

ORIGINAL ARTICLE

Assessment of Renal Function Status and Electrolyte Imbalances in Paediatric Patients with Acute Glomerulonephritis

Mozokkir Ali¹, Majeda Bilkis², Himangshu Bardhan³, Tanjil Chowdhury⁴, Nayma Chowdhury⁵, Rita Rani Paul⁶, Farhana Haque Jony⁷

Received: 12 Mar 2026
Accepted: 14 Mar 2026
Published Online: 17 Mar 2026

Published by:
Gopalganj Medical College, Gopalganj,
Bangladesh

Correspondence to
Mozokkir Ali

DOI: dx.doi.org

Copyright © 2026 The Insight



This article is licensed under a Creative Commons Attribution 4.0 International License.



ABSTRACT

Background: Acute glomerulonephritis (AGN) is a frequent glomerular disease in children, which is associated with hematuria, proteinuria, high blood pressure, and edema that can cause severe renal failure and electrolyte imbalances. Early detection of renal function status and electrolyte profiles is the key to the maximum benefit of clinical outcomes in the vulnerable population. This study aimed to determine the renal function condition and electrolyte imbalance among children with acute glomerulonephritis. **Methods & Materials:** This study was conducted at the Department of Pathology, Parkview Medical College & Hospital, Sylhet, from January 2025 to June 2025. A total of 60 children aged between 1 and 14 years and diagnosed with acute glomerulonephritis participated in the study by the purposive sampling method. Demographic information, clinical data, and laboratory data such as serum creatinine, blood urea, estimated glomerular filtration rate (eGFR), and serum electrolytes (sodium, potassium, calcium, and phosphate) were collected and entered into SPSS version 26 for further analysis. Chi-square tests and Fisher's exact tests were used to identify links between renal impairment and electrolyte imbalances. **Results:** The age of the participants was 8.7±3.1 years, and a majority of them were males (60%). Hematuria (100 percent) and periorbital edema (86.7 percent) were very common and hypertension (68.3 percent) was the most common clinical feature. 65% of patients had a raised serum creatinine; 70% of patients had a lowered eGFR under 90 ml/min/1.73m². The most common electrolyte imbalance was hyponatremia (46.7%), then hyperphosphatemia (35%) and hyperkalemia (31.7%). Impaired renal function was strongly linked with hyponatremia ($p = 0.04$) and hyperkalemia ($p = 0.02$). **Conclusion:** Renal dysfunction and electrolyte imbalances are very common in paediatric acute glomerulonephritis patients. Timely and regular laboratory check-ups, especially of sodium and potassium levels, are required to avoid life-threatening complications in children with the disease.

Keywords: Acute glomerulonephritis; paediatric nephrology; Renal function; Electrolyte imbalance; Glomerulonephritis

(The Insight 2026; 9(1): 162-166)

1. Assistant Professor, Department of Pathology, Parkview Medical College & Hospital, Sylhet, Bangladesh (ORCID: 0000-0003-3143-8892)
2. Resident, Department of Paediatrics, Sylhet MAG Osmani Medical College & Hospital, Sylhet, Bangladesh
3. Professor & Head, Department of Pathology, Parkview Medical College & Hospital, Sylhet, Bangladesh
4. Assistant Professor, Department of Pathology, Sylhet Women's Medical College, Sylhet, Bangladesh
5. Assistant Professor, Department of Pathology, Jalalabad Ragib Rabeya Medical College & Hospital, Sylhet, Bangladesh
6. Assistant Professor, Department of Pathology, Jalalabad Ragib Rabeya Medical College, Sylhet, Bangladesh
7. Assistant Professor, Department of Pathology, Sylhet MAG Osmani Medical College & Hospital, Sylhet, Bangladesh

