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Original Research Article

Clinical Profile and Short-Term Outcome of Acute Glomerulonephritis in Children: A Prospective Observational Study from South India

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Abstract

Background: Acute glomerulonephritis (AGN) is a common cause of acute nephritic syndrome and acute kidney injury in children. Although most cases are self-limiting and post-infectious, a minority have a non-infectious or atypical cause and a more complicated course, and distinguishing between the two at presentation remains a practical clinical challenge.

Objective: To describe the clinical, laboratory, and short-term outcome profile of children admitted with AGN at a tertiary-care centre in South India.

Methods: This prospective observational study enrolled 60 children under 18 years with a clinical diagnosis of AGN, admitted to the paediatric ward of Cheluvamba Hospital, Mysuru, over two years. Clinical features, laboratory parameters, complications, treatment, and outcome at 1 and 3 months were recorded on a structured proforma.

Results: Most children (35/60, 58.3%) were 11–15 years old, and 44 (73.3%) were male (male:female 2.7:1). Decreased urine output (53/60, 88.3%), oedema (40/60, 66.7%), and cola-coloured urine (32/60, 53.3%) were the commonest presenting complaints, and 59/60 (98.3%) were hypertensive at presentation. Nephritic-range proteinuria was seen in 42/60 (70%) and nephrotic-range proteinuria in 17/60 (28.3%). Antistreptolysin O (ASLO) was positive in 43/60 (71.7%) and serum C3 was low in 55/60 (91.7%). Thirty-six children (60%) developed at least one complication, most commonly acute kidney injury (18/60, 30%) and acute hypertensive encephalopathy (15/60, 25%); 19/60 (31.7%) required paediatric intensive care. By 3 months, 33/60 (55%) had achieved complete clinical and laboratory recovery.

Conclusion: Most children with AGN in this cohort had a typical, post-infectious presentation and a favourable early course, but a substantial minority developed acute kidney injury or hypertensive encephalopathy requiring intensive care, and fewer than two-thirds had fully recovered by 3 months. Systematic follow-up to at least 3 months, with attention to persistent proteinuria, hypertension, or hypocomplementaemia, is warranted to identify children who need further evaluation for a non-infectious or atypical cause.

Keywords: acute glomerulonephritis; children; nephritic syndrome; outcome; follow-up.

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Introduction

Acute glomerulonephritis (AGN) is defined by the sudden onset of the nephritic syndrome — haematuria, proteinuria, oliguria, hypertension, and oedema, with or without renal impairment — and remains one of the more common causes of acute kidney injury in school-aged children.[1,2]

Post-streptococcal, and more broadly post-infectious, glomerulonephritis (PIGN) accounts for the large majority of paediatric AGN and typically follows a self-limited course, with diuresis beginning within one to two weeks and serum creatinine normalising within about a month in most children.[2,3] A minority of children, however, have a non-infectious or atypical cause — systemic lupus erythematosus (SLE), Henoch–Schönlein purpura (HSP) nephritis, membranopr oliferative glomerulonephritis (MPGN), IgA nephropathy, or C3 glomerulopathy among them — in whom the clinical course, treatment, and long-term renal prognosis differ substantially from typical PIGN, and for whom delayed recognition can have lasting consequences.[3,4]

Because the initial clinical and laboratory presentation of AGN often cannot reliably

distinguish typical PIGN from these atypical causes, structured follow-up — particularly of persistent proteinuria, hypertension, or hypocomplementaemia — and, where indicated, renal biopsy, are central to identifying children who need a different treatment approach.[4] We undertook this prospective observational study to characterise the clinical presentation, laboratory findings, complications, and short-term (1- and 3-month) outcome of children admitted with AGN at a tertiary-care teaching hospital in South India, to describe both the typical course and the features that most often prompted further evaluation.

Methods

Study design and participants: This prospective observational study was conducted in the Department of Paediatrics, Mysore Medical College and Research Institute (Cheluvamba Hospital), Mysuru, over a two-year period, after institutional ethics committee approval and written informed consent from parents or legal guardians. All children under 18 years of age admitted with a clinical diagnosis of AGN, with or without systemic symptoms, were enrolled by purposive sampling; children with pre-existing renal disease presenting with a manifestation other than AGN were excluded.

Definitions and procedures: A structured proforma was used to record demographic details, history of preceding infection, clinical features, and laboratory and, where performed, histopathological findings. Hypertension was defined as systolic and/or diastolic blood pressure at or above the 95th centile for age, sex, and height, confirmed on three occasions. Haematuria was defined as more than 5 red blood cells per high-power field in a freshly voided, centrifuged mid-stream urine sample. Oliguria was defined as urine output below 1 mL/kg/hour. Proteinuria was quantified using standard urinary protein indices. Recent streptococcal infection was inferred from a positive ASLO titre. Renal biopsy was performed where clinically indicated (e.g., atypical features, persistent hypocomplementaemia, or a nephrotic-range or mixed nephritic–nephrotic picture), as detailed in a companion analysis of the biopsied subgroup.[5] All children were followed up at 1 and 3 months for renal function, blood pressure, urinalysis, and serum C3.

Statistical Analysis: Data were analysed using SPSS version 25. Continuous variables are presented as mean ± standard deviation; categorical variables as number (%). Group comparisons used the chi-square test for proportions and the independent-samples t-test for continuous variables, with $p<0.05$ considered statistically significant.

