Clinical Spectrum and Outcome of Acute Postinfectious Glomerulonephritis in Children: A Hospital Based Study

Arulkumaran Arunagirinathan¹, Dinesh Kumar Narayanaswamy², Bharathkumar Thirunavukaransu², Anupriya Raghavan³, V D Raghavendhran⁴

¹Associate Professor, Department of Pediatrics, Sri Manakula Vinayagar Medical College and Hospital, Kalitheerthalkuppam, Madagadipet, Puducherry, India, ²Assistant Professor, Department of Pediatrics, Sri Manakula Vinayagar Medical College and Hospital, Kalitheerthalkuppam, Madagadipet, Puducherry, India, ³Post-graduate Student, Department of Pediatrics, Sri Manakula Vinayagar Medical College and Hospital, Kalitheerthalkuppam, Madagadipet, Puducherry, India, ⁴Professor & Head, Department of Pediatrics, Sri Manakula Vinayagar Medical College and Hospital, Kalitheerthalkuppam, Madagadipet, Puducherry, India

Abstract

Background: Post-infectious glomerulonephritis (PIGN) has a wide spectrum of clinical presentation and may mimic a wide variety of glomerular diseases.

Aim: To evaluate, the clinical presentation, complications, and outcome of acute PIGN in children.

Materials and Methods: A retrospective observational study of all children in the age group of 1-13 years who were diagnosed to have acute PIGN based on the clinical features, urine analysis, and C_3 levels admitted in Sri Manakula Vinayagar Medical College and hospital between January 2012 and December 2014 are included in this study. Case sheets were analyzed to obtain data on the clinical characteristics, laboratory parameters, complications, and outcome of these children. Descriptive analysis of the collected data was performed.

Results: Out of 52 cases of PIGN, 88.4% the etiology was post-streptococcal. Pneumonia (11.5%) was another important cause identified. There was no difference in the occurrence between males and females, and the majority of cases (52%) are in the age group of 5-10 years. Among clinical features, hypertension was observed in 100% of the cases. Hematuria, oliguria, and edema were seen in 58%, 94%, and 90% of the children, respectively. Laboratory parameters include the presence of hematuria (100%) in all cases. Though mild proteinuria was seen in the majority of cases, nephrotic range proteinuria was observed in 11.5% of cases. The complications include acute kidney injury in 27%, congestive cardiac failure in 15%, hypertensive emergency in 23%, and encephalopathy in 7% of cases. No mortality was observed.

Conclusion: Post-streptococcal glomerulonephritis is an important cause of acute PIGN. Children in the age group of 5-10 years are most affected. Acute kidney injury, congestive cardiac failure, and hypertensive encephalopathy are potential serious complications of this disease which requires intensive care monitoring to yield a positive outcome.

Key words: Acute kidney injury, Child, Glomerulonephritis, Hematuria, Streptococcal infections

INTRODUCTION

Post-infections glomerulonephritis (PIGN) occur due to immunologically mediated injury to the glomerulus

Month of Submission: 07-0000
Month of Peer Review: 07-0000
Month of Acceptance: 07-0000
Month of Publishing: 08-0000

by various infectious agents such as viral, bacterial, or protozoa organisms. 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.¹ The nephrogenic strains include those associated with cutaneous infections and pharyngitis with strains M4, M12, M25, and M49 being the most common.^{2,3} In tropical areas, there is tendency to have pyoderma associated PSGN⁴ where in temperate climate there is predominance of pharyngitis associated PSGN.^{5,6} The incidence of PSGN has decreased in the

Corresponding Author: Dr. Arulkumaran Arunagirinathanm, No: 41, Perumal Koil Street, Villupuram - 605 602, Tamil Nadu, India. Phone: +91-9789722422. E-mail: arukumaran76@gmail.com

developed world but in developing nation like India it is still an important public health problem.⁷ PIGN is one of the leading cause requiring hospital admissions in children,⁸ and it is also an important cause of acute renal failure in developing countries.⁹ Though deaths due to this disease are rare it can cause serious complications such as hypertensive emergency, congestive cardiac failure, renal failure, encephalopathy, and retinopathy.¹⁰ Recent data on clinical profile and complications of PIGN are very few in India hence the present study was under taken.