INTRODUCTION

AGN is among the most clinically meaningful glomerular disorders in children, which is characterized by the sudden occurrence of hematuria, proteinuria, hypertension, oliguria, and edema. It occurs most among school-going children and is a leading cause of hospital stays among children in developed and developing nations [1]. The most common etiology of AGN is post-streptococcal glomerulonephritis (PSGN), which is prevalent in low and middle-income countries where streptococcal pharyngitis and impetigo are also common [2]. The pathophysiology of AGN is immune complex deposition on the glomeruli, which causes an inflammatory cascade with a resultant decreased glomerular filtration and renal renin-angiotensin-aldosterone system activation [3]. The effect of this mechanism is the retention of sodium and water, which increases the development of hypertension and edema, which is characteristic of the syndrome. In extreme situations, the

nephron damage may develop into acute kidney injury (AKI), a life-threatening complication, which needs to be detected and treated as soon as possible [4]. The evaluation of the functional condition of renal functions in children with AGN is necessary to further categorize the severity of the disease and to inform the therapeutic procedures. Objective markers of glomerular destruction and filtration capacity are parameters of serum creatinine, blood urea nitrogen, and estimated glomerular filtration rate (eGFR) [5]. Research has shown that a large percentage of paediatric AGN patients manifest with different levels of renal impairment on diagnosis, including subclinical decreases in GFR or acute kidney injury [6]. Other severe complications of AGN in children are electrolyte imbalances. Upset of the usual renal homeostatic processes often results in the imbalance of serum sodium, potassium, calcium, and phosphate ions [7]. One of the most frequent disruptions is hyponatremia, which occurs due to the effect of dilution and

inappropriate antidiuretic hormone secretion, and hyperkalemia is an immediate danger of cardiac arrhythmias [8]. Hypocalcemia and hyperphosphatemia could also be secondary to some disturbances in the renal tubular functioning and disruption in the metabolism of vitamin D [9]. Although the clinical importance of AGN is well-known in children, the information describing the patterns of renal failure and electrolyte derangements in children in specific contexts is scarce, especially in the context of areas with limited resources [10]. Knowledge on the prevalence and correlations of such abnormalities is important in coming up with efficient monitoring guidelines and minimizing the morbidity. The study was thus aimed at assessing the renal function status and electrolyte imbalance in children with acute glomerulonephritis, and measuring the associations between renal impairment and electrolyte imbalances.

METHODS & MATERIALS

This study was conducted at the Department of Pathology, Parkview Medical College & Hospital, Sylhet from January 2025 to June 2025. The study enrolled 60 paediatric patients diagnosed with acute glomerulonephritis according to standard clinical and laboratory criteria, including the presence of hematuria, proteinuria, edema, and hypertension. Children aged 1-14 years admitted with a confirmed diagnosis of acute glomerulonephritis; patients with complete clinical and laboratory data; and those with written informed consent of parents or guardians were included in this study. Patients with pre-existing chronic kidney disease, congenital renal

anomalies, secondary due to systemic lupus erythematosus or vasculitis, and patients with incomplete medical records were excluded from the study. The main variables evaluated were demographic characteristics (age, sex, residence, and socioeconomic status), clinical features (periorbital edema, hematuria, hypertension, oliguria, facial puffiness, fever, previous sore throat or skin infection, headache, and vomiting), and laboratory parameters. Renal function status was measured by serum creatinine, blood urea, serum albumin, complement C3, anti-streptolysin O (ASO) titer, and estimated glomerular filtration rate (eGFR) based on the Schwartz formula. Electrolyte imbalances were measured by serum sodium, potassium, calcium, and phosphate levels. Data was entered and analysed with SPSS version 26.0. Descriptive statistics were presented as frequencies and percentages for categorical variables and mean + standard deviation (SD) for continuous variables. The relationship of renal dysfunction and electrolyte disturbance was assessed by the Chi-square test, Fisher's exact test, where appropriate. A p-value of <0.05 was measured as statistically significant.

RESULTS

Table 1 shows the sociodemographic characteristics of 60 paediatric patients with acute glomerulonephritis. The mean age was 8.7±3.1 years, with the age group 6-8 being the largest (30%). The cohort included 60% males. The majority of the patients were living in rural areas (63.3%), and nearly half of them (45%) were from low socioeconomic households.