Results

Sixty children with AGN were enrolled. Seventeen (28.3%) were under 10 years, 35 (58.3%) were 11–15 years, and 8 (13.3%) were over 15 years; forty-four (73.3%) were male and 16 (26.7%) female (male: female 2.7:1). A history of preceding infection was elicited in 22 (36.7%), most often skin infection, while 33 (55%) had no such history.

The commonest presenting complaints were decreased urine output (53/60, 88.3%), oedema (40/60, 66.7%), and cola-coloured urine (32/60, 53.3%). Fifty-nine children (98.3%) were hypertensive at presentation, 42 (70%) with stage 1 and 10 (16.7%) with stage 2 hypertension. On urinalysis, 42/60 (70%) had nephritic-range and 17/60 (28.3%) had nephrotic-range proteinuria; gross haematuria was seen in 8/60 (13.3%) and microscopic haematuria in 24/60 (40%).

Table 1: Laboratory findings at presentation (n=60)

Parameter	n (%)
Elevated serum creatinine	18 (30.0)
Elevated cholesterol	16 (26.7)
Hyperkalaemia	13 (21.7)
Hyponatraemia	13 (21.7)
ASLO positive	43 (71.7)
Low serum C3	55 (91.7)
ANA/dsDNA positive	2 (3.3)
Altered renal echotexture on USG	14 (23.3)

Table 2: Complications, treatment, and outcome

Variable	n (%)
Any complication	36 (60.0)
Acute kidney injury	18 (30.0)
Acute hypertensive encephalopathy	15 (25.0)
Acute pulmonary oedema	3 (5.0)
Required antihypertensives	58 (96.6)
Required immunosuppressants	7 (11.6)
Required PICU admission	19 (31.7)
Complete recovery at 3 months	33 (55.0)

Eleven children (18.3%) required renal biopsy for atypical features, low or persistently low complement, or a mixed nephritic–nephrotic picture; findings in this subgroup, and their relationship to final aetiology and outcome, are presented in a companion article.[5] Post-infectious glomerulonephritis (PIGN, diagnosed clinically or, where biopsied, histologically) accounted for the large majority of cases (50/60, 83.3%); the remaining cases comprised lupus nephritis, HSP nephritis, MPGN, IgA nephropathy, and C3 glomerulonephritis, detailed alongside the biopsy findings in our companion analysis.

At 1 month, oedema persisted in 12/60 (20%), microscopic haematuria in 19/60 (31.7%), hypertension in 16/60 (26.7%), deranged renal function in 7/60 (11.7%), and proteinuria in 10/60 (16.7%). At 3 months, 33/60 (55%) had achieved complete clinical and laboratory recovery; among children with PIGN specifically, 31/50 (62%) had recovered completely by 3 months, a figure discussed further, alongside recovery in the non-PIGN subgroup, in our companion biopsy-focused analysis.[5]

Discussion

In this cohort, AGN most often presented in a pattern consistent with typical post-infectious disease — school-age children, a preceding skin or throat infection, oedema and oliguria, and low complement — and the large majority of cases were attributed to PIGN, broadly consistent with other Indian series.[6–9]

The proportion of children with hypertension at presentation (98.3%) was towards the higher end of the range reported in comparable studies, which have generally reported hypertension in 60–90% of children with AGN; this may reflect differences in blood-pressure definitions and measurement protocols across studies, or genuine differences in case mix, and underscores the value of using a standardised, centile-based hypertension definition, as was done here, when comparing across cohorts.[6,7]

A clinically important minority of children, however, had a more complicated course: 60% developed at least one complication, most commonly acute kidney injury or hypertensive encephalopathy, and nearly a third required intensive care. This complication profile is broadly comparable to other Indian cohorts, in which acute kidney injury and hypertensive encephalopathy have similarly been the leading complications of paediatric AGN, though the exact proportions have varied across studies, likely reflecting differences in case ascertainment, referral patterns, and the threshold used to define acute kidney injury.[7,8] By 3 months, only just over half of children in our cohort had achieved complete recovery, a reminder that even predominantly post-infectious AGN cohorts can have a substantial minority of children with ongoing, if often ultimately self-limited, abnormalities well beyond the acute presentation, and that structured follow-up, rather than a single hospital discharge assessment, is needed to confirm resolution. This study has several limitations. It was conducted at a single tertiary-care centre, which may over-represent more severe or referred cases relative to community-based AGN. Follow-up in the data available for this analysis extended to 3 months; longer follow-up, to 6–12 months or beyond, would better capture the minority of children who progress to chronic kidney disease, particularly among the non-PIGN aetiologies. Finally, aetiological classification for the majority of children was based on clinical and serological criteria rather than histopathology, since biopsy was reserved for children with atypical features; this is standard practice, and consistent with international guidance that most typical PIGN does not require biopsy confirmation, but it means that a small number of children with atypical but subtle presentations could, in principle, have been misclassified as typical PIGN.[3,9]

Conclusion

Children with AGN in this South Indian cohort most often had a typical post-infectious presentation, but a substantial proportion developed acute kidney injury or hypertensive encephalopathy, and just over half had fully recovered by 3 months. These findings support close early monitoring for complications in all children admitted with AGN, and structured follow-up to at least 3 months to confirm resolution and to identify children — particularly those with persistent hypocomplementaemia, atypical features, or a mixed nephritic–nephrotic picture — who warrant further evaluation, including renal biopsy, for a non-infectious cause.

Declarations: Ethics approval: Institutional Ethics Committee, Mysore Medical College and Research Institute. Informed consent was obtained from parents/legal guardians of all participants.

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