e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 1522-1525

Case Series

A Case Series of Post Streptococcal Glomerulonephritis (PSGN) in Young Adults in a Tertiary Care Teaching Institute

Pravin Soni[1], Niranjan Pathak[2], Ram Mundhe[3], Rajas Mudgerikar[4]

[1]Professor & HOD, Dept. of General Medicine, PCMC's PGI YCMH, Pimpri, Pune
[2]Asso Professor, Dept. of General Medicine, PCMC's PGI YCMH, Pimpri, Pune
[3]Asst Professor, Dept. of General Medicine, PCMC's PGI YCMH, Pimpri, Pune
[4]JR, Dept. of General Medicine, PCMC's PGI YCMH, Pimpri, Pune

Received: 01-05-2026 / Revised: 15-06-2026 / Accepted: 21-07-2026
Corresponding author: Dr. Rajas Mudgerikar
Conflict of interest: Nil

Abstract

Background: Post-streptococcal glomerulonephritis (PSGN) is an immune-mediated glomerular disease following infection with group A β-hemolytic streptococci. It commonly affects children, but clinical manifestations in young adults can vary significantly.

Objective: To study and compare the varied clinical presentations, laboratory profiles, and outcomes of PSGN in young adults admitted to a tertiary care hospital.

Methodology: A prospective case series of 4 patients aged 13–20 years admitted between July 2024 and May 2025 with a diagnosis of PSGN was conducted. All cases fulfilled clinical and laboratory criteria for PSGN. Patients above 20 years or with pre-existing renal or immunological disease were excluded.

Results: Presentations ranged from classical nephritic syndrome and hypertensive emergency to atypical isolated hematuria and AKI. All cases showed low serum C3 and elevated or borderline ASO titers. Renal recovery was complete in all, though durations varied based on severity.

Conclusion: PSGN in young adults presents with heterogeneous clinical manifestations. Timely recognition and supportive care result in favourable outcomes.

Keywords: PSGN, Western Maharashtra.

DOI: 10.25258/ijcpr.18.8.227

Introduction

Post-streptococcal glomerulonephritis (PSGN) is an immune-complex mediated glomerular disease that occurs following infection with nephritogenic strains of group A β-hemolytic streptococci. It commonly follows streptococcal pharyngitis or skin infection such as impetigo, with a latent period typically ranging from 1–3 weeks after pharyngitis and 3–6 weeks after impetigo. Although the incidence of PSGN has declined in developed countries, it continues to be an important cause of acute glomerulonephritis in resource-limited settings, particularly among children and young individuals.

The clinical presentation of PSGN is characterized by the acute nephritic syndrome, with manifestations including cola-colored or gross hematuria, periorbital or peripheral edema, hypertension, oliguria, and, in some cases, acute kidney injury. However, the clinical spectrum can be considerably broader, with some patients presenting with isolated hematuria, significant proteinuria, severe hypertension, or neurological complications related to hypertensive encephalopathy. This variability may make early recognition and diagnosis challenging, particularly when the classical nephritic presentation is absent.

Laboratory evaluation plays an important role in establishing the diagnosis. A reduction in serum complement C3, with a relatively preserved C4 level, is a characteristic finding. Evidence of recent streptococcal infection may be demonstrated by elevated anti-streptolysin O (ASO) and/or anti-DNase B titres. Urinalysis commonly reveals hematuria, red blood cell casts, and varying degrees of proteinuria. Renal biopsy is generally not required in typical cases but may demonstrate diffuse endocapillary proliferative glomerulonephritis with characteristic subepithelial "hump-like" deposits when performed.

While PSGN is classically regarded as a pediatric disease, its occurrence in adolescents and young adults can demonstrate a heterogeneous clinical

spectrum. Recognition of atypical presentations is important to avoid diagnostic delay, unnecessary investigations, and inappropriate treatment. The present case series was therefore undertaken to describe and compare the varied clinical presentations, laboratory profiles, and outcomes of PSGN among young patients admitted to a tertiary care teaching institute.