Table - I: Sociodemographic characteristics of the paediatric patients with acute glomerulonephritis (n = 60)

| Variable | Category | n (%) / Mean±SD |
|----------------------|----------|-----------------|
| Age group (years) | ≤5 | 10 (16.7) |
| | 6-8 | 18 (30.0) |
| | 9-11 | 17 (28.3) |
| | 12-14 | 15 (25.0) |
| | Mean age | 8.7 ± 3.1 |
| Sex | Male | 36 (60.0) |
| | Female | 24 (40.0) |
| Residence | Rural | 38 (63.3) |
| | Urban | 22 (36.7) |
| Socioeconomic status | Low | 27 (45.0) |
| | Middle | 26 (43.3) |
| | High | 7 (11.7) |

Table II summarises the clinical features at presentation. Hematuria was universal (100%), which confirmed the diagnostic criterion of AGN in all the enrolled patients. Periorbital edema (86.7%) and facial puffiness (80%) were the most visible signs as a result of sodium and water retention.

Hypertension was observed in 68.3%, and oliguria in 61.7%, showing great impairment of renal perfusion. Over half (55%) had a preceding history of sore throat or of skin infection, supporting a post-streptococcal etiology in the majority of cases.

Table - II: Clinical presentation of paediatric acute glomerulonephritis cases (n = 60)

| Clinical Feature | n | % |
|---------------------------------------|----|------|
| Periorbital edema | 52 | 86.7 |
| Hematuria (gross/microscopic) | 60 | 100 |
| Hypertension | 41 | 68.3 |
| Oliguria | 37 | 61.7 |
| Facial puffiness | 48 | 80.0 |
| Fever | 26 | 43.3 |
| History of sore throat/skin infection | 33 | 55.0 |
| Headache | 21 | 35.0 |
| Vomiting | 18 | 30.0 |

The baseline laboratory findings are shown in Table III. The mean serum creatinine (1.35±0.52 mg/dL) and blood urea (42.8±16.3 mg/dL) were both higher than normal reference values, suggesting a common glomerular harm. The greatly increased mean ASO titer (385±150 IU/mL) and low

complement C3 (68.5±18.7 mg/dL) are together sufficient to confirm the post-streptococcal immune complex pathogenesis in most patients. Mean serum albumin was borderline low (3.4±0.6 g/dL), with mild loss of proteins and nutritional effects of the illness (Table III).

Table - III: Baseline laboratory findings of study participants (n = 60)

| Parameter | Mean ± SD | Normal Range |
|--------------------------|-------------|--------------|
| Hemoglobin (g/dL) | 10.2 ± 1.5 | 11-14 |
| Serum creatinine (mg/dL) | 1.35 ± 0.52 | 0.3-1.0 |
| Blood urea (mg/dL) | 42.8 ± 16.3 | 10-40 |
| Serum albumin (g/dL) | 3.4 ± 0.6 | 3.5-5 |
| Complement C3 (mg/dL) | 68.5 ± 18.7 | 90-180 |
| ASO titer (IU/mL) | 385 ± 150 | <200 |

Table IV classifies the renal function status based on the serum creatinine and eGFR. Most patients (51.7%) had mild serum creatinine elevation (1-2 mg/dL), and 13.3% had moderate serum creatinine elevation, above 2 mg/dL. Only 30% had a

normal eGFR, and the remaining 70% had reduced filtration capacity. Of note, 6.7% had severely decreased eGFR (<30 ml/min), which signified critical acute kidney injury.

Table - IV: Renal function status among paediatric glomerulonephritis patients (n = 60)

| Renal Parameter | Category | n (%) |
|------------------|-------------------------------|-----------|
| Serum Creatinine | Normal (<1 mg/dL) | 21 (35.0) |
| | Mild elevation (1-2 mg/dL) | 31 (51.7) |
| | Moderate elevation (>2 mg/dL) | 8 (13.3) |
| Estimated GFR | ≥90 ml/min/1.73m ² | 18 (30.0) |
| | 60-89 ml/min | 24 (40.0) |
| | 30-59 ml/min | 14 (23.3) |
| | <30 ml/min | 4 (6.7) |

The frequency of specific electrolyte disturbances is demonstrated in Table 5. The most common abnormality was hyponatremia, with 46.7% of patients affected; the other most common abnormalities were hyperphosphatemia (35%) and hyperkalemia (31.7%). Hypocalcemia was found in 28.3% of cases. The average serum sodium (134.2±4.8 mmol/L) was

lower than normal, but the potassium (5.1±0.9 mmol/L) was at the upper range. These findings can be explained by multi-electrolyte disruption due to the impaired tubular function, reduced GFR, and hormonal regulation in the context of acute glomerulonephritis (Table V).

