International Journal of Web of Multidisciplinary Studies
(Peer-Reviewed, Open Access, Fully Refereed International Journal)
website: *http://ijwos.com*
Volume.03 Issue.01.
OPEN ACCESS
E-ISSN : 3049-2424
DOI: https://doi.org/10.71366/ijwos0301260414578

Post-Streptococcal Glomerulonephritis Presenting as Hypertensive Urgency in a Pediatric Patient: A Case Report

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Article Info

Article History:

Published:19 Jan 2026

Publication Issue:
Volume 3, Issue 01
January-2026

Page Number:
438-443

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Abstract:

Infection with nephritogenic strains of group A β-hemolytic Streptococcus usually results in the immune-mediated kidney disease known as post-streptococcal glomerulonephritis (PSGN). Due to the possibility of end-organ damage and difficulties in diagnosis, presentation with hypertensive urgency is nevertheless clinically noteworthy even though it is a common cause of acute nephritic syndrome in children. We describe the case of a nine-year-old boy who had a fever, extensive hematuria, developing widespread edema, and noticeably high blood pressure (160/110 mmHg). Significant proteinuria, hematuria, high anti-streptolysin O titers, hypocomplementemia, and maintained renal function were found in the laboratory assessment. It was determined that the condition was post-streptococcal glomerulonephritis aggravated with hypertensive urgency. Resolution of edema, normalization of blood pressure, and improvement in urine results were all signs of a progressive clinical recovery. Immune-complex-mediated glomerular damage, which results in decreased glomerular filtration, salt and water retention, and activation of the renin-angiotensin-aldosterone system, is thought to be the cause of the hypertensive urgency seen in PSGN. To avoid problems, early detection and timely treatment with an emphasis on blood pressure control and volume regulation are essential. This case study highlights the importance of recognising hypertensive urgency as a potential consequence of PSGN in pediatric patients. Long-term renal sequelae can be avoided and positive outcomes can be achieved with prompt diagnosis and proper supportive care.

Keywords: Pediatric Patient

1. Introduction

Untreated infections with specific nephritogenic strains of the A beta-haemolytic streptococcal family cause post-streptococcal glomerulonephritis (PSGN). One of the most common causes of acute nephritis in children globally is thought to be PSGN. 97% of the 470,000 cases worldwide were discovered in third-world nations [1]. Patients with PSGN present differently clinically. They may exhibit microscopic haematuria or a full-blown nephritic condition, or they may be asymptomatic. Red or brownish urine from an excess of protein (proteinuria) is a characteristic of nephritic syndrome. Edema, hypertension, and acute kidney damage can result from proteinuria if it falls within the nephritic range. PSGN frequently has a very good prognosis, particularly in youngsters. Rarely, the long-term effects could not be favorable. Acute nephritic syndrome is brought on by PSGN, an

immune-complex-mediated inflammation of the glomerulus, a group of capillaries in the kidney's functional unit, the nephron. Following an infection of the skin (impetigo) or throat (pharyngitis) by nephritogenic strains of group A beta-haemolytic streptococci, a gram-positive bacterium that enters the body through pores in the skin or mucous epithelia, PSGN is characterized by a proliferation of cellular elements secondary to an immunologic mechanism. Every year, it results in over 500,000 deaths from various illnesses, including PSGN, rheumatic fever, rheumatic heart disease, and other invasive infections [2]. The long-term prognosis varies; generally speaking, youngsters have a great prognosis, but older people and populations with additional risk factors for chronic renal disease have a much worse prognosis [3]. Although it is the most common kind of childhood glomerulonephritis, it rarely results in chronic kidney disease; however, less than 10% of individuals may experience persistent microscopic hematuria and proteinuria [4].

2. CASE REPORT

A nine-year-old male child reported with a four-day history of intermittent, moderate-grade fever along with chills, shortly followed by the onset of progressive generalized edema and dark-colored urine. Before the sickness, the child appeared to be asymptomatic. First observed over the face, edema later spread to the trunk and lower limbs. On the day of admission, gross hematuria was noticed.

On examination, the child was conscious and oriented, with a markedly elevated blood pressure of 160/110 mmHg. Generalized edema with prominent facial puffiness was observed. CVS examination showed normal heart sounds with no distinct murmurs. Abdominal PALPATION showed a soft, non-tender abdomen with no signs of organomegaly.

Laboratory tests showed a normal serum creatinine level of 0.8 mg/dL and an increased blood urea level of 53 mg/dL. Significant hematuria and proteinuria were found in the urine. The 24-hour urine protein excretion was 2330 mg/day, and the spot urine protein was 230.6 mg/dL. Serum electrolytes were within the normal range. Serum globulin levels were increased at 3.8 g/dL, while liver function tests were normal. Complement testing revealed a C4 level of 0.163 g/L. urine dipstick tests revealed positive results for both blood and protein, and urine microscopy revealed 8–10 red blood cells per high-power field. A peripheral blood smear indicated normocytic hypochromic anemia.

