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Original Article

Varied Presentations of Post-Infectious Acute Glomerulonephritis in Children: A Descriptive Study

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Abstract

Post infectious acute glomerulonephritis (AGN) remains a major cause of acute kidney injury in children in low- and middle-income countries. While most cases follow streptococcal infections and present with oedema, hypertension, and haematuria, some manifest atypically with nephrotic syndrome or severe renal dysfunction.

The Objective were to describe the clinical, laboratory, imaging, and histopathological characteristics of post-infectious AGN in Sri Lankan children and evaluate short term outcomes, including atypical presentations.

A one-year descriptive study was conducted at two tertiary paediatric centres. Sixty-six children aged 2–16 years with AGN were enrolled. Data on clinical presentation, laboratory tests (CBC, renal/liver function, electrolytes, urinalysis, C3/C4, ASO titre), imaging, and renal biopsy (in selected cases) were collected. Supportive therapy, dialysis, and immunosuppressive use were recorded. Follow-up was done at 1, 2, 3, 6, and 12 months.

Mean age was 9.0 ± 2.9 years; 57.6% were male. Oedema and hypertension occurred in 95.5%, macroscopic haematuria in 60.6%, and oliguria in 48.5%. Low C3 was found in 66.7% and elevated ASO titres in 80.3%. Nephrotic-range proteinuria occurred in 28.8%. Dialysis was required in 13.6%. Corticosteroids and cyclophosphamide were used in 30.3% and 9.1%, respectively. All achieved full recovery within 12 months; atypical AGN was seen in 6.1%.

Post-infectious AGN in children is generally self-limiting. Typical cases recover with supportive care, while atypical forms may require selective immunosuppression. Early diagnosis and appropriate management yield excellent outcomes.

Key Words

Acute glomerulonephritis, post streptococcal, children

Introduction

Acute glomerulonephritis (AGN) is a significant cause of acute kidney injury in children worldwide, with the highest burden in low- and middle-income countries [5,7]. Although the incidence has declined in high-income regions, AGN remains an important public health concern in Asia and other resource-limited settings [13,25]. Most cases follow infections caused by Group A β-haemolytic streptococci involving the pharynx or skin. Classical AGN typically presents with oedema, hypertension, haematuria, and mild renal impairment. However, a subset of children may present atypically, including nephrotic syndrome, persistent complement depression, or severe renal dysfunction [8,10]. Early recognition of these atypical features is crucial to prevent progression to chronic kidney disease (CKD).

This study describes the clinical, laboratory, imaging, and histopathological characteristics of post-infectious AGN in Sri Lankan children, with particular attention to atypical features and short-term outcomes.

Methods

This descriptive, hospital-based study was conducted over one year at the Lady Ridgeway Hospital for Children, Colombo, and the Teaching Hospital, Peradeniya. Ethical approval was obtained from institutional review boards, and written informed consent was obtained from parents or guardians.

Children aged 2–16 years with a clinical diagnosis of AGN were eligible. AGN was defined by the presence of at least one of the following: oedema, hypertension, haematuria, or oliguria. Children with pre-existing renal

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disease, chronic systemic disorders, or secondary causes of glomerulonephritis were excluded.

All participants underwent a standardised clinical evaluation, including anthropometry, blood pressure measurement, systemic examination, and grading of oedema.

Baseline and follow-up tests included full blood count, renal and liver function tests, urinalysis, urine protein–creatinine ratio, complement C3 and C4 levels, and antistreptolysin O (ASO) titres. Autoimmune markers (ANA, anti-dsDNA, and ANCA) were obtained in atypical cases [14,20].

All children underwent renal ultrasonography. Renal biopsy was performed when atypical features or persistent abnormalities were present. Biopsy specimens were examined using light microscopy and immunofluorescence [15,24].

All patients received supportive care, including salt and fluid restriction, antihypertensives, and diuretics. Dialysis was initiated in cases of refractory renal impairment. Corticosteroids, with or without cyclophosphamide, were used selectively for atypical AGN [10,19].

Participants were reviewed at 1, 2, 3, 6, and 12 months. Outcomes were categorised as:

Complete recovery means normal blood pressure, renal function, and urinalysis, Partial recovery is defined as persistent hypertension or proteinuria with normal renal function and Persistent abnormality defined as continued haematuria, proteinuria, or hypertension at follow-up.

Results

Sixty-six children were enrolled (mean age 9.0 ± 2.9 years; 57.6% male). Most children (84.8%) were between 6 and 12 years. Preceding infections were identified in 54 children (81.8%), including upper respiratory tract infections (42.4%) and skin infections (39.4%), while 18.2% had no identifiable preceding illness [6,13].

