	0.193
Gross hematuria	1.74	0.82 to 3.68	0.142
Proteinuria	4.48	1.57 to 12.72	0.005*

APSGN=acute poststreptococcal glomerulonephritis; AKI=acute kidney injury; OR=odds ratio; CI=confidence interval

* p<0.05 were statistically significant

encephalopathy in APSGN are shown in Table 5.

Discussion

The present cases studied were mostly boys and mean age was 8.6±2.7 years. Most of the cases had prodromal upper respiratory tract infections preceding APSGN onset, which is in accordance with the findings of other studies[13,14]. The initial clinical manifestations were edema in 99.4%, hypertension in 95.4%, and gross hematuria in 24.5% of the cases studied. Similarly, Rodriguez-Iturbe et al observed the manifestations of edema in 90%, hypertension in 60% to 80%, and gross hematuria in 33% of the cases in their studies[2]. An elevated ASO titer was seen in 79.4% of the cases in the present research, while Derakhshan et al reported 84% of the cases[15]. Although an elevated ASO titer represents an antecedent streptococci infection, it has an incidence rate of only 80% in most studies. Pyoderma-associated APSGN cases or those treated with antistreptococcal antibiotics may show a false-negative result. Due to limitations of ASO titer in common practice, other serological markers are used, such as anti-DNase

Table 5. Risk factors of hypertensive encephalopathy in APSGN

	HTE (n=10) n (%)	Non-HTE (n=165) n (%)	OR	95% CI	p-value
Sex				0.62 to 14.66	0.187
Male	8 (7.8)	94 (92.2)	3.02		
Female	2 (2.7)	71 (97.3)	1.00		
Obesity			8.41	1.82 to 38.75	0.018*
Yes	3 (27.3)	8 (72.7)			
No	7 (4.3)	157 (95.7)			
Upper respiratory tract infection			0.79	0.21 to 2.92	0.741
Yes	6 (5.3)	108 (94.7)			
No	4 (6.6)	57 (93.4)			
Skin infection			0.90	0.24 to 3.32	1.000
Yes	4 (5.4)	70 (94.6)			
No	6 (5.9)	95 (94.1)			
Gross hematuria			0.75	0.15 to 3.70	1.000
Yes	2 (4.7)	41 (95.3)			
No	8 (6.1)	124 (93.9)			
AKI			1.06	0.29 to 3.80	1.000
Yes	5 (5.9)	80 (94.1)			
No	5 (5.6)	85 (94.4)			
Proteinuria			0.93	0.89 to 0.97	0.362
Yes	10 (6.7)	139 (93.3)			
No	0 (0.0)	26 (100)			

APSGN=acute poststreptococcal glomerulonephritis; HTE=hypertensive encephalopathy; Non-HTE=non-hypertensive encephalopathy; OR=odds ratio; CI=confidence interval; AKI=acute kidney injury

* $p<0.05$ were statistically significant

B, for confirmation of an antecedent streptococcal infection. The anti-DNase B titer, unlike the ASO titer, can be high in both pharyngitis-associated APSGN and pyoderma-associated APSGN, which is much more sensitive than the ASO titer[4]. In the present study, an elevated Anti-DNaseB titer had an incidence rate of 96.9%, compared to an elevated ASO titer in 84% of the cases.

Furthermore, all cases had reductions of serum complement C3, like those in Kasahara et al[14] Similarly, Derakhshan et al and D'Cruz et al showed in their clinical works comparable reduction in 86% and 94.3% of their cases, respectively[15,16]. The present study also demonstrated that decreased levels of serum complement C4 was 17.4% of the cases, while Takeno et al and Natarajan et al observed incidence rates of 38.9% and 4.9%, respectively[17,18]. Although almost of APSGN patients had low serum complement C3, some of them might develop low serum C4. It shows that most of the activation of the complement system is an alternative complement pathway; however, the classic and lectin pathways might also play a role in this condition[2,4,19]. Proteinuria was seen in 76%, but severe proteinuria and nephrotic syndrome was only observed in 16% and 0.6% of the cases, respectively. In comparison, Yuniarchan et al reported proteinuria occurred in 52.5%[20], while Dreakhshan et al and Becquet et al showed that pediatric APSGN developed nephrotic range proteinuria in 24.5% and 25% of the cases, respectively[13,15]. This information implied that pediatric APSGN could lead to massive proteinuria at the rate of 16% to 25%, but the feature of nephrotic syndrome is rare in this condition[21,22].