The child became afebrile on day six and began to gradually improve clinically. Edema decreased, urine production improved, and blood pressure was better controlled, with readings falling to 130/70 mmHg. Over24-hours, urine output remained adequate at about 1.25 mL/kg/hour. Urine examination showed a specific gravity of 1.010 with the presence of bilirubin, blood, protein, nitrites, and leukocytes.

Amlodipine and torsemide were started to improve edema and hypertension management. Prednisolone and cefixime were administered. The child continued to pass urine that was dark in colour for a period of two days, after which the antibiotic treatment was changed to amoxicillin–clavulanate for a period of five days.

By the 12th day of the hospital stay, the edema had significantly decreased, the blood pressure was still under control, and there were no more episodes of dark urine. Erythrocyte sedimentation was found to occur at a rate of 45 mm per hour. Urine microscopy revealed epithelial cells and 6-8 red blood cells per high-power field. Benzathine penicillin was injected stat. Enalapril was started and nifedipine was gradually tapered off to optimize antihypertensive therapy. The child showed consistent clinical improvement with ongoing treatment.

For the next two days, the patient's vital signs and hemodynamic parameters remained stable, and they remained afebrile. After being moved to the ward, the child's recovery continued to be satisfactory.

Table 1 - Laboratory investigations (on the day of admission).

PARAMETERS	OBSERVED VALUE
BILI(T)[0.3-2.0]mg/dl	0.3

BILI(D)[0-0.6]mg/dl	0.1
BILI(ID)[0-0.4]mg/dl	0.2
SGOT(AST)[6-38]u/l	28
SGPT(ALT)[6-38]U/L	27
AIK.Phos[53-128]u/l	145
Globulin[2.0-3.5]g/dl	3.8
Albumin [3.5-5.5 g/dl]	3.2
Total protein	7.0

Table 2: Urine protein / creatinine ratio

PARAMETERS	**VALUES**
SPOT URINE PROTINE [<15 mg/dl]	230.6
SPOT URINE CREATININE [20-400 mg/dl]	46.2
RATIO	4.6
24 HRS URINARY PROTIENS [<140 mg/24hr urine]	2335

Table 3: ASO with Titre

PARAMETER	**VALUE**
ASO (anti streptolysin o) [<200 IU/ml]	800 IU/ml

Table 4: medication chart

TRADE NAME	**GENERIC NAME**	**ROA**	**DOSE**	**FREQUENCY**	**DURATION**
INJ. PCM	Paracetamol	IV	30ml	SOS	DAY 1-2
INJ. ZOFER	Ondansetron	IV	2cc	SOS	DAY 1-2 DAY 5-7
TAB. PARACETAMOL	Paracetamol	PO	250 mg	SOS	DAY1-8
TAB. NIFEDIPINE	Nifedipine	PO	15mg	TID	DAY 1-4
T. ENALAPRIL	Enalapril	PO	25mg	BD	DAY2-12

T. DYTOR	Torsemide	PO	10mg	OD	DAY4-11
T. AMLODIPINE	Amplodipine	PO	5mg	OD	DAY4-5
T. NICARDIA RETARD	Nifedipine sustained release	PO	10mg	BD	DAY5-12
SYP. SUCRAL	Sucralfate	PO	5ml	TID	DAY5-10
T. JR LANZOLE	Lansoprazole	PO	30 mg	0D	DAY5-10
T. CEFIXIME	Cefixime	PO	100mg	BD	DAY5-6
T. WYSOLONE	Prednisolone	PO	15mg	BD	DAY5-11
T. AUGMENTIN	Amoxycillin+ clavulanic acid	PO	650mg	BD	DAY7-12

3. DISCUSSION

Post-streptococcal glomerulonephritis (PSGN), a classic immune complex-mediated glomerular disease, typically arises following infection with nephritogenic strains of Streptococcus pyogenes. Examples of circulating and in situ immune complexes that support the pathogenesis are streptococcal pyrogenic exotoxin B and nephritis-associated plasmin receptor (NAPlr), two streptococcal antigens that accumulate across the glomerular basement membrane and mesangium [5,7]. These deposits trigger the alternate pathway of the complement cascade, which leads to hypocomplementemia, inflammatory cell recruitment, and glomerular capillary wall degradation [5]. Numerous factors contribute to the development of hypertensive urgency in PSGN. Acute glomerular inflammation causes a marked reduction in glomerular filtration rate, which increases intravascular volume and hinders the removal of water and salt [6]. Systemic vasoconstriction and increased blood pressure are caused by this volume overload as well as renal ischemia-induced activation of the renin-angiotensin-aldosterone system (RAAS) [8]. Moreover, endothelial dysfunction brought on by complement activation and inflammatory cytokines may worsen hypertension by raising peripheral vascular resistance [7].

