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Pediatric Acute Glomerulonephritis: Correlation of Clinical Presentation with early complications and histopathology

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ABSTRACT

Background: *Acute glomerulonephritis (AGN) is a major cause of illness in children. It is present in different ways and can lead to complications that affect long-term health. This study examines how clinical presentation, complications, and histopathology in children with acute glomerulonephritis correlate to improve risk assessment and treatment.* ***Methods & Materials:*** *This observational study involved 50 patients aged between 1 to 18 years who were diagnosed with AGN and admitted to a tertiary care hospital. We collected detailed clinical, biochemical, and histopathological data using structured forms. Renal biopsies were done on 18 patients. We used SPSS version 26 for statistical analysis and applied Pearson's correlation coefficient to check associations among clinical presentations, complications, and histopathological features.* ***Results:*** *The average age of the patients was 8.24±3.2 years, with a predominance of males (70%). Common symptoms included facial puffiness (96%), oliguria (98%), hypertension (98%), and hematuria (90%). Early complications occurred in 60% of the patients, while 40% had no complications. Hypertension and oliguria showed strong links to acute kidney injury (AKI), ICU admission, and heart failure. Histopathological findings indicated that endocapillary hypercellularity correlated strongly with clinical severity. The average hospital stay was 10.2±5.2 days, and 94% of the patients improved by the time of discharge.* ***Conclusion:*** *Clinical factors, especially oliguria, hypertension, and anuria, are reliable indicators of early complications and histological severity in pediatric AGN. These findings support using clinical markers for risk assessment and early intervention, which could improve patient outcomes and decrease the need for invasive diagnostic tests.*

Keywords: *Pediatric acute glomerulonephritis, Clinical-pathology, acute glomerulonephritis complications, Histopathology.*

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INTRODUCTION

Acute glomerulonephritis (AGN) is one of the most significant renal diseases in children worldwide, characterized by immune-mediated damage to the glomerular basement membrane, mesangium, or capillary endothelium [1]. It manifests with hematuria, proteinuria, and azotemia, typically accompanied by the classic triad of edema, hypertension, and oliguria [2]. Post-streptococcal glomerulonephritis is the most common form of AGN in children, particularly in developing countries where streptococcal infections are prevalent [3]. The clinical presentation of pediatric AGN is very diverse, ranging from asymptomatic microscopic hematuria to rapidly progressive glomerulonephritis with acute kidney injury requiring dialysis [4]. Acute post-streptococcal glomerulonephritis, as seen in research, usually presents with gross hematuria, mild edema, oliguria, hypertension, and some renal insufficiency, with school-aged children most commonly affected [5]. The pathophysiology is immune complex deposition with complement activation and an inflammatory cascade resulting in glomerular injury and dysfunction [6]. Early recognition and appropriate management of AGN complications are necessary to prevent long-term sequelae. Generalized edema, hypertension, and hematuria have been the most common presenting symptoms in recent publications, while persistent hypertension, renal impairment, and proteinuria can signal a more severe clinical course that may require renal biopsy [7]. Evolution to complications such as acute kidney injury, hypertensive

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encephalopathy, and heart failure significantly influences patient outcomes and needs intensive care management [8]. Histopathology remains the gold standard for making a definitive diagnosis and identifying the types of glomerulonephritis. Immunofluorescence typically shows characteristic patterns, e.g., poststreptococcal glomerulonephritis with irregular, coarse granular deposits of polyclonal IgG and C3 along glomerular capillary walls [9]. Renal biopsy is not always feasible in children due to the risks and technical challenges involved, and thus correlation with clinical presentation and histopathological findings is particularly helpful [10]. Correlation of the clinical presentation with the initial complications and histopathology can significantly improve the management options for the patients. While the majority of the patients recover completely, there remains a 3-6% risk of chronic kidney disease development, which calls for the identification of predictive factors for poor outcomes [11]. The aim of this study is to assess the correlation between clinical presentation, early complications, and histopathology in children presenting with acute glomerulonephritis to improve risk stratification and direct therapeutic interventions in this high-risk population.