MATERIALS AND METHODS

This retrospective observational study was carried out at Sri Manakula Vinayagar Medical College and Hospital after due permission from the hospital authorities. All case records of children with the diagnosis of acute PIGN admitted from January 2012 to December 2014 were taken from the medical records department and the case sheets were analyzed. Children in the age groups of 1-13 years. Presenting with acute nephritic syndrome is included in the study. Acute nephritic syndrome was defined as acute onset of hematuria, hypertension and oliguria, and edema. 11 Acute PSGN was diagnosed in the presence of (a) features of acute nephritic syndrome, (b) evidence of recent streptococcal infection, and (c) lower serum complement C3 levels. PIGN was defined as features of acute nephritic syndrome combined with the evidence of an infectious etiology, e.g., PSGN, Pneumonia, Varicella, etc. Hematuria was defined as presence of 5 red blood cells per high power field on a centrifuged urinary specimen. 12 Hypertension was defined as systolic and or diastolic blood pressure values exceeding the 95th centile for age sex and height.¹³

The patients are reviewed with respect to the age, sex, skin or throat infection, blood pressure at admission, general, and systemic examination findings. The laboratory parameters included in the study were hemoglobin (Hb) at admission, urine analysis, blood urea, serum creatinine, serum albumin, serum cholesterol, urine spot protein creatinine ratio, antistreptolysin O (ASO titer), and serum complement C3 levels at admission. ASO titer >200 units/ml was considered as evidence of recent streptococcal infection. 14,15 Nephrotic range proteinuria was defined as urinary protein: Urinary creatinine ratio >2. Acute kidney injury was defined as an abrupt reduction in renal function leading to increase in serum creatinine >0.3 mg/dl, or a percentage increase in serum creatinine of more than or equal to 1.5 fold from the baseline.16 Details of the treatment given including the usage of one or more antihypertensives and the complications observed during the hospital stay, whether the child recorded fully or partially were all entered in the structured proforma. Full recovery at discharge was defined as absence of edema, hypertension, and normal renal function.¹²

The data were entered and analyzed using Epi info version 3.5.4. The clinical features, laboratory parameters, and outcome of the children were taken for analysis.

RESULTS

Of the 52 children diagnosed to have PIGN between January 2012 and December 2014, 46 (88.4%) was post-streptococcal in etiology. Upper respiratory tract infections, pyoderma and chicken pox preceded APGN in 17%, 6.5%, and 5.5% of the cases, respectively. Pneumonia (11.5%) was found to be an important cause of PIGN other than PSGN. The male to female ratio was 1.08:1. Age of patients ranged from 2.6 years to 13 years with large proportion of cases, 27(52%) in the age group of 5-10 years. Table 1 deficits the distribution of cases in different age groups.

Clinically, hypertension was noted in all cases (100%) at admission. Gross hematuria, oliguria, and edema were noted in 58%, 94%, and 90% of children, respectively. The other clinical features of these children include abdominal pain (23%), dysuria 11.5%, and fever (28.5%). Some presented with central nervous system manifestations (7%) such as seizures, headache, and/or altered sensorium, while few others (8%) presented with congestive cardiac failure. Table 2 elaborates the pattern of clinical presentation in PIGN.

Laboratory parameters of these children included the presence of hematuria in all cases by urine microscopy.

Table 1: Distribution of acute PIGN cases in different age groups

Age group	Number (n)	Percentage
1-5 years	21	40.3
5-10 years	27	52
>10 years	4	7.7

PIGN: Post-infectious glomerulonephritis

Table 2: Clinical presentation of acute PIGN

Signs and symptoms	Number (<i>n</i>)	Percentage
Hypertension	52	100
Stage I hypertension	36	69.3
Stage II hypertension	16	30.5
Oliguria	49	94
Generalized body swelling	47	90
Abdominal pain	12	23
Dysuria	6	11.5
Fever	15	28.5
Central nervous system manifestations	4	7
Cardiovascular system manifestations	8	15

PIGN: Post-infectious glomerulonephritis

Mild proteinuria was seen in many children, but nephrotic range proteinuria (Spot polymerase chain reaction >2) was seen in 6 (11.5%) of cases. ASO was positive in 24 (46%) of the children, and the C3 value was low in all (100%) of cases. Renal biopsy was not done any case. Tables 3 and 4 illustrate the various laboratory parameters observed in the present study.