Hypertensive urgency or emergency can occur when blood pressure quickly increases to levels linked to the risk of end-organ damage, as this instance has shown. Blood pressure management, fluid overload correction, and preservation of renal function are the primary goals of supportive therapy for PSGN made worse by hypertensive urgency. The mainstays of treatment are dietary restrictions on salt and water [6]. Because they encourage natriuresis and lower intravascular volume, loop diuretics especially furosemide are first-line treatments for hypertension and edema. In cases of severe hypertension, short-acting antihypertensive medications, such as intravenous vasodilators or calcium channel blockers, may be necessary to achieve regulated blood pressure [6]. Because they may increase hyperkalemia or induce acute kidney injury, angiotensin receptor blockers and angiotensin-converting enzyme inhibitors are often delayed during the acute phase, despite the fact that they may be useful in lowering intraglomerular pressure [6].

Renal function, electrolyte balance, and urine production all need to be closely watched. Acute renal replacement therapy may be necessary for patients with severe hyperkalemia, intractable fluid overload, or uremic sequelae [6]. Even while PSGN is typically self-limiting, especially in juvenile populations, adults have a higher risk of chronic renal impairment and require regular monitoring [5].

Emerging research suggests new therapeutic targets for severe or atypical PSGN. The plasminogen–plasmin system has been connected to hypertension and sodium retention by aberrantly activating epithelial sodium channels; amiloride inhibition of this pathway has been proposed as a potential additional therapy [7]. Immunomodulatory techniques such corticosteroids, plasmapheresis, or B-cell-targeted therapies have been documented in rare cases of rapidly developing or crescentic

glomerulonephritis, despite the paucity of reliable clinical trial data [9,10]. Examples of experimental strategies that are being researched and may have therapeutic promise in the future include complement system inhibitors and the development of a streptococcal vaccine [7,9].

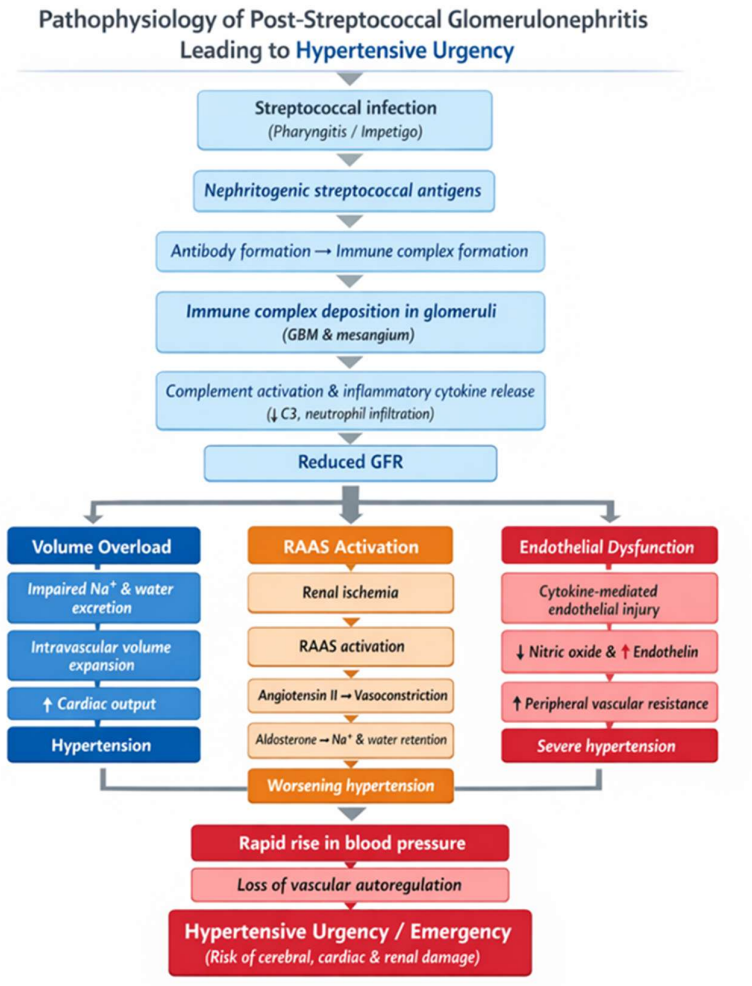

Fig. 1

4. CONCLUSION

Post-streptococcal glomerulonephritis continues to be a major cause of acute nephritic syndrome in children, particularly in low-resource settings. This illustration highlights hypertensive urgency as a noteworthy and potentially hazardous PSGN consequence that arises from immune-mediated glomerular injury that results in renin-angiotensin-aldosterone system activation and water and salt retention. Early detection of clinical signs such edema, hematuria, and elevated blood pressure, along with prompt laboratory testing, are essential for prompt diagnosis. With supportive care that focused on blood pressure control, volume regulation, and appropriate antimicrobial therapy, this patient had positive clinical outcomes. This example highlights the importance of careful monitoring and timely management in order to prevent acute issues and long-term renal consequences in children with PSGN.

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