METHODS & MATERIALS

This observational study was conducted at the pediatric nephrology department of Bangladesh Shishu Hospital & Institute from October 2024 to March 2025. Fifty patients aged 1 to 18 years diagnosed with acute glomerulonephritis (AGN) were admitted to a tertiary care hospital and included in the study. Structured data collection form was used to gather detailed information, including demographic characteristics, immune status, and history of recent infections. Clinical features at presentation-including hematuria, proteinuria, oliguria, oedema, facial puffiness, hypertension, and systemic complications meticulously documented. Renal biopsy and histopathological examinations were performed among patients with RPGN, persistent hypertension more than 10days, nephrotic range proteinuria beyond 2 weeks and AKI persisting more than 10 days. Patient outcomes were evaluated based on the resolution of symptoms, renal function recovery, complications, ICU requirement, and hospital stay duration.

Data were compiled, checked, and analyzed using IBM SPSS Statistics version 26. Descriptive statistics were used to summarise the baseline characteristics, clinical features, biochemical parameters, and histopathological findings. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. Pearson's correlation coefficient was applied to assess the strength and direction of association between clinical presentations and early complications, as well as histopathological features. A *p*-value of less than 0.05 was considered statistically significant.

RESULTS

Table I provides the demographic and baseline characteristics of the study population. Most patients (70%) were aged between 5 and 10 years, and there was a strong male majority (70%). A rural residence was more common (64%), and many patients came from a lower-middle-class socioeconomic background (54%). The average BMI was 15.8±3.2 kg/m^2, and all patients were immunocompetent. [Table I].

Table – I: Distribution of study population based on basic characteristics (*n*=50)

Basic characteristics	N/ Mean±SD	Percentage (%)
Age		
<1 yr	0	0
1-3 yr	1	2
> 3-5 yrs	4	8
> 5-10 yrs	35	70
>10-18 yrs	10	20
Gender		
Male	35	70
Female	15	30
Residence		
Rural	32	64
Urban	18	36
Socioeconomic condition		
Poor	15	30
Middle class	7	14
Upper Mid Class	1	2
Lower Middle class	27	54
BMI		
Mean±SD	15.8 ± 3.2 Minimum 11.01 kg/m^2 Maximum 25.20 kg/m^2	
Immune status	50	100
Mean±SD	8.24 ± 3.2 with Min 3 days Max 15 days	
Duration of illness		
≤ 7 days	32	64
≤ 7 days	18	36

Clinical symptoms at presentation in Table II showed a typical nephritic syndrome pattern. Almost all patients had hypertension (98%) and oliguria (98%), while facial swelling was noted in 96% of cases. Hematuria was present in 90% of patients, and proteinuria was found in 78%. Systemic complications included encephalopathy (18%), convulsions (20%), heart failure (10%), and pneumonia (14%). Only 2% of patients had anuria, and ascites was seen in 14% of cases, indicating a range of disease severity at presentation. A history of sore throat was found in 54% of cases. Skin infections were noted in 28% of patients, with fever associated in 42% of cases. [Table II].

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Table – II: Distribution of study population based on clinical presentation at admission (*n* = 50)

Clinical presentation at admission	Number	Percentage (%)
Gross Hematuria	45	90
Proteinuria	39	78
Oliguria	49	98
Anuria	1	2
Oedema	22	44
Facial Puffiness	48	96
Ascites	7	14
Hypertension	49	98
Pneumonia	7	14
Encephalopathy	9	18
Heart Failure	5	10
Convulsion	10	20
H/O sore throat	27	54
H/O skin infection	14	28
Associated fever	21	42

Laboratory tests in Table III showed significant kidney involvement and systemic effects. Moderate proteinuria (0.5-2.0 mg/mg) was the most common finding (56%), whereas severe proteinuria was rare (4%). Serum albumin was moderately low in 56% of patients. Complement C3 levels were reduced for all the patients (100%), and C4 level was raised in 4% patinets. Most patients (70%) had elevated serum creatinine levels above 0.9 mg/dL, with 96% meeting stage 3 CKD by GFR criteria. ASO titers were elevated (≥200 IU/ml) in 98% of patients, while ANA and anti-dsDNA were negative in all cases, supporting a post-infectious cause. [Table III].

