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Original Article

Clinical course and prognosis of acute post-streptococcal glomerulonephritis in children: A single-center experience

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ABSTRACT

Objective: This study aimed to evaluate the impact of clinical and laboratory findings at presentation on renal function and their predictive value for long-term complications in children diagnosed with acute post-streptococcal glomerulonephritis (APSGN).

Materials and Methods: Retrospective data of 37 APSGN patients aged 2–18 years, who were followed at the Konya City Hospital Pediatric Nephrology Clinic between January 2023 and January 2026, were analyzed. Clinical symptoms, laboratory parameters, ultrasonography findings, and treatment details were recorded.

Results: Macroscopic hematuria was present in 73% of the patients and edema in 27%; approximately 70% of cases presented during the autumn and winter seasons. Patients who developed acute kidney injury (AKI) had significantly higher levels of C-reactive protein (CRP), anti-streptolysin O (ASO) and uric acid (p=0.004, p=0.025 and p=0.035, respectively). Increased renal parenchymal echogenicity on urinary system ultrasonography was observed in all patients with AKI (p=0.012). Hemoglobin levels were significantly lower in patients with nephrotic-range proteinuria (p=0.015). A total of 70.3% of the patients required hospitalization with a mean length of stay of 7.5±3.0 days. During follow-up renal function returned to normal in all patients and proteinuria regressed to normal levels in the majority of patients.

Conclusion: Elevated inflammatory markers (CRP, ASO), high uric acid levels and increased renal parenchymal echogenicity are valuable early indicators of AKI in APSGN. Careful assessment of these non-invasive parameters at presentation is crucial for the early identification of high-risk patients and for guiding the management of potential complications.

Keywords: Acute kidney injury, complement C3, hematuria, glomerulonephritis, proteinuria

Introduction

Acute post-infectious glomerulonephritis is a disease resulting from an immunological response of the kidney following an extrarenal infection. The most common clinical presentation is acute post-streptococcal glomerulonephritis (APSGN) primarily triggered by Group A β -hemolytic streptococci (GAS) (1). As the predominant form of infection-related glomerulonephritis APSGN remains a major cause of pediatric hospital admissions particularly in developing regions. It develops as an immune-mediated response following infection caused by nephritogenic GAS strains (2). The median incidence of APSGN in children is estimated to be 24.3 per 100.000 per year in developing countries, whereas it is 6.2 per 100.000 per year in developed countries (3).

In APSGN, granular complement 3 (C3) deposition is observed along the glomerular capillary loops and mesangium with or without IgG (4-5). The classic clinical triad includes macroscopic hematuria, hypertension and edema (3-5). Oliguria and varying degrees of proteinuria are also common (6). Although the vast majority of cases follow a mild course severe complications may develop in a subset of patients. According to literature data, clinically challenging conditions such as rapidly progressive glomerulonephritis (RPGN), severe acute kidney injury (AKI), hypertensive crisis, pulmonary edema and nephrotic-range proteinuria have also been reported (3,5,6,7). Anemia is one of the most common laboratory abnormalities in APSGN. The primary mechanism is hemodilution resulting from intravascular volume expansion secondary to sodium and water retention. In addition, a transient reduction in erythropoietin production due to acute kidney dysfunction may also contribute to the development of anemia (8). In APSGN which generally follows a benign clinical course with a favorable prognosis, the presence of concomitant nephrotic syndrome the detection of crescent formation on renal biopsy and the presence of renal failure at admission are considered indicators of poor prognosis (9).

Although APSGN generally has a favorable prognosis in children, some patients may develop AKI and therefore require closer monitoring. Identifying early clinical, laboratory, and ultrasonographic parameters that can predict high-risk patients may contribute to timely follow-up and appropriate therapeutic strategies. Therefore, this study aimed to evaluate the impact of clinical, laboratory, and ultrasonographic findings on kidney function in children with APSGN, to determine the prognostic significance of early-phase parameters, and to identify practical markers that may contribute to early risk stratification in pediatric APSGN.