Oedema and hypertension were the most common clinical features, each occurring in 95.5% of children. Macroscopic haematuria occurred in 60.6%, oliguria in 48.5%, headache in 33.3%, and convulsions in 9.1%. (Figure 1)

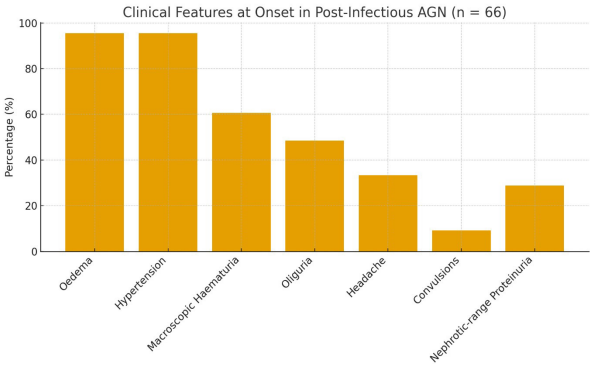

Figure 1 : Clinical features at onset

Complement C3 levels were low in 66.7% of children at presentation and normalised by six months. C4 levels were low in 7.6%. Elevated ASO titres were found in 80.3%. ANA positivity occurred in 12.1%, with no dsDNA or ANCA positivity [14,20]. Nephrotic-range proteinuria was observed in 28.8% and resolved within one month. Mean serum creatinine at presentation was 1.6 ± 0.7 mg/dL and normalised by three months.

Ultrasonography showed increased cortical echogenicity in 45.5% of children [15]. Renal biopsy was performed in 21 children (31.8%), revealing mesangial proliferation with endocapillary hypercellularity and IgG/C3 deposition in 93.3% [24,25]. (Table 1)

Table 1: Renal Biopsy findings

Findings	n	%
Mesangial matrix expansion	20	95.2
Endocapillary proliferation	17	81.0
Leukocyte infiltration	17	81.0
IgG (n=17)	14	93.3
C3(n=15)	14	93.3
Crescents	09	42.9
Basement membrane thickening	05	23.8

All children received supportive care. Dialysis was required in 13.6% but never beyond two weeks. Corticosteroids were administered in 30.3% and cyclophosphamide in 9.1%. Atypical AGN was diagnosed in 6.1%. All children achieved complete recovery at 12-month follow-up. (Table 2)

Table 2: Management options

Management Options	n	%
Prednisolone	20	30.3
IV methylprednisolone	20	30.3
IV cyclophosphamide	10	15.2
Haemodialysis	07	10.6
Mycophenolate mofetil	02	3.0
Hydroxychloroquine	02	3.0
Peritoneal dialysis	02	3.0
No special treatment needed	46	69.7

Discussion

This study evaluated the clinical presentation, laboratory features, imaging findings, and outcomes of children with acute glomerulonephritis. Most children demonstrated typical manifestations, although a small proportion (6.1%) had atypical features such as nephrotic-range proteinuria at onset. This contrasts with a large study from Nepal, where only 35.9% presented with classical AGN and 64.1% showed atypical features or complications, emphasising regional variability.

The male predominance (ratio 1.35:1) aligns with previous literature, which commonly reports ratios between 1.6:1 and 2:1. Although the mean age of 9.03 years is slightly older than the classic peak of 5–6 years, it remains within the recognised risk group.

Hypertension was extremely common, affecting 95.5% of children—higher than the 60–80% reported elsewhere. In many cases, hypertension persisted for several months, although complete resolution occurred by 12 months. Steroid therapy may have contributed to transient prolongation. Neurological symptoms, including headaches and convulsions, were likely manifestations of hypertensive encephalopathy.

Microscopic haematuria was universal at presentation, gradually improving to near resolution at 12 months. Macroscopic haematuria persisted in a minority (3%) at one month, which is atypical for AGN. Nephrotic-range proteinuria was observed more frequently than reported in most PSGN studies (~5%), although it resolved rapidly. Complement levels showed the expected pattern of depressed C3 with normalisation within 6–8 weeks.

Ultrasonographic abnormalities, predominantly increased cortical echogenicity, were noted in almost half the cohort. Prior studies suggest that such findings may correlate with more severe presentations or prolonged disease courses. Renal biopsy findings were typical of post-infectious glomerulonephritis, showing mesangial hypercellularity, endocapillary proliferation, and IgG/C3 deposition.

Most children required only supportive therapy, with immunosuppressive treatment reserved for atypical or severe cases. The overall prognosis was excellent, with complete recovery in all children by 12 months. Although poor prognostic indicators such as nephrotic-range proteinuria, persistent hypertension, elevated creatinine, and crescentic lesions were observed, these resolved with appropriate treatment.

Study limitations include a modest sample size, hospital-based recruitment, and limited follow-up duration. Larger multicentre studies with extended follow-up (24–36 months) are needed to better characterise long-term renal outcomes [3,23].

Conclusion

Post-infectious acute glomerulonephritis in children is generally benign and self-limiting. Supportive therapy remains the cornerstone of management, while early immunosuppression may benefit children with atypical or severe presentations. Prompt recognition and regular follow-up are essential to ensure full recovery and prevent long-term complications.

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