Table – III: Distribution of study population based on Biochemical parameters (*n*=50)

Biochemical Parameter	N	Percentage (%)
Urine Culture Positive	10	20
Urine Protein Present	45	90
Urine RBC	48	64
Urine Pus cell Present	16	32
Urine Hyaline Cast	1	2
Urine RBC Cast	2	4
Spot PCR (mg/mg) Nephrotic Range Proteinuria	2	4
S. Albumin (g/L) Reduced	35	70
S. Cholesterol (mmol/L) Raised	8	16
S. C3 Level (gm/L) Reduced	50	100
S. C4 Level (gm/L) Raised	2	4
GFR Level (ml/1.73m^2) Reduced	48	96
S. Creatinine Level (mg/dL) Raised	35	70
Blood Urea(mmol/L) Raised	25	50
ASO titre (IU/ml) Raised	42	84
ANA Negative	50	100
Anti DsDNA Negative	50	100
Sodium (mmol/L)		
Hyponatremia	3	6
Hypernatremia	6	12
Potassium (mmol/L)		
Hypokalemia	0	0
Hyperkalemia	7	14
Chloride (mmol/L)		
Hypochloremia	6	12
Hyperchloremia	15	30

Histopathological examination was done on 18 patients (36%), showing widespread glomerular enlargement and increased matrix, which is demonstrated in Table IV. Endocapillary hypercellularity was most common (61.11%), and mesangial hypercellularity occurred in 27.78% of cases. Crescentic changes were seen in 5.55% of patients. Immunofluorescence analysis showed variable intensity of immune deposits, with IgM being the most prominent (72% with 2+ or 3+ staining), followed by IgA (72%) and C3 (66%). The most common diagnosis was infection-associated glomerulonephritis (50%), with crescentic forms indicating more severe disease patterns. [Table IV].

Table – IV: Distribution of study population based on Renal Histopathology (*n*=18)

Renal Histopathology	N	Percentage (%)
Glomerular Size		
Enlarge glomeruli	18	100
Cellularity		
Endocapillary hypercellularity	11	61.11
Mesangial Hypercellularity	1	5.55
Endocapillary & Mesangial Hyper Cellularity	5	27.78
Endocapillary hypercellularity With the Presence of crescents	1	5.55
Matrix Increased	18	100
GBM		
Irregularly thick	2	11.11
Mildly thick	2	11.11
Not thick	14	77.78
IgG		
Trace	3	16.67
1+	10	55.55
2+	5	27.78
IgM		
Trace	2	11.11
1+	3	16.67
2+	9	50
3+	4	22.22
IgA		
Trace	1	5.55
1+	4	22.22
2+	7	38.89
3+	6	33.33
C3		
Trace	3	16.67
1+	5	27.78
2+	7	38.89
3+	1	5.55

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Diagnosis		
Infection associated with GN	9	50
Infection-associated proliferative GN	3	16.67
Proliferative Crescentic GN	1	5.55
Infection associated with Crescentic GN	1	5.55
Post-infectious Proliferative GN	4	22.22

Table V shows that the treatment is varied based on disease severity and complications. All patients needed furosemide for fluid management, while 82% required calcium channel blockers for hypertension. Corticosteroids were given to 50% of patients (14% received intravenous methylprednisolone, 36% took oral prednisolone). Mycophenolate mofetil was used in 16% of cases with severe disease. Hemodialysis was necessary for 12% of patients with acute kidney injury, while no patients required peritoneal dialysis. Antibiotics were prescribed to 42% of patients for concurrent or suspected bacterial infections. [Table V].