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Materials and Methods

In this retrospective study, medical records of patients aged 2–18 years diagnosed with APSGN and followed at the Konya City Hospital Pediatric Nephrology Clinic between January 2023 and January 2026 were reviewed. The diagnosis of APSGN was established based on a combination of clinical features, including macroscopic hematuria, edema, hypertension, and oliguria, and laboratory findings comprising microscopic hematuria, proteinuria, reduced C3 levels, and elevated ASO titers. Patients with known chronic kidney disease, structural urinary tract anomalies or secondary glomerulonephritis were not included. Those with hypocomplementemia lasting beyond 12 weeks, incomplete records or inconsistent follow-up were likewise excluded.

Demographic variables including age at diagnosis, sex, weight, height, and season of onset were documented for each patient. Admission laboratory data encompassed blood urea nitrogen (BUN), creatinine, total protein, albumin, uric acid, electrolytes, complete blood count, CRP, ASO, C3 and complement 4 (C4) along with spot urinary protein measurements and radiological findings from renal ultrasonography and chest X-rays. Renal biopsy was carried out when clinically indicated, including cases with normal complement at presentation, persistent macroscopic hematuria, nephrotic-range proteinuria, features suggestive of systemic disease, rapidly progressive renal failure or hypocomplementemia exceeding 12 weeks (10).

Physical examination findings blood pressure, edema and pleural effusion were systematically recorded. Treatment protocols and length of hospital stay were retrieved from patient records. Throughout follow-up, the interval to C3 normalization was documented and final renal outcomes including BUN, creatinine and urinary hematuria or proteinuria were retrospectively evaluated.

Hematuria was defined as more than 5 red blood cells per high-power field or ≥2+ on dipstick urinalysis. Proteinuria was considered clinically significant when the spot urine protein to creatinine ratio exceeded 0.2 mg/mg and nephrotic range when it surpassed 2 mg/mg (10). The estimated Glomerular Filtration Rate (eGFR) was calculated for all patients using the updated Schwartz formula with a k-value of 0.413 (eGFR = 0.413 × height/serum creatinine) (11). Acute kidney injury was classified according to the KDIGO criteria. Stage 1 AKI was defined as an increase in serum creatinine to 1.5–1.9 times the baseline, Stage 2 as 2.0–2.9 times the baseline and Stage 3 as times the baseline or the requirement for renal replacement therapy (12). Since recent pre-admission serum creatinine values were not available for all patients, baseline creatinine was estimated as the mean normative reference value corresponding to each patient's specific age and sex. Blood pressure was obtained manually with an appropriately sized cuff via auscultation. Hypertension was defined and classified per the 2017 Clinical Practice Guideline for Children and Adolescents with readings exceeding the 95th percentile for age, height and gender considered elevated (13).

Statistical analyses:

Statistical analyses were performed with IBM Statistical Package for the Social Sciences, version 25.0 (SPSS Inc., Armonk, NY, IBM Corp., USA). Variable distribution was assessed through both visual approaches (histograms, probability plots) and analytical tests (Kolmogorov-Smirnov, Shapiro-Wilk). Normally distributed continuous data are presented as mean and standart deviation with minimum and maximum ranges; non-normally distributed variables are given as median with interquartile ranges. Categorical data are reported as frequencies and percentages. Group comparisons employed the Student's t-test for parametric data, while the Mann-Whitney U test was applied to non-parametric variables. Chi-square or Fisher's exact test was used for categorical comparisons, including sex-based clinical outcomes and AKI prevalence. Statistical significance was set at $p<0.050$.

Results

The clinical and laboratory findings of the 37 patients included in the study are presented in Table I-II. The male-to-female ratio was 1.3, with a mean age of 9.28±3.35 years (range: 4.6–17.5). Hospital admissions were most frequent during autumn (n=12, 32.4%) and winter (n=14, 37.8%). Macroscopic hematuria was the predominant presenting symptom (n=27, 73%), followed by edema (n=10, 27%), abdominal pain (n=3, 8.1%), headache (n=3, 8.1%), and rash (n=1, 2.7%).

Anthropometric assessments at admission revealed a mean weight SDS of –0.22±0.87 (range: –2.19 to 1.53), height SDS of –0.24±0.83 (–1.62 to 1.77), and BMI SDS of –0.14±0.92 (–2.18 to 1.87). Underweight and overweight status were each identified in two patients (5.4%). Mean systolic and diastolic blood pressure SDS values were 0.55±0.86 and 0.72±0.76, respectively; hypertension was noted in 17 patients (45.9%). Periorbital edema alone was present in 11 patients (29.7%), whereas combined periorbital and pretibial edema was seen in 12 (32.4%). Pulmonary edema or pleural effusion was documented in 7 patients (18.9%).