The complications observed include acute kidney injury in 14 (27%) of the cases of which all improved by conservative management and none required dialysis. Other complications include hypertensive emergency 12 (23%), congestive cardiac failure in 8 (15%), and encephalopathy in 4 (7%) of cases. No case had hypertensive retinopathy in our study. Table 5 shows the various complications observed due to PIGN.

While most of them (72%) were managed with the use of only diuretics and calcium channel blockers, few required

Table 3: Laboratory parameters of the patients with PIGN

Laboratory characteristics	PIGN cases (n=52)	Percentage
Low Hb level at admission	25	48
Urine spot PCR		
0.2	14	27
0.2-2	32	61
>2	6	11
Raised renal function test	7	13.4
ASO positivity (>200 units/ml)	24	46
Low C3 value	52	100
Low serum albumin	13	25

ASO: Antistreptolysin O, PIGN: Post-infectious glomerulonephritis, PCR: Protein creatinine ratio, Hb: Hemoglobin

Table 4: Laboratory values in cases of PIGN

Laboratory parameters	Values*
Hb levels	9.3 (5.8-12)
Urea	40.75 (5-148)
Creatinine	0.644 (0.3-1.2)
C3 levels	0.37 (0.12-0.87)
S. albumin	3.67 (2.4-4.7)

*Values depicted as median (interquartile range), PIGN: Post-infectious glomerulonephritis

Table 5: Complications noticed in subjects due to acute PIGN

Complications	Number	Percentage	
Acute kidney injury			
Stage I	8	15	
Stage II	5	9.5	
Stage III	1	1.9	
Hypertensive emergency	12	23	
Hypertensive encephalopathy	4	7	
Congestive cardiac failure	8	15	

PIGN: Post-infectious glomerulonephritis

the need of other antihypertensives. Complete recovery was note in 76.9% of cases while few were discharged on oral medications for the control of hypertension.

DISCUSSION

Fifty-two children who had features of acute nephritic syndrome following an infectious cause were included in the study period of which 88.4% the etiology was found to be post-streptococcal. Similar observations were made by other studies.^{8,10,17}

There is no difference in male to female ratio, but the previous studies^{10,12,17} indicate a male preponderance. The lowest age of presentation was seen in a 2 years 6 months old child but many falls in the age group of 5-10 years, which was in accordance with previous studies.^{1,10,11}

Hypertension was noted in all patients. Cerebral complications of hypertension was seen in 7% of cases which is similar to the observations made by other studies, 10,18 but few studies 17 showed higher occurrences of central nervous system manifestations.

The percentage of children presenting with congestive cardiac failure (15%) was high which was similar to a previous studies^{10,18} However, in a study done by Lagunju *et al.*, ¹⁹ children presenting with cardiac failure was very low.

On analyzing the laboratory parameters all of them had hematuria by urine analysis. Proteinuria in the nephrotic range was seen in 6 (11.5%) of cases which was less compared to other studies^{6,10,20} were a higher percentage was noted. The Hb value was low (<10 g/dl) in 48% of cases. The serum albumin values in majority of them (64%) was between 2.6 g and 3.5 g per deciliter which is similar to observations made by Malla *et al.*,¹⁷ but in 25% of the children the serum albumin was very low (<2.5 g/dl). The low hemoglobin and serum albumin was probably due to the high prevalence of malnutrition among these children.

ASO titer was positive in 48% of children which was similar to other studies^{6,17,21} but in some studies¹⁰ the positivity was very low. Serum C3 value was low in all patients.

In the present study, acute kidney injury as per acute kidney injury network classification was seen in 27% of cases which is slightly higher compared to study done by Gunasekaran *et al.*¹⁰ where he reported an occurrence of 20.8%. The mean duration of hospital stay was 9.2 days. There was no mortality observed in the study period.

The present study is important as recent studies based on the clinical features of PIGN are very few and secondly it highlights the potential serious complications of this disease in detail so that timely interventions of these problems may bring down the morbidity and mortality of this illness.

The present study has a few limitations as it was a retrospective study, and the long-term outcome of these children could not be done as many were lost on follow-up in our nephrology clinic.