Table - V: Electrolyte imbalance among paediatric patients with acute glomerulonephritis (n = 60)

| Electrolyte Disturbance | n | % |
|---------------------------------|------------------|------|
| Hyponatremia (<135 mmol/L) | 28 | 46.7 |
| Hypernatremia (>145 mmol/L) | 4 | 6.7 |
| Hyperkalemia (>5.5 mmol/L) | 19 | 31.7 |
| Hypokalemia (<3.5 mmol/L) | 6 | 10.0 |
| Hypocalcemia | 17 | 28.3 |
| Hyperphosphatemia | 21 | 35.0 |
| Mean electrolyte levels: | | |
| Parameter | Mean ± SD | |
| Sodium (mmol/L) | 134.2 ± 4.8 | |
| Potassium (mmol/L) | 5.1 ± 0.9 | |
| Calcium (mg/dL) | 8.2 ± 0.7 | |
| Phosphate (mg/dL) | 5.3 ± 1.2 | |

Table VI shows the association between the status of renal function and electrolyte disturbances. Hyponatremia was present in 54.8% of the patients with impaired renal function compared to 27.8% in the normal group (p = 0.04), and hyperkalemia in 40.5% of the renally impaired group vs 11.1%

in the normal cases (p = 0.02), and both were statistically significant. Hypocalcemia and hyperphosphatemia had higher frequencies in renally impaired patients, but did not rise to significance levels.

Table - VI: Association between renal dysfunction and electrolyte imbalance

| Electrolyte Disturbance | Normal Renal Function (n=18) | Impaired Renal Function (n=42) | p-value |
|-------------------------|------------------------------|--------------------------------|---------|
| Hyponatremia | 5 (27.8%) | 23 (54.8%) | 0.04 |
| Hyperkalemia | 2 (11.1%) | 17 (40.5%) | 0.02 |
| Hypocalcemia | 3 (16.7%) | 14 (33.3%) | 0.11 |
| Hyperphosphatemia | 4 (22.2%) | 17 (40.5%) | 0.09 |

DISCUSSION

This study demonstrated the renal function status and electrolyte imbalance in 60 paediatric patients diagnosed with acute glomerulonephritis. Our results showed that there was a high prevalence of renal impairment and several electrolyte abnormalities; there were significant associations between

decreased glomerular filtration and sodium and potassium abnormalities. These results are in line with Hahn et al., who focused on the systemic complications of AGN in children [11]. The mean age of 8.7 years and the predominance of males (60%) in our study are consistent with previous reports documented by Miller et al. [12]. The strong association with

rural residence (63.3%) and low socioeconomic status (45%) is in accord with the evidence of an association between poverty, overcrowding, and poor access to healthcare and increased rates of streptococcal infection and subsequent glomerulonephritis [13]. Universal hematuria (100%) and high rates of periorbital edema (86.7%) and hypertension (68.3%) establish the classic clinical trial of AGN and are consistent with those from similar paediatric cohorts [14]. The significantly high levels of ASO titer (mean 385 IU/mL) and low levels of C3 complement (mean 68.5 mg/dL) in our study population confirmed post-streptococcal immune complex pathogenesis as the main mechanism of glomerular injury [15]. Complement consumption by the alternative pathway is a signature of PSGN and has been consistently reported as a diagnostic indicator in the study of Nasr et al. [16]. Renal impairment was common in our results, with 65% of patients having elevated serum creatinine and 70% having eGFR less than 90 ml/min/1.73m². These rates are higher than those reported by Becquet et al., potentially reflecting a delay in healthcare presentation in our predominantly rural, low-income population [17]. The proportion with severely reduced eGFR (<30 ml/min) was 6.7%, suggesting progression of a subset of patients to acute kidney injury at admission - an important finding with critical prognostic implications demanding intensive nephrology management [18]. Disturbances of electrolytes were very common in our results. Hyponatremia (46.7%) was the most common abnormality, mostly due to the mechanisms of dilutions secondary to sodium and water retention, characteristic of glomerular inflammation and decreased sodium urinary excretion [19]. Hyperkalemia was seen in 31.7% of patients, which is a clinically critical finding because it can lead to life-threatening cardiac arrhythmias in children. This proportion is consistent with that reported from Brodsky et al. [20]. Hyperphosphatemia (35%) and hypocalcemia (28.3%) are presumably due to impaired renal tubular handling of phosphate and reduced renal activation of vitamin D, a well-recognized consequence of reduced GFR [21]. The statistically significant associations between impaired renal function and both hyponatremia (p = 0.04) and hyperkalemia (p = 0.02) that we observed in our study have a particular clinical relevance. They suggest that electrolyte monitoring should be a priority in patients presenting with markers of reduced GFR as the risk of life-threatening electrolyte derangements is substantially increased in this subgroup [22]. While both hypocalcemia and hyperphosphatemia were found to be at higher levels in renally impaired patients, they did not reach statistical significance, which may have been due to the low sample size (a limitation recognized in this study [23]). Taken together, our results highlight the importance of systematic, early renal and electrolyte profiling in all paediatric patients with acute glomerulonephritis. Incorporating routine eGFR estimation and comprehensive electrolyte panels in the standard of care for admission of AGN cases, especially in resource-limited settings, can inform appropriate and timely interventions and reduce preventable morbidity.