Table – V: Distribution of study population based on Treatment (*n*=50)

Treatment	N	Percentage (%)
Hemodialysis	6	12
Methylpednisolon	7	14
Oral Pednisolon	18	36
MMF	8	16
Frusemide	50	100
Calcium channel blocker	41	82
Antibiotic	21	42

Clinical outcomes, which are shown in Table VI, were generally positive, with 94% of patients showing improvement at discharge. Early complications occurred in 60% of patients, with acute kidney injury being common (10% isolated and 6% with other complications). Other major complications included septicemia (8%), respiratory issues (8%), and rapidly progressing glomerulonephritis (10%). ICU admission was necessary for 26% of cases. [Table VI].

Table – VI: Distribution of study population based on Complications (*n*=50)

Outcome	N/Mean±SD	Percentage (%)
Complication		
AKI	2	4
AKI with other complications	3	6
CRBSI	1	2
Encephalopathy	5	10
Heart Failure	3	6
Complications in the respiratory tract	3	6
RPGN	2	4
RPGN with other complications	4	8
Septicemia	20	40
UTI	13	2
No complications		
ICU Needed		

Table VII represents the distribution of outcomes among the study population (N = 50). Most participants, 94%, showed improvement. A small number, 6%, were discharged with ongoing hypertension (HTN). Recovery times varied: hypertension resolved in 9.9±5.1 days, swelling in 6.7±4.3 days, and gross hematuria in 7.4±5.0 days. The average hospital stay was 10.2±5.2 days, and only 6% were discharged with ongoing hypertension. [Table VII].

Table – VII: Distribution of the Study population based on Outcome (*n*=50)

Outcome	N	Percentage (%)
Improved	47	94
Discharged with persistent HTN	3	6
Duration to resolve HTN Mean±SD		9.9 ± 5.1 Min 4 days Max 30 days
Duration to resolve Oedema Mean±SD		6.7 ± 4.3 Min 2 days Max 28 days
Duration to resolve gross hematuria Mean±SD		7.4 ± 5.0 Min 3 days Max 21 days
Duration to improve renal function		9.9 ± 5.5 Min 3 days Max 30 days
Mean Hospital Stay		10.2 ± 5.2 Min 4 days Max 30 days

The correlation analysis in Table VIII shows strong statistical links between certain clinical features and complications. Hypertension had the strongest association with acute kidney injury (r=0.55, p=0.002), heart failure (r=0.39, p=0.03), and ICU admission (r=0.50, p=0.01). Oliguria also had a strong correlation with AKI (r=0.52, p=0.005), encephalopathy (r=0.44, p=0.01), and ICU requirement (r=0.56, p=0.002). Although rare (2%), anuria had the highest correlation coefficients with serious complications, particularly AKI (r=0.61, p=0.001) and ICU admission (r=0.58, p=0.002). Proteinuria and facial puffiness showed moderate correlations with neurological complications. [Table VIII].

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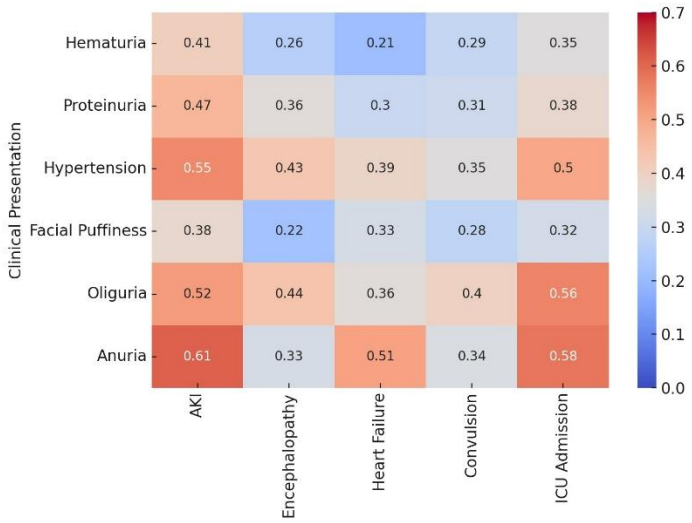
Table – VIII: Correlation between Clinical Presentations and Early Complications

