

Epidemiology and clinical outcomes of acute glomerulonephritis in a teaching hospital in North India

Kapil Bhalla¹, Ashish Gupta², Sanjiv Nanda¹, Shuchi Mehra³

Departments of ¹Paediatrics, ²Trauma Centre and ³Microbiology, Pandit Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences, Rohtak, Haryana, India

Abstract

Context: Acute glomerulonephritis (AGN) is a major cause of morbidity in developing countries like India. AGN includes both infectious and non infectious causes leading to immunological insult to the kidney. **Aims:** This study is an attempt to evaluate the clinical characteristics, complications and outcome of acute glomerulonephritis. **Settings and Design:** This hospital based descriptive study was conducted at a tertiary care hospital in North India. Materials and Methods: 50 children with AGN were recruited and analyzed over a period of one year. Detailed clinical examination and relevant laboratory investigations were done. Children were followed up for 6 months. **Statistical Analysis Used:** Data analysis was done using SPSS software and the results obtained are shown in the form of frequencies along with percentages. **Results:** 50 patients were enrolled in the study with male to female ratio of 1.72:1. Pharyngitis was the most common predisposing condition (70.2%). 22(40.4%) of patients had developed complications. Of this acute kidney injury with significant pulmonary edema was seen in 16 patients and 6 patients had encephalopathy with seizures. Two patients had to be managed with hemodialysis. 42 patients had positive CRP (>10) and ASO titres were >200 Todd units in all patients. At 6 months' majority of patients had complete clinical recovery with microscopic hematuria present only in 8 patients, persistent hypertension in 2 patients, 8 patients had proteinuria. These patients are being still followed up. **Conclusions:** Complications and morbidity is significantly high during the acute phase in AGN. Non infectious causes should also be kept in mind. This study highlights the complications requiring intensive care and need for long term follow up.

Keywords: Child, epidemiology, glomerulonephritis, hospital, India, streptococcus

Introduction

Diseases involving the renal glomeruli are encountered frequently in pediatric clinical practice and are the most common causes of end stage renal disease. Some do not cause progressive renal failure but are important causes of morbidity and parental worry. Acute glomerulonephritis (AGN) is defined as those glomerular diseases that may present with an acute nephritic syndrome which is characterized by sudden onset of gross hematuria, edema, hypertension, and renal insufficiency.^[1] It occurs due to immunologically mediated injury to the glomerulus by various infectious agents such as viral, bacterial, or protozoa organisms

> Address for correspondence: Dr. Ashish Gupta, House number 624, first floor, Sector 17 A, Gurgaon, Haryana, India. E-mail: ashishg19995@gmail.com

Access this article online					
Quick Response Code:	Website: www.jfmpc.com				
	DOI: 10.4103/jfmpc.jfmpc_57_19				

or also due to non-infectious causes like Henoch–Schonlein purpura (HSP). Among the infectious causes, post-streptococcal glomerulonephritis (PSGN) is the most common, and it is a non-suppurative sequel to Group A β -hemolytic streptococci.^[2] In tropical areas, there is tendency to have pyoderma-associated PSGN^[3] where in temperate climate, there is predominance of pharyngitis associated with PSGN.^[4] In this study, however, it was pharyngitis which was the predominant cause. The incidence of PSGN has decreased in developed world, but in developing nation like India, it is still an important public health problem.^[5] AGN is one of the leading cause requiring hospital admissions in children, and it is also an important cause of acute renal failure in developing countries.^[6] Though deaths due to this disease are rare, it can cause serious complications such as hypertensive emergency, congestive cardiac failure, renal failure,

For reprints contact: reprints@medknow.com

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

How to cite this article: Bhalla K, Gupta A, Nanda S, Mehra S. Epidemiology and clinical outcomes of acute glomerulonephritis in a teaching hospital in North India. J Family Med Prim Care 2019;8:934-7.

encephalopathy, and retinopathy.^[7] Very few studies of AGN are published in literature regarding clinical profile detailing complications from hospitals in north India; hence, this present study was undertaken.