CONCLUSION

PSGN is the commonest cause of Acute PIGN. Acute kidney injury, congestive cardiac failure, and hypertensive encephalopathy are the dangerous complications of this disease which necessitates the need for monitoring these patients in pediatric intensive care set up. Though the incidence of this disease is strongly influenced by social, environmental and better economic conditions, the availability of a vaccine against Group A streptococcus in near future may curtain its occurrence.

ACKNOWLEDGMENT

The authors sincerely like to thank Mrs. Vijayalakshmi clerk, Sri Manakula Vinayagar Medical College, Puducherry for his immense help in typing the entire study.

REFERENCES

- Eison TM, Ault BH, Jones DP, Chesney RW, Wyatt RJ. Post-streptococcal acute glomerulonephritis in children: Clinical features and pathogenesis. Pediatr Nephrol 2011;26:165-80.
- Vinen CS, Oliveira DB. Acute glomerulonephritis. Postgrad Med J 2003;79:206-13.
- 3. Srivastava RN. Acute glomerulonephritis. Indian J Pediatr 1999;66:199-205.
- 4. 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.
- Ilyas M, Tolaymat A. Changing epidemiology of acute post-streptococcal glomerulonephritis in Northeast Florida: A comparative study. Pediatr Nephrol 2008;23:1101-6.
- Roy S 3rd, Stapleton FB. Changing perspectives in children hospitalized with poststreptococcal acute glomerulonephritis. Pediatr Nephrol 1990;4:585-8.
- Rodriguez-Iturbe B, Musser JM. The current state of poststreptococcal glomerulonephritis. J Am Soc Nephrol 2008;19:1855-64.
- 8. Barbiano Di Belgiojoso G, Genderini A, Ferrario F. Post-infectious glomerulonephritis. G Ital Nefrol 2003;20:184-99.
- 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.
- Gunasekaran K, Krishnamurthy S, Mahadevan S, Harish BN, Kumar AP. Clinical characteristics and outcome of post-infectious glomerulonephritis in children in Southern India: A prospective study. Indian J Pediatr 2015.
- Vijayakumar M. Acute and crescentic glomerulonephritis. Indian J Pediatr 2002:69:1071-5
- Sarkissian A, Papazian M, Azatian G, Arikiants N, Babloyan A, Leumann E. An epidemic of acute postinfectious glomerulonephritis in America. Arch Dis Child 1997;77:342-4.
- National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004;114 2 Suppl:555-76.
- Bisno AL, Stevens DL. Sreptococcus pyogenes. In: Mandell GL, Bennett JE, Dolin R, editors. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. Philadelphia: Elsevier; 2009. p. 198.
- Low DE. Non-pneumococcal streptococcal infections, rheumatic fever. In: Goldman L, Schafer A, editors. Goldman's Cecil Medicine. Philadelphia: Elsevier Saunders; 2011. p. 298.
- Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute Kidney Injury Network: Report of an initiative to improve outcomes in acute kidney injury. Crit Care 2007;11:R31.
- Malla K, Sarma MS, Malla T, Thaplial A. Varied presentations of acute glomerulonephritis in children: Single centre experience from a developing country. Sultan Qaboos Univ Med J 2008;8:193-9.
- Woo KT, Chiang GS, Edmondson RP, Wu AY, Lee EJ, Pwee HS, et al. Glomerulonephritis in Singapore: An overview. Ann Acad Med Singapore 1986;15:20-31.
- Lagunju IA, Omokhodion SI. Childhood heart failure in Ibadan. West Afr J Med 2003;22:42-5.
- Olowu WA. Systemic complications of acute glomerulonephritis in Nigerian children. Niger Postgrad Med J 2002;9:83-7.
- Travis LB. Acute postinfective glomerulonephritis. In: Rudolph AM, Hoffman JI, Rudolph CD, editors. Rudolph's Pediatrics. Stamford, CT: Appleton and Lange; 1996. p. 1356-8.

How to cite this article: Arunagirinathan A, Narayanaswamy DK, Thirunavukaransu B, Raghavan A, Raghavendhran VD. Clinical Spectrum and Outcome of Acute Post-infectious Glomerulonephritis in Children: A Hospital Based Study. Int J Sci Stud 2015;3(5):163-166.

Source of Support: Nil, Conflict of Interest: None declared.