Clinical Presentation	AKI	Encephalopathy	Heart Failure	Convulsion	ICU Admission
Hematuria	r = 0.41, p = 0.03	r = 0.26, p = 0.09	r = 0.21, p = 0.16	r = 0.29, p = 0.08	r = 0.35, p = 0.05
Proteinuria	r = 0.47, p = 0.01	r = 0.36, p = 0.04	r = 0.30, p = 0.07	r = 0.31, p = 0.06	r = 0.38, p = 0.03
Hypertension	r = 0.55, p = 0.002	r = 0.43, p = 0.02	r = 0.39, p = 0.03	r = 0.35, p = 0.04	r = 0.50, p = 0.01
Facial Puffiness	r = 0.38, p = 0.03	r = 0.22, p = 0.13	r = 0.33, p = 0.05	r = 0.28, p = 0.10	r = 0.32, p = 0.07
Oliguria	r = 0.52, p = 0.005	r = 0.44, p = 0.01	r = 0.36, p = 0.04	r = 0.40, p = 0.03	r = 0.56, p = 0.002
Anuria (2%)	r = 0.61, p = 0.001	r = 0.33, p = 0.05	r = 0.51, p = 0.01	r = 0.34, p = 0.06	r = 0.58, p = 0.002



Heatmap 1 shows the correlation between clinical presentations and early complications in pediatric acute glomerulonephritis. It reveals that anuria and oliguria have the highest associations with severe complications such as acute kidney injury (AKI) and ICU admission, with correlation coefficients reaching up to 0.61 and 0.58, respectively. Hypertension also shows strong links to complications like AKI (r = 0.55) and heart failure (r = 0.39), underscoring its role as a critical early marker. Proteinuria and facial puffiness exhibit moderate correlations, particularly with neurological complications such as encephalopathy and convulsions

Clinical-pathological correlations in Table IX. Oliguria had the strongest correlation with endocapillary hypercellularity (r=0.65, p=0.001), meningeal hypercellularity (r=0.44, p=0.02), and C3 deposition (r=0.48, p=0.02). Hypertension also correlated with endocapillary changes (r=0.62, p=0.001) and crescent formation (r=0.38, p=0.04). Anuria, despite being infrequent, showed strong associations with severe histological features, including endocapillary hypercellularity (r=0.69, p<0.001) and crescent formation (r=0.61, p=0.01).[Table IX].

Table – IX: Correlation between Clinical Presentations and Histopathological Features

Clinical Presentation	Endocapillary Hypercellularity	Mesangial Hypercellularity	Crescent Formation	IgA Deposition	C3 Deposition
Hematuria	r = 0.58, p = 0.01	r = 0.35, p = 0.06	r = 0.26, p = 0.10	r = 0.22, p = 0.14	r = 0.30, p = 0.08
Proteinuria	r = 0.50, p = 0.02	r = 0.28, p = 0.09	r = 0.31, p = 0.07	r = 0.33, p = 0.06	r = 0.42, p = 0.03
Hypertension	r = 0.62, p = 0.001	r = 0.41, p = 0.03	r = 0.38, p = 0.04	r = 0.29, p = 0.08	r = 0.45, p = 0.02
Facial Puffiness	r = 0.49, p = 0.02	r = 0.26, p = 0.11	r = 0.22, p = 0.14	r = 0.25, p = 0.11	r = 0.33, p = 0.06
Oliguria	r = 0.65, p = 0.001	r = 0.44, p = 0.02	r = 0.40, p = 0.03	r = 0.31, p = 0.07	r = 0.48, p = 0.02
Anuria (2%)	r = 0.69, p < 0.001	r = 0.36, p = 0.05	r = 0.61, p = 0.01	r = 0.30, p = 0.09	r = 0.53, p = 0.01