Inclusion Criteria: Patients between 13-20 yrs of age fulfilling the clinical & laboratory criteria for PSGN

Exclusion Criteria: Pre-existing renal disease/immunological disease

Case Presentation

Case 1 – Classic Nephritic Syndrome: 16 yrs old girl presented to OPD with cola colored urine since 4 days, bipedal edema since 3 days, facial puffiness since 3 days, and decreased urine output since 5 days. Bp 130/90. On UR/M RBC+ Protein absent. C3 levels on lower side 0.15. ASO 1:2 = 400 IU/L. Patient was given supportive management, low dose Lasix & patient improved on Day 3 of admission, with complete resolution of complaints including facial puffiness & cola colored urine by Day 15 after initial presentation.

Case 2 – PSGN with HTN emergency: A 15 y/o male patient brought by parents to OPD with c/o decreased urine output, pain in abdomen associated with abdominal distension since 5 days with h/o passing high colored urine. BP= 200/110, UR/M RBC +, WBC nil. No Protein, glucose. Patient started on antihypertensive agents, patient responded & showed improvement on day 4 of admission.

Case 3 - Atypical PSGN: A 19 y/o boy presented with convulsions 2 episodes since morning, BP=140/90, MRI Brain showed e/o PRES secondary to HTN.

UR/M RBC + No protein. BP controlled after starting anti HTN.

Case 4 – PSGN with Nephrotic-Range Proteinuria but Preserved Renal Function: 13-year-old female presented with facial puffiness, periorbital edema, and leg swelling for 7 days. BP 120/80 mmHg.

No gross hematuria. UR/M showed protein +++ with microscopic RBCs 10–12/HPF. 24h urine protein 3.5 g/day. Serum albumin 2.5 g/dL. Serum creatinine 0.8 mg/dL. C3 was low at 0.16 g/L. C4 normal. ASO titer 450 IU/mL. Patient received supportive care with low-dose loop diuretics. She had complete remission by day 14.

Case 5 – Normotensive PSGN with Isolated Gross Hematuria: 18-year-old male presented with cola-colored urine for 2 days. No edema, oliguria, or systemic complaints. BP 110/70 mmHg. UR/M showed RBC >100/HPF with RBC casts, mild proteinuria. 24h urine protein was 0.4 g/day. Serum creatinine 0.9 mg/dL. C3 was 0.14 g/L, C4 normal. ASO titer was 520 IU/mL. Managed as outpatient with dietary sodium restriction. Hematuria resolved by day 10; C3 normalized by 6 weeks.

Discussion

PSGN is an immune-complex mediated glomerulonephritis typically following infection with nephritogenic strains of group A β-hemolytic streptococci (GAS). Common after pharyngitis or impetigo; latency period is ~1–3 weeks post-pharyngitis and 3–6 weeks post-impetigo. Incidence has declined in developed countries but remains common in resource-limited settings, especially in children aged 5–15 years. Deposition of immune complexes (IgG, IgM, C3) in the glomerular basement membrane (GBM) and mesangium triggers complement activation via the alternative pathway. Streptococcal antigens such as nephritis-associated plasmin receptor (NAPlr) and streptococcal pyrogenic exotoxin B (SPEB) are implicated. Endocapillary proliferation, infiltration by neutrophils, and subepithelial "hump-like" deposits are classical findings.

Clinical findings: Sudden onset of Gross hematuria ("cola-colored" urine), oedema, hypertension, oliguria. Nephritic syndrome presentation, sometimes with acute kidney injury (AKI). Systemic symptoms of antecedent infection may still be present.

Laboratory findings: Low serum C3 levels (in >90% of patients), normal C4. Elevated anti-streptolysin O (ASO) and/or anti-DNase B titres support recent streptococcal infection.