Considering the complication due to APSGN,

hypertensive encephalopathy was found in 5.7% of the cases. Takeno et al observed that APSGN cases with convulsion secondary to severe high blood pressure had an incidence rate of 3.3%[17], while Ilyas et al showed a rate of 6%[23], Roy et al and Burke et al reported incidence rates of hypertensive cerebral complications of 30% and 35%, respectively[24,25]. The reports on hypertensive encephalopathy and/or cerebral complications of hypertension in APSGN cases have surprising disparities. The reason for the difference in incidence rates is unclear, but it may correlate with the variability in the definition of hypertensive encephalopathy and the severity of the groups studied. Interestingly, evidence of acute severe hypertension and cerebral complications were commonly found in the early series[24,25].

In the present study showed both pulmonary edema and pleural effusion were uncommon when compared with AKI and RPGN. However, these can happen in severe cases of pediatric APSGN. Ilyas et al reported a pleural effusion incidence rate of 55%[23], while the present study observed a rate of 2.3%. The authors believed the difference in detection rates stem from differences in the diagnostic procedure. In the present study, chest X-rays were performed only when patients complained of respiratory symptoms. Hence, not all of the present APSGN cases had such data available in these conditions.

The results of immune complex and inflammatory reactions in the glomeruli, especially during the acute phase, lead to a reduction in the glomerular filtration rate (GFR) and acute nephritis symptoms. However, the renal blood flow is usually normal[2]. Significantly high serum Cr or AKI at presentation can point to a severe inflammatory process. The present study showed proteinuria in pediatric APSGN correlated with a significant reduction of GFR or AKI. Proteinuria is a good marker of glomerular and tubular damage, which indicates the severity of disease, especially in long-term persistent proteinuria. The relationships between gender, obesity, preceding infection, clinical manifestations, complications, and low serum albumin were not associated with an increased risk of AKI.

Obesity was observed in 6.3% of the present cases and was only one risk factor of hypertensive encephalopathy. Like a previous report from Eddy, obesity influenced hypertension in pediatrics[26]. Surprisingly, after recovery from the acute phase of APSGN, all the obese patients had a normal blood pressure. It might explain that the obese children tend to develop hypertension much more easily than normal-weight children in stress condition. This is especially true regarding kidney disease. The authors suggested that long-term follow-up of blood pressure should be indicated for this population.

All the patients in the present study were discharged without any serious problems, such as recurrence, mortality, or progression to CKD after initial treatment. However, after three years of follow-up, a previous healthy 10-year-old boy developed non-nephrotic range proteinuria. Another case, a 9-year-old boy who had history of posterior urethral valve S/P valve ablation, developed proteinuria and slow deterioration of kidney function. Because of maintaining normal kidney function without proteinuria during the subsequent three years, the authors believe that the deterioration of kidney function of the second case might be aggravated by APSGN. Such findings corresponded to those in Rodriguez-Iturbe et al who reported that pediatric APSGN cases developed non-nephrotic proteinuria in 7.2%, microscopic hematuria in 5.4%, hypertension in 3%, and azotemia in 0.9% of the cases, after 15 to 18 years of follow-up[1]. The authors conclude that the short-term prognosis in pediatric APSGN cases is excellent. However, long-term outcome is not uniformly benign, even after a complete recovery.

Conclusion

Based on the findings, it can be concluded that APSGN is more common in males and mostly occurs following a sore throat. The most common clinical presentation is edema. Proteinuria is correlated with AKI, while obesity is significantly linked to hypertensive encephalopathy in pediatric APSGN cases. The short-term prognosis is excellent, but long-term prognosis is not uniformly benign. However, the limitation of the present study is that it is a retrospective case review, so it might miss some data and patients. A prospective cohort study in a large multicenter is needed to corroborate these findings.

What is already known on this topic?

APSGN is more common in males and mostly occurs following a sore throat. The most common clinical presentation is edema. The result of treatment is excellent.

What this study adds?

Proteinuria is the only significant risk factor of AKI, while obesity is associated with hypertensive encephalopathy in pediatric APSGN cases. Because long-term prognosis is still uncertain, the authors

suggest that long-term follow-up of pediatric APSGN cases should be indicated.

Conflicts of interest

The authors declare no conflict of interest.

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