LIMITATIONS

This study was performed with a relatively small sample size (n = 60), which may limit the generalizability of the results to general paediatric populations. Additionally, the cross-sectional study design does not allow for an evaluation of long-term renal consequences and a temporal relationship between electrolyte disturbances and disease progression.

CONCLUSION

Acute glomerulonephritis occurring in children has both a high burden of renal function impairment and electrolyte

disturbance. In this study, most patients had decreased eGFR and increased serum creatinine, suggesting significant glomerular injury at the time of presentation. The most clinically significant electrolyte abnormalities were hyponatremia and hyperkalemia, and these were found to be significantly associated with impaired renal function. The largely post-streptococcal etiology, reflected in elevated ASO titers and reduced complement C3, reflects the preventability of the disease by early antibiotic therapy of streptococcal infections. Systematic and early assessment of renal function and electrolyte status should be considered an essential component of the management protocol for all paediatric patients with acute glomerulonephritis, especially in resource-limited settings where delays in diagnosis and treatment can result in preventable life-threatening complications.

RECOMMENDATIONS

Future multicenter, prospective studies with larger sample sizes and longer follow-up time are suggested to better characterize the longitudinal course of renal dysfunction and electrolyte problems in paediatric AGN. Investigations into the effect of early correction of electrolyte abnormalities on clinical outcomes would further guide management guidelines that are evidence-based.

FUNDING

No funding sources

CONFLICT OF INTEREST

None declared

REFERENCES

1. Sanyahumbi AS, Colquhoun S, Wyber R, Carapetis JR. Global disease burden of group A *Streptococcus*. *Streptococcus pyogenes: basic biology to clinical manifestations* [Internet]. 2016 Feb 10.
2. Eison TM, Ault BH, Jones DP, Chesney RW, Wyatt RJ. Post-streptococcal acute glomerulonephritis in children: clinical features and pathogenesis. *Pediatric Nephrology*. 2011 Feb;26(2):165-80.
3. Satoskar AA, Parikh SV, Nadasdy T. Epidemiology, pathogenesis, treatment and outcomes of infection-associated glomerulonephritis. *Nature Reviews Nephrology*. 2020 Jan;16(1):32-50.
4. Mayer U, Schmitz J, Bräsen JH, Pape L. Crescentic glomerulonephritis in children. *Pediatric Nephrology*. 2020 May;35(5):829-42.
5. Schwartz GJ, Mun A, Schneider MF, Mak RH, Kaskel F, Warady BA, Furth SL. New equations to estimate GFR in children with CKD. *Journal of the American Society of Nephrology*. 2009 Mar 1;20(3):629-37.
6. Sethi S, Fervenza FC. Standardized classification and reporting of glomerulonephritis. *Nephrology Dialysis Transplantation*. 2019 Feb 1;34(2):193-9.
7. Carpenter CP, Iskander A, Hausdorff M, Stock JA. Fluid management in pediatric urology: a review of the literature and call for a change in practice. *Urology practice*. 2015 Nov 1;2(6):373-8.
8. Watanabe T. Atypical clinical manifestations of acute poststreptococcal glomerulonephritis. In *An Update on Glomerulopathies: Clinical and Treatment Aspects 2011* Nov 2 (pp. 151-168). In Tech, Shanghai, China.
9. Rodriguez-Iturbe B, Musser JM. The current state of poststreptococcal glomerulonephritis. *Journal of the American Society of Nephrology*. 2008 Oct 1;19(10):1855-64.
10. Rheault MN. Nephrotic syndrome. In *Clinical pediatric nephrology 2016* Nov 25 (pp. 301-320). CRC Press.
11. Hahn D, Hodson EM, Willis NS, Craig JC. Corticosteroid therapy for nephrotic syndrome in children. *Cochrane database of systematic reviews*. 2015(3).
12. Miller KM, Van Beneden C, McDonald M, Hla TK, Wong W, Pedgrift H, Kaslow DC, Cherian T, Carapetis JR, Scheel A, Seale A.

