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Case Presentation

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Poststreptococcal Glomerulonephritis in an 11-Month-Old Female Infant: Case Report and Literature Review

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Abstract

Background: Acute post-streptococcal glomerulonephritis (APSGN) is a well-recognized immune-mediated complication following group A β-hemolytic streptococcal infections. It predominantly affects children aged 5-12 years and is exceedingly rare in infants below two years of age due to their lower immunogenicity and decreased susceptibility to streptococcal pharyngitis.

Case-diagnosis/treatment: We report an 11-month-old female infant who presented with fever, gross hematuria, and nephrotic-range proteinuria. Laboratory findings revealed acute kidney injury, anemia, hypocomplementemia, and bilateral nephromegaly. Despite negative ASLO titers and sterile cultures, the clinical and laboratory features were consistent with APSGN. Follow-up testing revealed positive anti-DNase B antibodies,

confirming recent streptococcal infection. The patient was managed conservatively with close monitoring of fluid balance and renal function. She demonstrated gradual improvement, with complete resolution of proteinuria and normalization of renal function by 18 months post-presentation, allowing for discharge from nephrology care.

Conclusions: This case, representing one of the youngest reported cases of APSGN in the literature, underscores the importance of considering this diagnosis in infants presenting with hematuria, proteinuria, and acute kidney injury, even in the absence of documented streptococcal infection. Our comprehensive literature review identified only three previous reports of APSGN in children under the age of two, making this case particularly noteworthy. This report contributes to the limited literature on infantile APSGN and underscores the need for a high index of suspicion when evaluating urinary abnormalities in very young children.

Keywords: Acute post-streptococcal glomerulonephritis; Infant; Atypical presentation; Nephrotic-range proteinuria; Hypocomplementemia

Introduction

The association between renal disease and infection has been known since the time of Bright in the mid-1800s **[1].** Post-Infectious Glomerulonephritis (PIGN) is aglomerular disease caused by an immune response triggered by bacterial, viral, or protozoal infections **[2].** Group A B-Hemolytic Streptococcus (GAS) infections, commonly occurring in children, can lead to a wide range of diseases, from superficial infections to invasive diseases and postinfectious complications like Acute Post-Streptococcal Glomerulonephritis (APSGN) **[3,4].** APSGN remains the most common form of Glomerulonephritis (GN) in children worldwide, characterized by inflammation in the glomeruli and vasculature of the kidneys resulting from immune complex formation following a GAS infection **[3-5]**. An estimated 470,000 cases occur annually, with 97% in developing countries where impetigo and other pyodermal skin infections are prevalent, and around 5,000 cases (1%) resulting in death, mainly in children **[4-5]**. APSGN exhibits seasonal patterns, peaking in summer/fall in temperate regions due to skin infections and winter/spring due to pharyngitis **[5].** It disproportionately affects poorer, rural, and indigenous communities all over the world **[5].** The serotypes most associated with pharyngitis are M types 12, followed by 1, 4, and 25, whereas types 49, 2, 42, 56, 57, and 60 cause skin infections **[5,6].** APSGN is rare in individuals under two years old due to their low immunogenicity, which results in weaker immune complex formation and, consequently, less APSGN **[7].** The spectrum of clinical manifestations in PSGN ranges from asymptomatic to acute renal failure. Morbidity arises from volume overload (causing edema, hypertension, and congestive heart failure) and renal manifestations (macroscopic hematuria and usually mild renal insufficiency); severe cases may require dialysis. Long-term outcomes in children are generally excellent, although some controversy exists regarding them, and mortality is low **[1,2,8]**.

Recent reports highlight potential delays in diagnosis, often due to the expectation of a clear history of preceding documented streptococcal infection. Some studies have excluded patients without such a history. While textbooks typically describe PSGN onset as 1-2 weeks post-streptococcal pharyngitis or 3-6 weeks post-pyoderma, this expectation may lead to missed diagnoses in atypical presentations **[8].** There is no

definitive treatment for APSGN, apart from penicillin, to eradicate GAS carriage. While steroids have been used in severe adult cases, their use is not established in children. Management primarily focuses on providing supportive care and controlling symptoms **[7].** We report an 11-month-old girl presenting with typical APSGN symptoms and lab findings but failure to prove streptococcal infection.

Case Presentation

A previously healthy 11-month-old girl presented to the Emergency Room (ER). For a week, she had a fever, and in the last four days, her parents noticed her urine was red. She had a cough and decreased intake, with a recent resolution of vomiting and diarrhea. Two weeks earlier, she'd seen her pediatrician for fever and enlarged, red tonsils. Given her age, it was thought to be viral tonsillitis. No culture was taken, and the fever resolved spontaneously. There was no history of pyodermal skin infections. Of note, her father had been diagnosed with streptococcal pharyngitis via rapid antigen test at an urgent care clinic approximately a week before the onset of her symptoms. Prior to the main presentation, the patient had been seen at our ER for a fever a few days earlier. At that time, examination and tests were normal except for a mildly elevated CRP (3.88 mg/dL) and urine showing +4 erythrocytes with mild proteinuria (+1). Her urine culture was sterile. She was discharged with instructions for follow-up, likely assuming it would resolve on its own. The patient's medical history was unremarkable for kidney diseases, and her only recent medication was weight-appropriate ibuprofen for fever management, about ten doses over the week she had been sick.