Urine shows red cell casts, hematuria, and mild proteinuria. Renal biopsy (rarely needed) shows diffuse endocapillary proliferative GN with "humps" on electron microscopy (EM).

Histopathology: Light Microscopy (LM): Diffuse endocapillary proliferation with PMN infiltration.

Immunofluorescence (IF): Coarse granular deposits of IgG and C3 along the GBM and mesangium (“starry sky” appearance).

Electron Microscopy (EM): Subepithelial electron-dense "hump-like" deposits.

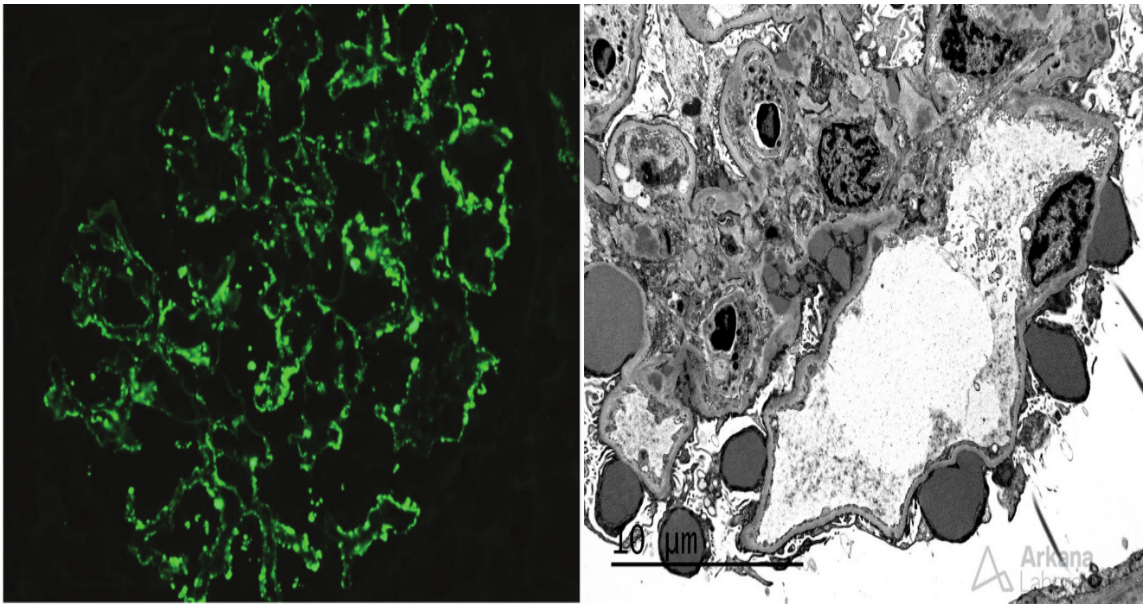
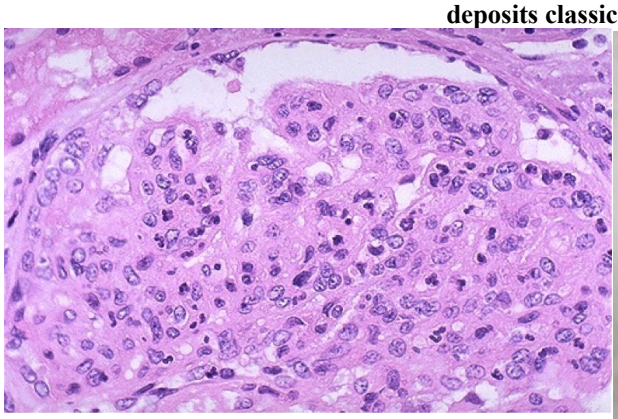


Figure 1 & 2: Immunofluorescence & electron microscopy showing starry sky appearance & hump like deposits classical of PSGN.