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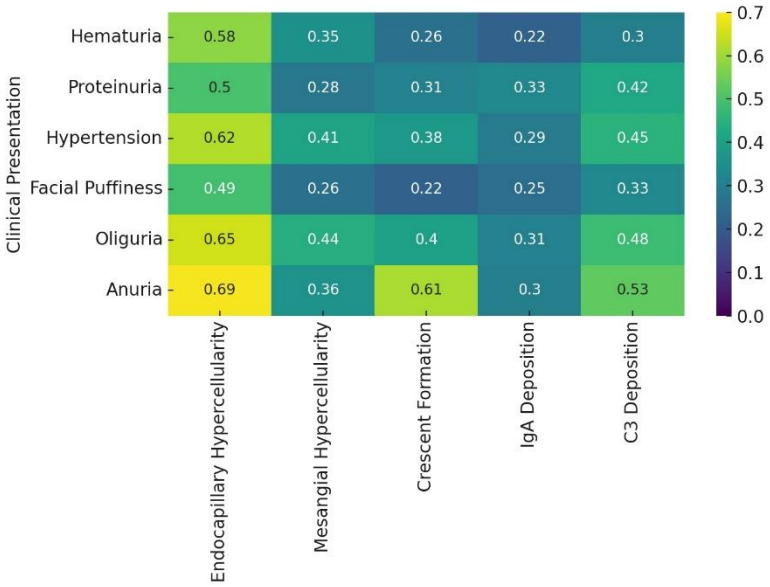

Heatmap 2 highlights the relationship between clinical presentations and histopathological features observed in renal biopsy. Anuria and oliguria again demonstrate the strongest correlations with severe histological changes, including endocapillary hypercellularity (r = 0.69 for anuria) and crescent formation (r = 0.61). Hypertension also correlates significantly with endocapillary changes and immune complex deposition, suggesting more aggressive kidney involvement.

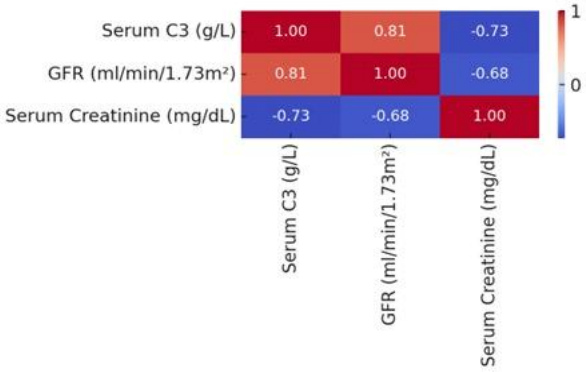

Figure – 3: Pearson Correlation between Serum C3, GFR, and Serum Creatinine

This heatmap demonstrates the strong correlation among Serum C3, GFR, and Serum Creatinine levels in the study population. The Pearson correlation analysis between serum C3, GFR, and serum creatinine revealed strong interrelationships that align with the expected pathophysiological patterns in glomerulonephritis. Serum C3 demonstrated a strong positive correlation with GFR (r = 0.81), indicating that higher complement levels are associated with better renal function. Conversely, serum C3 showed a strong negative correlation with serum creatinine (r = –0.73), suggesting that as complement levels decline, often due to ongoing immune-mediated kidney injury, while serum creatinine rises, reflecting reduced filtration capacity. GFR also showed a strong inverse correlation with serum creatinine (r = –0.68), consistent with the well-established clinical marker relationship in kidney function assessment.

DISCUSSION

This study of fifty pediatric patients with acute glomerulonephritis provides valuable insights into the clinical-pathological correlations and predictive factors for early complications. The demographic profile, with a predominance of school-age children and male gender, aligns with established epidemiological patterns of post-streptococcal glomerulonephritis in developing countries, which is shown by Nasr et al. [12]. Post-streptococcal glomerulonephritis remains one of the most common causes of acute nephritis among children, particularly in regions with limited access to healthcare and poor hygiene conditions [13]. The clinical presentation in our cohort demonstrated the classic nephritic syndrome with near-universal presence of hypertension (98%), oliguria (98%), and facial puffiness (96%). These findings are consistent with Pinto et al., that the characteristic triad of hematuria, oedema, and hypertension that defines acute glomerulonephritis results from sodium and water retention in the setting of renal impairment [14]. The