Subjects and Methods

This hospital-based prospective study was conducted at a tertiary care hospital in North India, after obtaining approval from institutional ethics committee. Children admitted with AGN in pediatric wards were taken up for study over a period of 1 year and further followed up for 6 months. The objectives were to study the various clinical manifestations and complications and to identify those children who needed long-term follow-up. All children less than 14 years of age with acute nephritic syndrome who presented in the department of pediatrics during 1 year were included in this study. Those children more than 14 years and with evidence of preexisting renal disease were excluded. AGN was characterized by hematuria, proteinuria, oliguria, hypertension, edema, red blood cells (RBC) casts in urine, and circulatory congestion. Oliguria was defined as urine volume less than 1 mL/kg/hr. Edema as puffiness of face, bilateral pitting pedal edema and abdominal wall edema. Macroscopic hematuria was defined as visible pink or brown colored urine owing to the presence of RBC confirmed by microscopic examination. Microscopic hematuria was defined as more than five RBCs per high power field in the sediment of 10 mL of centrifuged freshly voided urine sample. Hypertension was defined as average systolic blood pressure (BP) or diastolic BP that was $\geq 95^{\text{th}}$ percentile for gender, age, and height on more than three occasions. Abnormal proteinuria was defined as 4-40 mg/m²/hr. Those patients who fulfilled the inclusion criteria were included in the study after initial clinical and laboratory evaluation. Antistreptolysin O titer (ASO titer) was measured by latex agglutination and titers more than 200 IU/mL were considered as evidence for recent streptococcal infection. Serum complement 3 (C3) levels were measured by immunoturbidimetric method. Radiological investigations included ultrasonography of kidney, ureter, and bladder. PSGN was diagnosed based on the following criteria: features of acute nephritic syndrome and evidence of recent streptococcal infection (recent pyoderma or pharyngitis with positive ASO titer or throat swab positive for group A streptococcus) and low serum C3 with normalization of C3 on 12 weeks follow-up. Patients who were discharged were advised regular follow-up for any evidence of residual renal injury. Follow-up protocol was done every fortnight for first 3 months and every month for next 3 months. Eight patients are still on regular follow-up. Data analysis was done using SPSS software and the results obtained are shown in the form of frequencies along with percentages.

Results

There were 48 children with diagnosis of PSGN and two children had nephritis post HSP. Mean age of presentation was 8.7 years with male to female ratio of 1.72:1. Out of total 48 children of PSGN, 33 (68.75%) reported in the period from July to January, that is, months having rainfall and winter season. Also, 36 (72%) patients were referred from other centers. Forty-five (90%) children belonged to low socioeconomic status. Duration of stay in hospital ranged from 4 to 22 days (mean = 12 days).

Facial puffiness and/or pedal edema was seen in 49 patients, and hypertension was seen in 40 (80%) patients. Oliguria was present in 42 (84%), gross hematuria in 16 (32%), and microscopic hematuria in 30 (60%) patients. Thirty-three (66%) patients had other features like fever, abdominal pain, headache, cough, difficulty in breathing, and syncopal attack. Six patients had developed altered sensorium and seizures at the time of presentation, and congestive cardiac failure along with pulmonary edema was seen in 16 patients. Recent history of sore throat was seen in 36 (72%) and skin lesion in only 5 (10%) patients [Figure 1].

Laboratory findings revealed elevated ASO titer in 46 (95.83%), elevated C-reactive protein (CRP) in 42 (87.5%), and decreased C3 in all 48 (100%) patients with PSGN. Two patients had AGN post HSP without any antecedent history of infection. Urinalysis showed RBC casts were present in 20 and granular casts in 6 patients. Elevated blood urea (>40 mg%) was seen in 20 patients and elevated serum creatinine (>1 mg%) was seen in 38 patients. Glomerular filtration rate (GFR) <100 mL/min/m² was present in 39 patients [Table 1].

Ultrasound of the kidney in 42 patients was normal and parenchymal disease with increased cortical echogenicity was seen in 8 patients. Out of 48 patients with PSGN, 10 patients were managed on outdoor basis and 38 patients were hospitalized. Salt restriction and diuretics were used in 22 patients and antihypertensive drug was used in 12 patients. Hypertension disappeared within 1 week in 36 patients and only 2 patients are taking antihypertensive for more than 4 weeks. Similarly, pedal edema disappeared within 2 to 16 days with mean duration of 6 days in 48 patients. Dialysis was required in two patients out of total 41 patients with PSGN. Encephalopathy improved with antihypertensives by day 2 in all six patients. At 6 months, majority

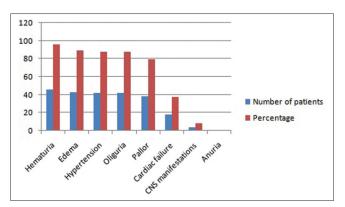


Figure 1: Bar diagram depicting occurrence of various signs and symptoms

Table 1: Frequency and percentage of different laboratory											
investigations											
			•		B .T		c			D	

Investigations	Number of patients	Percentage
Hemoglobin		
>11 gm%	7	14.58
7-11 gm%	39	81.25
<7 gm %	2	4.17
ASO (>200 Todd units)	48	100
CRP		
Positive	20	41.67
Negative	28	58.33
Serum creatinine		
<1 mg%	10	20.83
>1 mg%	38	79.17
GFR (in mL/min/m ²)		
>100	9	18.75
60-100	29	60.42
<60	10	20.83
Ultrasound abdomen		
Ascites	18	37.5
Increased renal echogenecity	44	91.67
X-ray chest		
Normal	19	39.58
Hilar prominence	26	54.17
Other abnormalities	3	6.25
Throat swab culture		
Sterile	26	54.17
Normal flora	22	45.83
Growth	0	0

of patients had complete clinical recovery with microscopic hematuria present only in eight patients; these patients are being still followed up. In this study, no case of nephritis following other bacterial or viral infections or systemic vasculitis was observed.