Admission laboratory findings included a mean white blood cell count of 10.441±3.230/mm³, hemoglobin of 11.6±1.5 g/dL, and platelet count of 299.040±68.619/mm³. Median serum creatinine was 1.04 mg/dL (IQR: 0.57–1.53). Based on KDIGO criteria, AKI was identified in 15 patients (40.5%): stage I in 9 (24.3%), stage II in 3 (8.1%), and stage III in 3 (8.1%). Mean albumin was 3.5±0.5 g/dL, with hypoalbuminemia present in 16 patients (43.2%). Median CRP was 21.3 mg/L (IQR: 2.2–29.7), elevated in 15 patients (40.5%). Median ASO was 817 IU/mL (IQR: 488.5–1318.5), with elevation documented in 35 patients (94.6%). Mean C3 was

Table I: Demographic and clinical characteristics of patients

Variables	Values
Age (years)*	9.28±3.35 (4.6-17.5)
Gender†	
Female	16 (43.2)
Season at initial admission†	
Spring	5 (13.5)
Summer	6 (16.2)
Fall	12 (32.4)
Winter	14 (37.8)
Complaints†	
Macroscopic Hematuria	27 (73)
Body swelling (edema)	10 (27)
Abdominal Pain	3 (8.1)
Headache	3 (8.1)
Rash	1 (2.7)
Anthropometric measurements	
Height SDS*	-0.24±0.83 (-1.62-1.77)
Weight SDS*	-0.22±0.87 (-2.19-1.53)
BMI SDS*	-0.14±0.92 (-2.18-1.87)
Underweight†	2 (5.4)
Normal†	33 (89.2)
Overweight†	2 (5.4)
Office BP Measurements	
Systolic BP (mmHg)*	0.55±0.86 (-1.75-2.33)
Diastolic BP (mmHg)*	0.72±0.76 (-0.67-2.33)
Hypertension†	17 (45.9)
Physical examination†	
Periorbital edema	23 (62.1)
Pretibial edema	12 (32.4)
Pulmonary edema and/or Pleural Effusion	7 (18.9)

**: mean±SD (min-max), †: n(%)*

Table II: Clinical and laboratory characteristics of patients

Variables	Values	Reference Range
Laboratory Evaluations		
Hb (g/dL)*	11.6±1.5 (8.0-14.0)	13-17.1
WBC (/mm³)*	10.441±3.230 (6.100-17.990)	4000-10000
Platelets (/mm³)*	299.040±68.619 (191.000–441.000)	150000-450000
BUN (mg/dL)‡	23 (17-47)	7-20
eGFR (mL/min/1.73 m²)‡	58.4 (36.6-100.0)	90-120
Creatinine (mg/dL)‡	1.04 (0.57-1.53)	0.26-0.77
AKI Stage I†	9 (24.3)	-
AKI Stage II†	3 (8.1)	-
AKI Stage III†	3 (8.1)	-
Uric acid (mg/dL)*	7.3±1.8 (4.7-11.0)	3.5-7.2
Hyperuricemia†	7 (18.9)	-
Protein (g/dL)*	6.1±0.9 (4.4-7.8)	6.2-8.2
Albumin (g/dL)*	3.5±0.5 (2.4-4.7)	3.5-5.2
Hypoalbuminemia†	16 (43.2)	-
Ca (mg/dL)*	8.6±0.5 (7.8-9.4)	8.4-10.2
Mg (mg/dL)*	2.27±0.28 (1.67-2.76)	1.2-2.5
P (mg/dL)*	5.3±1.0 (4.0-7.7)	2.3-4.5
Na (mmol/L)*	136.4±2.9 (131-140)	136-146
K (mmol/L)*	4.8±0.5 (4.1-5.8)	3.5-5.1
Cl (mmol/L)*	104.5±3.9 (97-111)	96-106
CRP (mg/L)‡	21.3 (2.2-29.7)	< 5
High†	15 (40.5)	-
ASO (IU/mL)‡	817 (488.5-1318.5)	< 200
High†	35 (94.6)	-
C3 (g/L)*	0.33±0.27 (0.01-1.01)	0.9-1.8
Low†	35 (94.6)	-
C4 (g/L)*	0.18±0.06 (0.05-0.28)	0.1-0.4
Low†	2 (5.4)	-
Urine Erythrocyte (/HPF)‡	226 (71-845)	< 5
Spot Urine Protein/creatinine (mg/mg)‡	1.30 (0.45-2.6)	< 0.2
Non-nephrotic Range Proteinuria†	20 (54.1)	-
Nephrotic Range Proteinuria†	13 (35.1)	-
Urinary ultrasound†		
Increased echogenicity	5 (13.5)	-
Treatment period		
Hospitalization†	26 (70.3)	-
Length of hospital stay (day)*	7.5±3.0 (3-36)	-
Drugs†		
Furosemide	21 (56.7)	-
Amlodipine	8 (21.6)	-
Enalapril	3 (8.1)	-
Immunosuppression	1 (2.7)	-
Plasmapheresis	1 (2.7)	
Last Control		
Follow-up duration (month) ‡	12 (3.25-18)	-
Serum Creatinine (mg/dL)‡	0.44 (0.41-0.62)	0.26-0.77
Serum C3 (g/dL)‡	1.15 (1.03-1.23)	0.9-1.8
Urine Erythrocyte (/HPF)‡	0 (0-6)	< 5
Spot Urine Protein/creatinine (mg/mg)*	0.12±0.05 (0.05-0.30)	< 0.2