The hypercellularity of post-infectious glomerulonephritis is due to increased numbers of epithelial, endothelial, and mesangial cells as well as neutrophils in and around the glomerular capillary loops. (high power microscopy)

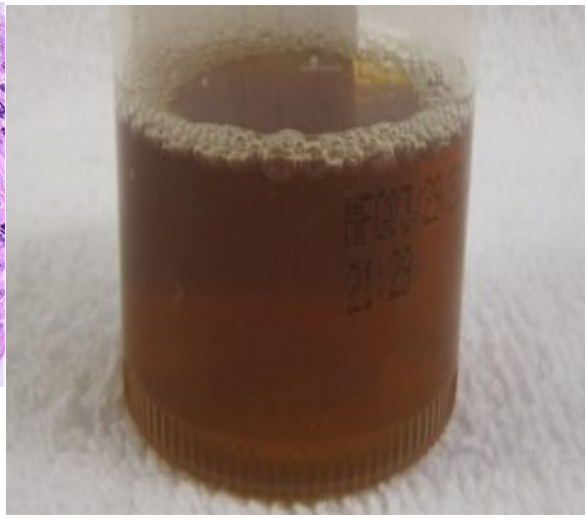


Figure 4 & 5: Histopathology image of a kidney biopsy of a child diagnosed with PSGN, classical cola colored urine seen in the same child.

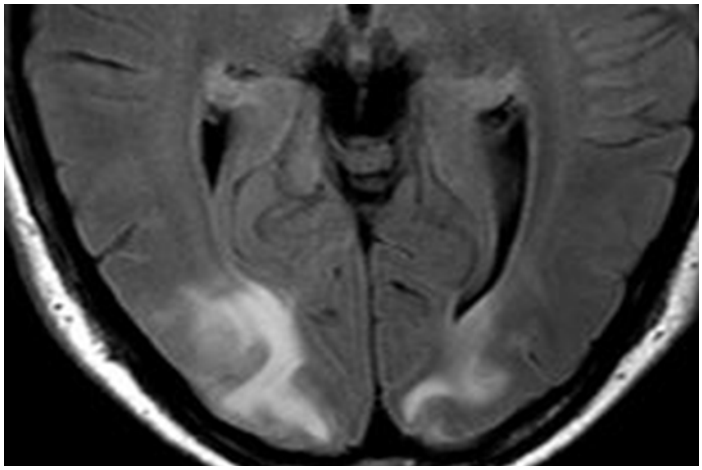


Figure 5: MRI image of a patient with PSGN presenting as PRES

Table 1: Clinical and Laboratory Characteristics of Patients with Post-Streptococcal Glomerulonephritis (PSGN)

Patient number	Age/Sex	Edema	Hematuria	Hypertension	C3 levels	Urine Routine RBCs	ASO titres
1	13/F	+	+	130/90	0.15	+	1:2=400 IU/L
2	15/M	+	+	180/100	0.12	++	1:2=400IU/L
3	27/M	-	+	200/120	0.16	+	1:3=600 IU/L
4	13/F	+	-	120/80	0.16	+	450 IU/L
5	18/M	-	+	110/70	0.14	+	520 IU/L

Conclusion

PSGN in young adults has a diverse clinical spectrum, from benign hematuria to hypertensive emergency. Despite the variation in presentations, prompt recognition and basic supportive management ensure good prognosis in most cases. Awareness of atypical features is essential to avoid misdiagnosis and overtreatment.

References

1. Poststreptococcal Glomerulonephritis, Prashanth Rawla; Sandeep A. Padala; Dipesh Ludhwani.
2. Bacterial infection–related glomerulonephritis in adults Samih H. Nasr1, Jai Radhakrishnan2 and Vivette D. D'Agati3
3. Clinical Manifestation Patterns and Trends in Poststreptococcal Glomerulonephritis, Kee Hyuck Kim, M.D. Child Kidney Dis 2016;20:6-10 DOI: http://dx.doi.org/10.3339/jkspn.2016.20.1.6
4. Harrisons Principles of Internal Medicine 21st edition, Chapter 314 page 2335-2340.