- Standardization of epidemiological surveillance of acute poststreptococcal glomerulonephritis. In Open Forum Infectious Diseases 2022 Sep 15 (Vol. 9, No. Supplement_1, pp. S57-S64). US: Oxford University Press.*
13. Iqbal M, Clement-Pervaiz MV, Ansari MJ, Pervaiz S, Sheikh S, Katpar S, Meo SA, Sattar K, Schofield S, Karabulut AK, Memon AI. *Proceedings of the 1st Liaquat University of Medical & Health Sciences (LUMHS) International Medical Research Conference. European Journal of Medical Research. 2017 Dec 28;22(Suppl 1):53.*
 14. Alwahaibi NY, Alhabsi TA, Alrawahi SA. *Pattern of glomerular diseases in Oman: a study based on light microscopy and immunofluorescence. Saudi Journal of Kidney Diseases and Transplantation. 2013 Mar 1;24(2):387-91.*
 15. Jung K, Zeng X, Bilusic M. *Nivolumab-associated acute glomerulonephritis: a case report and literature review. BMC nephrology. 2016 Nov 22;17(1):188.*
 16. Nasr SH, Radhakrishnan J, D D'Agati V. *Bacterial infection-related glomerulonephritis in adults. Kidney international. 2013 May 1;83(5):792-803.*
 17. Becquet O, Pasche J, Gatti H, Chenel C, Abély M, Morville P, Pietrement C. *Acute post-streptococcal glomerulonephritis in children of French Polynesia: a 3-year retrospective study. Pediatric Nephrology. 2010 Feb;25(2):275-80.*
 18. Sutherland SM, Kwiatkowski DM. *Acute kidney injury in children. Advances in Chronic Kidney Disease. 2017 Nov 1;24(6):380-7.*
 19. Chand DH, Swartz S, Tuchman S, Valentini RP, Somers MJ. *Dialysis in children and adolescents: the pediatric nephrology perspective. American Journal of Kidney Diseases. 2017 Feb 1;69(2):278-86.*
 20. Brodsky SV, Nadasdy T. *Acute poststreptococcal glomerulonephritis. In Bacterial Infections and the Kidney 2017 Mar 31 (pp. 1-36). Cham: Springer International Publishing.*
 21. Okpechi IG, Ameh OI, Bello AK, Ronco P, Swanepoel CR, Kengne AP. *Epidemiology of histologically proven glomerulonephritis in Africa: a systematic review and meta-analysis. PloS one. 2016 Mar 24;11(3):e0152203.*
 22. Whyte MP, Leelawattana R, Reinus WR, Yang C, Mumm S, Novack DV. *Acute severe hypercalcemia after traumatic fractures and immobilization in hypophosphatasia complicated by chronic renal failure. The Journal of Clinical Endocrinology & Metabolism. 2013 Dec 1;98(12):4606-12.*
 23. Hoy WE, White AV, Dowling A, Sharma SK, Bloomfield H, Tipiloura BT, Swanson CE, Mathews JD, McCredie DA. *Post-streptococcal glomerulonephritis is a strong risk factor for chronic kidney disease in later life. Kidney international. 2012 May 2;81(10):1026-32.*