: mean±SD (min-max), †: n(%), ‡: median (IQR)

0.33±0.27 g/L, reduced in 35 patients (94.6%). The median spot urine protein-to-creatinine ratio was 1.30 (IQR: 0.45–2.6); non-nephrotic and nephrotic-range proteinuria were present in 20 (54.1%) and 13 (35.1%) patients, respectively. Increased renal parenchymal echogenicity on ultrasonography was found in 5 patients (13.5%).

Regarding management, 11 patients (29.7%) were monitored as outpatients, while 26 (70.3%) required hospitalization, with a mean stay of 7.5±3.0 days. Fluid and nutritional regulation with bed rest alone was sufficient for 15 patients (40.5%). Furosemide was administered to 21 patients (56.7%), amlodipine to 8 (21.6%), and enalapril to 3 (8.1%).

One patient developed nephritis following streptococcal infection, presenting with purpuric rash, edema, hypertension, nephrotic-range proteinuria, elevated ASO, and low C3. Biopsy confirmed diffuse proliferative glomerulonephritis consistent with APSGN. Renal function deteriorated rapidly, necessitating 18 days of hemodialysis and five plasmapheresis sessions. Treatment included steroids, cyclophosphamide, and azathioprine. No other patient received immunosuppressive therapy.

Median follow-up was 12 months (IQR: 3.25–18). At follow-up completion, median creatinine normalized to 0.44 mg/dL (IQR: 0.41–0.62) across all patients, and hypoalbuminemia resolved entirely.

Table III: A Comparison of clinical and laboratory characteristics of patients with acute kidney injury

Variables	Acute kidney injury	Non-acute kidney injury	p
Number of patients	15	22	-
Age (years) *	10.3±3.6 (5.3-16.6)	8.5±3.0 (4.6-17.5)	0.112
Gender (Female)†	7 (43.8)	9 (56.3)	0.495
Office BP Measurements			
Systolic BP (mmHg)*	0.63±0.92 (-0.81-2.33)	0.50±0.84 (-1.75-1.88)	0.659
Diastolic BP (mmHg)*	0.70±0.74 (0.0-2.33)	0.73±0.80 (-0.67-2.05)	0.905
Hypertension†	4 (26.7)	3 (13.6)	0.283
Laboratory Evaluations			
Hb (g/dL)*	11.7±1.5 (8.8-14)	11.6±1.5 (8-13.7)	0.819
WBC (/mm^3)*	11.606±3.382 (6,530-17.990)	9275±2715 (6100-15510)	0.055
Platelets (/mm^3)*	281.930±60.202 (191.000-389.000)	316140±74329 (217000-441000)	0.192
Uric acid (mg/dL)*	7.2±1.8 (4.6-11)	5.7±1.2 (3.7-7.4)	0.035
Hyperuricemia†	5 (33.3)	2 (9.1)	
Albumin (g/dL)*	3.3±0.4 (2.8-4.2)	3.6±0.5 (2.4-4.7)	0.059
Hypoalbuminemia†	8 (53.3)	8 (36.4)	0.247
CRP (mg/L)‡	25 (19-41)	3 (1.4-5)	0.004
High†	13 (86.7)	2 (9.1)	<0.001
ASO (IU/mL)‡	967 (554-1614)	488 (432-817)	0.025
C3 (g/L)‡	0.21 (0.16-0.47)	0.23 (0.12-0.55)	0.991
Urine Erythrocyte (/HPF)‡	535 (74-1312)	181 (34-429)	0.089
Urine Protein/creatinine (mg/mg)‡	1.10 (0.70-2.30)	1.45 (0.35-3.10)	0.904
Urinary Ultrasound			
Increased echogenicity†	5 (33.3)	0 (0)	0.012
Treatment Period			
Hospitalization†	13 (86.7)	13 (59.1)	0.073
Length of hospital stay (day)*	7.1±3.9 (3-16)	6±1.9 (4-9)	0.349