Upon presentation to the ER, the patient appeared visibly ill, weak, and moderately dehydrated, with dry lips and delayed capillary refill. While her vitals were: Blood Pressure (BP) 108/67 mmHg (95^{th} percentile), Heart Rate (HR) 120 beats/minute, she also had a fever. Lab results revealed: normal CBC without leukocytosis but with mild anemia Hb (10.5 g/dL), Chemistry with elevated creatinine (0.83 mg/dL), urea (126 mg/dL), and mildly elevated potassium (5.7 mEq/L), CRP (3.88 mg/dL), triglycerides (256 mg/dL), and cholesterol (160 mg/dL). Complement studies showed low C3 (18 mg%) with normal C4. ASLO was normal, while IgG and IgA were elevated (1190 mg/dl, 116 mg/dl) **(Table 1)**. Urine analysis via catheter revealed significant proteinuria (500 mg/dL protein), hematuria (250 cells/µL of red blood cells), and 75 cells/µL leukocytes, with erythrocytes visible on microscopy but without casts, indicating an elevated protein-to-creatinine ratio of 14, consistent with nephrotic range proteinuria. Blood and urine cultures were sterile **(Table 1)**. Furthermore, EKG and coagulation profile were normal, renal ultrasound demonstrated bilateral kidney enlargement (right 8 cm, left 7.5 cm) (6.2 cm the normal value for her age), and Doppler ultrasound ruled out renal vein thrombosis **(Figure1).**

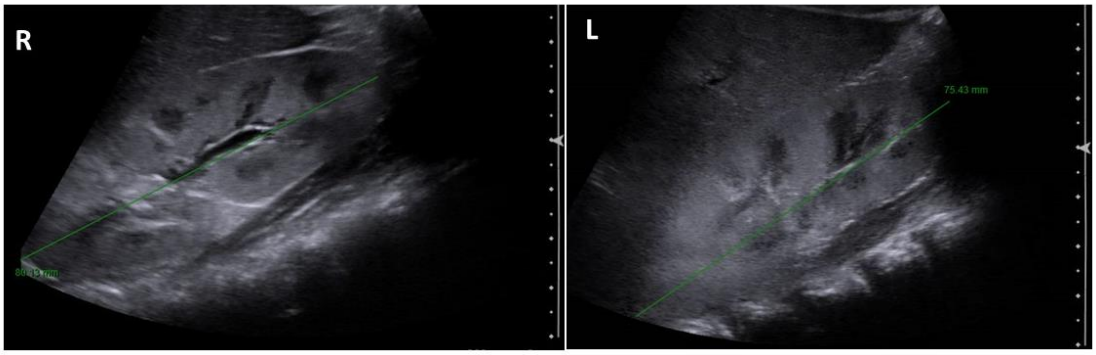

Figure 1: Renal ultrasound demonstrated bilateral kidney enlargement.

The patient's condition was closely monitored during her hospitalization with repeated laboratory tests. Her albumin briefly dropped to 3.1 mg/dL but later normalized. She experienced mild foot edema with a 1 kg weight gain, which resolved as she improved. The protein-to-creatinine ratio initially worsened to 17, then decreased to 8.1. Blood pressure remained stable at the 75th percentile. Her clinical presentation was consistent with glomerulonephritis, likely post-streptococcal, associated with nephrotic-range proteinuria and low C3. However, close follow-up was necessary to confirm the final diagnosis with extensive testing, including repeated ASLO, throat culture, and comprehensive testing for infectious causes, including Hepatitis C, Influenza A and B, RSV, Parainfluenza, Metapneumovirus, Rhinovirus, and Adenovirus, which yielded negative results. Serological testing for Parvovirus indicated a past infection. Additionally, rheumatological tests were unremarkable. Follow-up evaluations were conducted at two and four weeks post-discharge. These visits revealed significant improvement in both clinical presentation and laboratory parameters. During the two-week follow-up appointment, additional serological testing was performed, including an anti-DNase B test, which returned positive (380 units, reference range <170 units). This finding, in conjunction with the clinical presentation and the epidemiological link to her father's streptococcal infection, further supported the diagnosis of post-streptococcal glomerulonephritis, even in the absence of a positive ASLO test. While no specific pathogen was identified, the patient's initial presentation, characterized by low complement levels and the overall clinical picture, remained consistent with post-streptococcal glomerulonephritis. However, this diagnosis was made with the limitation that the causative pathogen could not be identified.