Discussion

Majority of studies are available on PSGN, a common cause for acute nephritic syndrome. A total of 48 patients were enrolled to study the various clinical manifestations and complications and to identify those children who needed long-term follow-up. Out of total 48 children of PSGN, 33 (68.7%) reported in the period from July to January, that is, months having rainfall and winter season, when there is increase in incidence of pharyngitis and upper respiratory tract infections. Certain other studies^[8,9] also reported peak of PSGN during winter months.

Tejani *et al.* observed AGN affecting age group of 2 to 10 years.^[10] Very few patients (\leq 5%) were below 2 years of age. The mean age was observed to be 6.8 years in a South Indian study with more than three fourth children above 5 years of age.^[11] The PSGN is rare in the very young children because of immature immune response.^[12,13] In this study, the age group ranged from 4 to 14 years. The M: F ratio was 1.72:1 which is similar to studies by Rodriguez Iturbe *et al.*^[14] and Gunashekaran *et al.*^[11] The observed family size was 7.5 with a large number of patients (86%)

reporting from family size of six or more. So, it was observed that PSGN is more prevalent in children of families who are crowded due to bigger family size and poor socioeconomic status.

In our study, 81.25% cases complained of either pharyngitis or skin infection in the recent past. Sore throat was the antecedent event in 70.83% of cases and skin infection in 10.42% cases. Pharyngitis was rarely seen in patients less than 6 years age. Nissenson *et al.* also reported that pharyngitis is less common in younger age groups.^[15] In the study by Nissenson *et al.*, latent period was observed to be 1 to 3 weeks.^[15] It was found to be 1 to 3 weeks in this study also. Also, 4.6% of children with PSGN developed hypertensive encephalopathy in a study by Gunashekaran *et al.*^[11] In our study, 8.33% patients developed hypertensive encephalopathy in a study by Gunashekaran *et al.*^[11] In our study, 8.33% patients developed hypertension, central nervous system (CNS) manifestations, and other non-specific manifestations were similar to those observed in various studies.^[9,16-18]

In this study, one patient who presented with features of acute nephritic syndrome developed grade III systolic murmur on sixth day of admission along with tachycardia and breathlessness. By the time patient was having dieresis, edema was subsiding. On chest radiography, cardiomegaly was present and echocardiography on seventh day of admission showed prominence of right atrium and right ventricle. There was a history of preceding throat infection 10-12 days prior to admission. So the patient was diagnosed as a case of acute rheumatic fever following PSGN. Although some serotypes are both rheumatogenic and nephritogenic, it cannot be said with certainty whether a single pharyngeal infection resulted in both PSGN and acute rheumatic fever. But, as the latency period of acute rheumatic fever (approximately 20 days) is slightly more than that of PSGN, the possibility of a single infection causing both PSGN and rheumatic fever cannot be ruled out. This patient when observed 8 weeks after discharge was clinically asymptomatic; blood pressure was normal, and urinalysis was also normal but grade III systolic murmur was persisting.

ASO titers were >200 Todd units in a patient presenting with severe hypertension but only minimal urinary findings. Blood pressure was 150/90 mm of mercury. Urine albumin was in traces and only 3-3 RBC/HPF was observed. Moreover, 24 hours urinary protein was 300 mg/day; blood urea and serum creatinine was normal. Such cases have also been reported by Albert *et al.*^[19] and Cohen *et al.*^[20]

Regarding outcome evaluation, as per the operational definitions, recovery was complete at the end of 6 months in all the patients (100%) with microscopic hematuria present only in eight patients, persistent hypertension in two patients, and proteinuria in eight patients. These patients are being still followed up. All these patients were grouped under resolved category and none of the patients had findings suggestive of resistant or progressive nature. Dodge *et al.*^[21] had reported that epidemic PSGN healed

in virtually 100% of cases while sporadic PSGN healed in only 90% of cases.

The family physicians should make an early diagnosis of AGN and timely refer the patient to a higher center as it has good prognosis if detected early and timely treated; otherwise the patient may land up into serious fatal complications.