**: mean±SD (min-max) (Independent Samples t-test), †: n(%) (Fisher's exact test), ‡: median (IQR) (Mann-Whitney U)*

Median urinary erythrocyte count was 0 (IQR: 0–6), with microscopic hematuria persisting in 6 patients (16.2%). Mean spot urine protein-to-creatinine ratio declined to 0.12±0.05, with residual non-nephrotic proteinuria in only 3 patients (8.1%). C3 levels normalized in all patients within a mean of 6.2±2.6 weeks.

Factors related to renal involvement severity are detailed in Table III. Hyperuricemia was found in 33.3% of AKI patients (7.2±1.8 mg/dL) versus 9.1% of non-AKI patients (5.7±1.2 mg/dL), with a statistically significant between-group difference (p=0.035). Elevated CRP at admission was present in 86.7% of AKI patients (p<0.001), and median CRP was markedly higher in the AKI group (25 (19-41) vs. 3 (1.4-5) mg/L, p=0.004). Median ASO was similarly elevated in AKI patients compared to those unaffected (967 (554-1614) vs. 488 (432-817), IU/mL, p=0.025). All five patients with increased renal echogenicity on ultrasonography developed AKI (p=0.012). No other clinical or laboratory parameters showed significant association with AKI. Among the patients who developed AKI, nine were classified as stage 1, three as stage 2, and three as stage 3. Due to the limited number of patients, a comparative analysis of stage 3 patients with the other groups could not be performed, despite their high clinical significance. Hemoglobin was lower in patients with nephrotic-range proteinuria than in those without (10.7±1.0 vs. 12.1±1.5 g/dL, p=0.015). Macroscopic hematuria was more prevalent among male patients (76.2%) than female patients (43.8%; p=0.047). No further significant associations were identified between the remaining variables and proteinuria or hematuria.

Discussion

Although childhood APSGN is generally considered to have a favorable prognosis, the 40.5% AKI rate observed in our study demonstrates that the clinical presentation is not always mild and that the disease carries a significant risk of renal injury. In our study, elevated CRP and ASO levels, high serum uric acid levels, and increased renal parenchymal echogenicity were identified as non-invasive predictors showing a statistically significant association with the development of AKI. Comprehensive assessment of these parameters at admission will facilitate the early identification of high-risk patients, effective management of complications, and timely planning of further diagnostic evaluation and treatment options when necessary.

Epidemiological data in the literature indicate that APSGN occurs approximately twice as frequently in boys as in girls (2,14). The male predominance observed in this study (M/F: 1.3) is consistent with the literature. Furthermore, the mean age of 9.28 years confirms that APSGN typically peaks among school-aged children between 5 and 12 years (14).

In the study by Tizki et al. (15), APSGN was more frequent in winter (41%) and autumn (33.7%), with the most common clinical findings being hematuria (90.3%), edema (77.1%), hypertension (66.3%), and oliguria (12%). In our study, approximately 70% of cases occurred during autumn and winter, likely related to the seasonal increase in upper respiratory tract infections caused by nephritogenic streptococci. The most frequent finding in our patients was macroscopic hematuria (73%), while hypertension (45.9%) and edema (27%) indicative of volume overload were also common. Additionally, hematuria was significantly more frequent in boys than in girls (p=0.047), which may reflect gender-related differences in clinical presentation.