high prevalence of elevated ASO titers (98%) and negative autoimmune markers supports the post-infectious aetiology in the majority of cases [15]. The correlation analysis revealed significant associations between clinical parameters and both early complications and histopathological severity. The strong correlation between oliguria, hypertension, and adverse outcomes (AKI, ICU admission, heart failure) underscores the importance of these clinical markers for risk stratification. Early recognition of rapidly progressive patterns is crucial to prevent further renal function loss [16]. Although anuria was uncommon (2%), it demonstrated the highest correlation coefficients with severe complications, emphasising its clinical significance as a marker of advanced disease [17]. The histopathological findings in our study population revealed predominant endocapillary hypercellularity (61.11%) with variable immune complex deposition patterns. 50% patients of this had shown infection associated with GN, while post-infection proliferative GN was shown 22.2% that differs now-a-days. The correlation between immunofluorescence findings and clinical course has important prognostic implications [18]. The strong correlation between clinical severity markers (oliguria, hypertension) and histological features (endocapillary hypercellularity, crescent formation) suggests that clinical assessment can provide valuable information about underlying pathological severity even when renal biopsy is not immediately available [19].

Treatment outcomes were generally favourable, with 94% of patients showing improvement at discharge. However, the requirement for ICU admission in 26% of cases and dialysis in 12% highlights the potential severity of this condition. While most children recover completely, the 3-6% risk of progression to chronic kidney disease necessitates careful long-term follow-up [20]. The mean hospital stays of 10.2±5.2 days reflect the need for close monitoring and management of complications during the acute phase [21]. The biochemical profile demonstrated notable patterns, with universal C3 reduction (100%), a finding lower than expected compared with historical cohorts, possibly reflecting earlier presentation or regional disease variation.

Limitations of the Study:

The study took place at a single tertiary care centre, which may limit how applicable the findings are to other groups and healthcare settings. Kidney tissue examinations were done in only 36% of patients, which may cause bias towards more severe cases and restrict a complete assessment of clinical and pathological connections.

CONCLUSION

Clinical indicators such as reduced urine output, elevated blood pressure, anuria and associated infection serve as dependable predictors of early complications and kidney injury in children with acute glomerulonephritis. The close association between these clinical features and pathological findings suggests that non-invasive markers can be effectively used to assess risk and guide treatment decisions. These findings emphasize the importance of early monitoring of oliguria and hypertension to promptly identify high-risk patients requiring intensive care and timely intervention.

RECOMMENDATIONS

Future studies across multiple centers with larger groups and consistent biopsy methods are needed to confirm these clinical and pathological links in different populations. Long-term follow-up studies should also be conducted to check how well early clinical signs predict the development of chronic kidney disease and to create evidence-based guidelines for assessing risk in children with acute glomerulonephritis. Research into new biomarkers and non-invasive diagnostic tools could further improve the early identification of high-risk patients and lead to better treatment results.

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Conflict of interest: *None declared*