We conclude that pharyngitis is most common antecedent infection in PSGN peaking in winter and rainy season. The incidence is more common in children of bigger family size due to overcrowding. The complications and morbidity is significantly high during the acute phase in AGN. Non-infectious causes should also be kept in mind. Lack of proper laboratory facilities is an area of concern in developing countries; however, good prognosis in this study suggests that early admission, intensive care, and long-term follow-up minimizes the fatal outcome.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

- 1. Vinen CS, Oliveria DBG. Acute glomerulonephritis. Postgrad Med J 2003;79:206-13.
- 2. Bullen A, Shah MM. De novo postinfectious glomerulonephritis secondary to nephritogenic streptococci as the cause of transplant acute kidney injury: A case report and review of the literature. Case Rep Transplant 2018;2018:2695178. doi: 10.1155/2018/2695178.
- Sulyok E. Acute proliferative glomerulonephritis. In: Avner ED, Harmon WE, Niaudet P, editors. Pediatric Nephrology. 5th ed. Philadelphia: Lippincott, Williams and Wilkins; 2004. p. 601-13.
- 4. Roy S 3rd, Stapleton FB. Changing perspectives in children hospitalized with poststreptococcal acute glomerulonephritis. Pediatr Nephrol 1990;4:585-8.
- Trivedi M, Pasari A, Chowdhury AR, Kurien AA, Pandey R. The epidemiology, clinical features, and outcome of infection-related glomerulonephritis from East India: A single center experience. Indian J Nephrol 2017;27:307-12.
- Barbiano Di Belgiojoso G, Genderini A, Ferrario F. Post-infectious glomerulonephritis. G Ital Nefrol 2003;20:184-99.

- 7. Arora P, Kher V, Rai PK, Singhal MK, Gulati S, Gupta A. Prognosis of acute renal failure in children: A multivariate analysis. Pediatr Nephrol 1997;11:153-5.
- Travis LB. Acute Post Infections Glomerulonephritis. Pediatric Kidney Disease. 1st ed. Boston: Little, Brown and Company; 1978. p. 611-31.
- 9. Khuffash FA, Sharda DC, Majeed HA. Sporadic pharyngitis associated acutepost streptococcal nephritis. A four year prospective clinical study of the acute episode. Clin Pediatr 1986;25:181-4.
- 10. Tejani A, Ingulli E. Post streptococcal glomerulonephritis: Current clinical and pathologic concepts. Nephron 1990;55:1-5.
- 11. Gunashekaran K, Krishnamurthy S, Mahadevan S, Harish BN, Kumar AP. Clinical characteristics and outcome of post-infectious glomerulonephritis in children in Southern India. Indian J Pediatr 2015;82:896-903.
- 12. Eison TM, Ault BH, Jones DP, Chesney RW, Wyatt RJ. Post-streptococcal acute glomerulonephritis in children: Clinical features and pathogenesis. Pediatr Nephrol 2011;165-80.
- 13. Bingler MA, Ellis D, Moritz ML. Acute post-streptococcal glomerulonephritis in a 14 year old boy: Why is this uncommon? Pediatr Nephrol 2007;22:448-50.
- 14. Rodriguez-Iturbe B, Rubio L, Gracia R. Attack rate of post streptococcal nephritis in families. Lancet 1981;1:401-3.
- 15. Nissenson AR, Baraff LJ, Fine RN. Post streptococcal acute glomerulonephritis: Fact and controversy. Ann Int Med 1979:91:76.
- 16. Lewy JE, Salinas-Madrigal L, Herdson PB, Pirani CL, Metcoff J. Clinico-pathologic correlations in acute post-streptococcal glomerulonephritis. Medicine (Baltimore) 1971;50:453-501.
- 17. Southwest Pediatric Nephrology study group, University of Texas, Health Sciences Centre at Dellas. Texas USA: A Clinico-pathologic study of crescentic glomerulonephritis in 50 children. Kidney Int 1985;27:450-8.
- Srivastava RN, Mudgil A, Bagga A, Vasudeva AS, Bhuyan UN, Karumassery PS. Crescentic glomerulonephritis in children: A review of 43 cases. Am J Nephrol 1992;12:155-61.
- 19. Albert MS, Leeming JM, Scaglione PR. Acute glomerulonephritis without abnormality of the urine. J Pediatr 1996;68:525-9.
- 20. Cohen JA, Levit MF. Acute glomerulonephritis with few urinary abnormalities. Report of two cases proved by renal biopsy. N Eng J Med 1963;268:749-53.
- 21. Dodge WF, Spargo BF, Travis LB, Srivastava RN, Carvajal HF, DeBeukelaer MM, *et al.* Post streptococcal glomerulonephritis. A prospective study in children. N Eng J Med 1972;286:273-8.