In our study, 70.3% of the patients required inpatient treatment, and the mean hospital length of stay was 7.5 days. This finding clearly demonstrates that APSGN in its acute phase requires close clinical and laboratory monitoring due to the risk of severe complications such as hypertension, pulmonary edema, AKI, and electrolyte imbalances. The high rate of AKI and the frequency of nephrotic-range proteinuria observed in our study are considered the main determinants of hospital stay exceeding one week. In these patients, daily monitoring of renal function, close assessment of fluid balance, and strict blood pressure control constitute essential components of management.

This study identified a relationship between systemic inflammatory markers and the development of AKI, suggesting that the interaction between streptococcal virulence and the host immune response may determine the extent of kidney injury. In a study conducted by Wan Yusof et al. (16), the prevalence of AKI was reported as 21.7%. In our study, the prevalence of AKI was found to be 40.5%, which exceeds the rates reported in the literature. As our center is a tertiary care facility, patients with more severe clinical presentations are more likely to be referred, which may account for the higher rate of AKI observed in our study. Analysis of inflammatory markers between groups revealed significantly higher CRP (p=0.004) and ASO (p=0.025) levels in patients who developed AKI compared to those with preserved renal function. This finding suggests a potential relationship between the severity of systemic inflammation and the extent of renal parenchymal injury. Elevated ASO titers indicate a higher streptococcal antigen load, while the concomitant high CRP levels reflect the intensity of the acute-phase response against these antigens. The literature reports that CRP is not only an infection marker but may also contribute to the pathogenesis of glomerular injury by triggering complement activation and promoting endothelial damage (17,18). In this context elevated CRP levels detected at the time of admission can be considered an early biomarker for predicting the development of AKI in patients with APSGN. Early detection of patients at high risk is clinically significant and supports the development of more intensive follow-up strategies.

Increased renal parenchymal echogenicity detected on urinary system ultrasonography is considered an indirect reflection of interstitial edema, inflammatory cell infiltration, tubular damage, and glomerular injury in the kidney tissue. This finding is a non-invasive and reliable ultrasonographic parameter reflecting pathological changes in the renal parenchyma, and it is well established that echogenicity may increase in both acute and chronic kidney diseases. In particular, in immune-mediated inflammatory processes such as glomerulonephritis, cellular proliferation and inflammatory infiltration occurring at the glomerular and interstitial levels increase renal echogenicity, rendering it detectable on ultrasonography (19). The development of AKI in all patients with increased echogenicity highlights the importance of ultrasonography. In light of these findings, cases with increased echogenicity should be considered high-risk and closely monitored.

Shrestha et al. (20) reported non-nephrotic proteinuria in 28.57% and nephrotic-range proteinuria in 3.17% of patients with APSGN. Chong et al. (21) reported nephrotic-range proteinuria in 57.7% of patients. In our study, non-nephrotic proteinuria was observed in 54.1% of patients, while nephrotic-range proteinuria was detected in 35.1%. Hemoglobin levels were significantly lower in patients with nephrotic-range proteinuria (p=0.015). This finding may be explained by the hypoalbuminemia accompanying nephrotic-range proteinuria, which reduces intravascular oncotic pressure, promotes fluid accumulation, and thereby predisposes to dilutional anemia. Indeed, in APSGN, hypervolemia leads to expansion of plasma volume without a true reduction in red cell mass, causing hemoglobin values to appear falsely low. The dilutional anemia developing through this mechanism tends to resolve spontaneously as hypervolemia and proteinuria subside during the clinical course of the disease (22).

In the APSGN study conducted by Atasever Yıldırım et al. (23), decreased C3 levels were observed in all patients. In a pediatric APSGN study conducted by Demircioğlu Kılıç et al. (24), decreased C3 levels were observed in 98.7% of patients and decreased C4 levels in 16% of patients, and these findings were found to be associated with disease activity. In our study, serum C3 reduction, a marker for APSGN, was detected in 94.6% of patients, confirming the predominance of alternative pathway activation in the disease's pathogenesis. During follow-up, C3 levels returned to normal within an average of 6.2 weeks, consistent with the 6–8 week recovery period reported in the literature (25). The normalization of C3 levels within this time frame confirms the diagnosis of APSGN whereas persistent hypocomplementemia should prompt consideration of chronic conditions such as C3 glomerulopathy in the differential diagnosis (26). In our study, two patients had transiently decreased C4 levels at presentation, which normalized during follow-up. Although APSGN is typically characterized by isolated C3 consumption, cases with concomitant reductions in both C3 and C4 levels have also been reported in the literature. Similarly, a pediatric study from Cape Town reported patients with concurrent low C3 and C4 levels, none of whom showed clinical or biochemical evidence suggestive of lupus nephritis (1).