Ethical approval: *The study was approved by the Institutional Ethics Committee*

REFERENCES

1. *Anders HJ, Kitching AR, Leung N, Romagnani P. Glomerulonephritis: immunopathogenesis and immunotherapy. Nature Reviews Immunology. 2023 Jul;23(7):453-71.*
2. *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.*
3. *Hahn RG, Knox LM, Forman TA. Evaluation of poststreptococcal illness. American family physician. 2005 May 15;71(10):1949-54.*
4. *Dhakal AK, Shrestha D, Singh SK, Acharya S. Clinical profile of children with acute post-streptococcal glomerulonephritis. Pediatric Nephrology. 2023 Oct;38(10):3327-36.*
5. *Nasr SH, Radhakrishnan J, D D'Agati V. Bacterial infection–related glomerulonephritis in adults. Kidney international. 2013 May 1;83(5):792-803.*
6. *Mastrangelo A, Serafinelli J, Giani M, Montini G. Clinical and pathophysiological insights into immunological mediated glomerular diseases in childhood. Frontiers in Pediatrics. 2020 May 12;8:205.*
7. *Lim WH, Pleass H. Early Medical and Surgical Complications After Kidney Transplantation. Evidence-Based Nephrology. 2022 Dec 2;2:271-93.*
8. *Kanjanabuch T, Kittikowit W, Eiam-Ong S. An update on acute postinfectious glomerulonephritis worldwide. Nature Reviews Nephrology. 2009 May;5(5):259-69.*
9. *Simon P, Ramee MP, Boulahrouz R, Stanescu C, Charasse C, Ang KS, Leonetti F, Cam G, Laruelle E, Autuly V, Rioux N. Epidemiologic data of primary glomerular diseases in western France. Kidney international. 2004 Sep 1;66(3):905-8.*
10. *Couser WG. Glomerulonephritis. The Lancet. 1999 May 1;353(9163):1509-15.*
11. *Tangri N, Kitsios GD, Inker LA, Griffith J, Naimark DM, Walker S, Rigatto C, Uhlig K, Kent DM, Levey AS. Risk prediction models for patients with chronic kidney disease: a systematic review. Annals of internal medicine. 2013 Apr 16;158(8):596-603.*
12. *Nasr SH, Markowitz GS, Stokes MB, Said SM, Valeri AM, D'Agati VD. Acute postinfectious glomerulonephritis in the modern era: experience with 86 adults and review of the literature. Medicine. 2008 Jan 1;87(1):21-32.*
13. *Sanjad S, Tolaymat A, Whitworth J, Levin S. Acute glomerulonephritis in children: a review of 153 cases. Southern medical journal. 1977 Oct;70(10):1202-6.*

14. *Pinto SW, Sesso R, Vasconcelos E, Watanabe YJ, Pansute AM. Follow-up of patients with epidemic poststreptococcal glomerulonephritis. American journal of kidney diseases. 2001 Aug 1;38(2):249-55.*
15. *Dixon A, Blanchette E, Kendrick J. A lack of KDIGO guidelines for adolescents and young adults with IgA nephropathy. Pediatric Nephrology. 2024 Jan;39(1):297-304.*
16. *Baier E, Kluge IA, Hakroush S, Korsten P, Tampe B. Serum uric acid associates with systemic complement C3 activation in severe ANCA-associated renal vasculitides. International Journal of Molecular Sciences. 2024 Jan 5;25(2):713.*
17. *Cho SY, Kim Y, Park S, Paik JH, Chin HJ, Park JH, Lee JP, Kim YJ, Park SH, Lee HC, Cho H. Deep learning-based quantitative analysis of glomerular morphology in IgA nephropathy whole slide images and its prognostic implications. Scientific Reports. 2025 Jul 2;15(1):23566.*
18. *Liu X, Wang C, Sun Z, Liu M, Zhou N. Predictive role of Oxford Classification for prognosis in children with IgA nephropathy: a systematic review and meta-analysis. Renal Failure. 2024 Dec 31;46(2):2411846.*
19. *Vivarelli M, Samuel S, Coppo R, Barratt J, Bonilla-Felix M, Haffner D, Gibson K, Haas M, Abdel-Hafez MA, Adragna M, Brogan P. IPNA clinical practice recommendations for the diagnosis and management of children with IgA nephropathy and IgA vasculitis nephritis. Pediatric Nephrology. 2025 Feb;40(2):533-69.*
20. *Amjad W, Hamaad Rahman S, Schiano TD, Jafri SM. Epidemiology and management of infections in liver transplant recipients. Surgical infections. 2024 May 1;25(4):272-90.*
21. *Mengstie LA, Tesfa T, Addisu S, Shewasinad S. Treatment outcome of post-streptococcal acute glomerulonephritis and its associated factors among children less than 15 years at the referral hospital of East Amhara, Ethiopia. BMC Research Notes. 2024 Oct 17;17(1):313.*