In our study, serum uric acid levels were found to be significantly higher in patients who developed AKI compared to those with preserved renal function (p=0.035). This finding suggests that hyperuricemia may serve as a contributing factor to renal parenchymal injury in the setting of APSGN. Uric acid adversely affects kidney function through several mechanisms. At high levels, it forms monosodium urate crystals that accumulate in the renal interstitium and lead to tissue damage. It also induces intracellular oxidative stress, resulting in DNA damage, inflammation and cell death. In addition, it activates the renin-angiotensin-aldosterone system, contributing to hypertension, inflammation, fibrosis and endothelial dysfunction (27). In a study conducted by Mengstie et al. (28), all patients received diuretics and antihypertensive therapy was initiated in 56.7% of patients. In our study, 21 patients (56.7%) received diuretics and 11 patients (29.7%) received antihypertensive therapy. The cornerstone of treatment in APSGN is a supportive approach, with fluid restriction, diuretic use and blood pressure control constituting the indispensable components of this management. Hypervolemia-induced hypertension and edema are the most frequently encountered clinical conditions requiring intervention during the acute phase of the disease; therefore, fluid balance and blood pressure management represent the primary focus of treatment. In our study, one patient developed RPGN requiring dialysis, plasmapheresis and immunosuppressive therapy. Furthermore, RPGN is a rare but life-threatening complication of APSGN and early diagnosis along with an aggressive treatment approach are of utmost importance. In this context, renal biopsy and the evaluation of advanced treatment options are of great importance in patients with rapidly deteriorating renal function, those who fail to respond to standard therapy or those presenting with an atypical clinical picture.

Follow-up data from this study indicate that APSGN in childhood generally has a favorable prognosis. Serum creatinine normalized in all patients, and proteinuria largely resolved. However, elevated CRP and ASO levels, high serum uric acid levels and increased renal parenchymal echogenicity at presentation may serve as early indicators for the development of AKI. Therefore, the combined assessment of inflammatory markers, serum uric acid levels and radiological findings is of great importance for the early identification of high-risk cases and appropriate follow-up planning.

Limitations

Several limitations should be acknowledged. The retrospective single-center design and modest cohort of 37 patients restrict the broader applicability of these results. While the 12-month follow-up yielded meaningful insight into short and medium term disease course, large-scale prospective investigations are warranted to comprehensively assess the long-term sequelae of APSGN. Lastly, because our center is a tertiary care institution, patients with more severe clinical presentations are frequently referred to our hospital; this factor may be the primary reason for the high rate of AKI observed in our study.

Conclusion

This study demonstrated that elevated inflammatory markers (CRP and ASO), hyperuricemia, and increased renal parenchymal echogenicity on ultrasonography were associated with AKI in children with APSGN. Although APSGN generally follows a favorable course with complete recovery, the 40.5% incidence of AKI observed in our cohort indicates that a considerable proportion of patients may develop significant renal involvement. The main clinical implication of our findings is that these readily available parameters obtained during the initial evaluation may facilitate the early identification of children at increased risk of AKI. Early recognition of high-risk patients may help prevent serious complications such as severe hypertension, pulmonary edema, and progressive renal dysfunction. Therefore, incorporating these easily accessible markers into the initial assessment of pediatric APSGN may improve early risk stratification and optimize patient management.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Konya City Hospital (09/03/2026, reference number: 2026/64).

Contribution of the authors

İY: contributed to the conception and design of the study, data collection, and the drafting of the manuscript. MS: participated in the analysis and interpretation of the data and provided a substantial critical review of the intellectual content. EL: collaborated in the data collection and contributed to the critical revision of the final manuscript. All authors have approved the final version to be published and agree to be accountable for all aspects of the work

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Conflict of interest

The authors declare that there is no conflict of interest.

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