In summary, this case study follows an 11-month-old girl who presented with hematuria, nephrotic-range proteinuria, low C3 with normal C4, and acute renal failure. Despite a negative ASLO test, her clinical picture strongly indicated post-infectious glomerulonephritis, likely post-streptococcal. Additional serological workup, including anti-DNase B testing, was performed during follow-up to confirm our clinical suspicion, which returned positive, providing definitive evidence of a recent streptococcal infection. Regular follow-ups at the nephrology clinic showed steady improvement. By 18 months post-incident, the patient had achieved full

recovery with normal kidney function, allowing for her discharge from ongoing nephrology care.

Discussion

APSGN, a well-known complication of GAS infections, is the most common form of glomerulonephritis in children. It typically affects preschool and school-age children, between 5 and 12 years old, and is uncommon in children under three years old and rare in infants [7,9-12]. However, cases in very young children are unusual; reports dating back to the 1950s document acute nephritis in infants as young as four months [9,13]. The incidence of APSGN ranges from 30–300 per million population annually, predominantly affecting children over two years old [14,15]. Our comprehensive literature review revealed only three other reported cases of APSGN in children under two years, with the youngest being eight months old, making this case of an 11-month-old patient particularly noteworthy [12]. The rarity of APSGN in very young children <2 years old can be attributed to two main factors: the infrequency of GAS pharyngitis in this age group and impaired immunogenicity.GAS is carried in both the throat and nose of humans.GAS carriage sites vary with age, predominantly found in the nares of younger children and the pharynx of older ones [9,16,17]. In addition, the rates of carriage and colonization have also been shown to vary by age, being highest in children 5–10 years of age [18]. Recent studies have suggested that GAS throat isolation from the throat of children less than three years of age may be more common than previously thought, especially between ages 2-3. However, distinguishing between true infections and carrier states remains challenging [19,20]. Nussinovitch et al. demonstrated that the incidence of both carrier state and true streptococcal pharyngitis (confirmed by positive throat culture and elevated ASLO titers)gradually increases with age, with true infection found in 3–4% in children less than 2 and 10% in children 2–10 [9,17,21]. The clinical presentation of APSGN can vary widely. Standard features include macroscopic hematuria (64%), microscopic hematuria (32%), arterial hypertension (64%), mild renal failure (43.7%), and nephrotic syndrome (25%); notably, 22% of cases present with severe symptoms such as congestive heart failure, severe hypertension, or encephalopathy. Despite this range of presentations, APSGN is generally self-limiting, with most patients achieving complete recovery [7]. Our patient exhibited several classic features of APSGN, including hematuria, proteinuria, and hypocomplementemia. The presence of nephrotic-range proteinuria in our case is noteworthy, as it occurs in only 20-25% of APSGN cases and may indicate a more severe clinical course. This underscores the importance of thorough evaluation and monitoring in such cases, even though APSGN typically resolves on its own.

The role of complement in APSGN pathogenesis and prognosis has been a subject of debate. Some view it as a poor prognostic factor, while others consider it a key sign of post-streptococcal APIGN [22-26]. The low C3 levels observed in our patient, along with normal C4 levels, are typical of the alternative complement pathway activation seen in APSGN. ASLO titers typically peak within 15 days and normalize after several months; for optimal diagnosis of streptococcal infection, at least two ASLO measurements and simultaneous anti-DNASE B testing are recommended [25,26]. One study demonstrated that only 30% of patients received two ASLO dosages, with positive results in just one case.

This contrasts with a 37% positive ASLO rate reported at the University Hospital in Donka, Guinea [22,26]. Despite normal ASLO results in our case, the positive anti-DNase B test obtained during follow-up provided strong serological evidence of a recent streptococcal infection, confirming our clinical suspicion of APSGN. This highlights the importance of comprehensive serological testing in suspected post-streptococcal glomerulonephritis cases, particularly when initial ASLO tests are negative, as anti-DNase B antibodies often remain elevated for longer periods than ASLO titers and may be more reliable in cases involving renal manifestations. Anemia was present in most patients (90%) in a Nigerian study, and severe anemia affected one-third of the study cases, a finding similar to those in Guinea (35%). While anemia due to hemodilution is common in glomerulonephritis, severe cases warrant investigation for chronic causes [26-28]. Furthermore, a 2015 study across 11 French-speaking African countries reported anemia in 72% of cases, highlighting its prevalence in diverse settings [26]. Our patient presented with anemia as well, similar to the other two cases by Bingler and Kari [9,29].

Table 1-3 summarize the four case reports from different countries, including our case of patients under two years old. The cases share several common features typical of APSGN, including a recent history of upper respiratory infections, presentation with hematuria, proteinuria, and edema. Laboratory findings consistently show low complement C3 levels, elevated ASLO titers (excluding our case), and varying degrees of renal impairment. Urinalysis in all cases revealed significant proteinuria and hematuria. The cases differ in severity and specific presenting symptoms, with some patients showing more severe manifestations like hypertension or nephrotic-range proteinuria. These cases highlight that the consistent finding of low C3 levels across all cases underscores its importance as a diagnostic marker for APSGN, even in this young age group.

Table 1: Clinical Characteristics of Reported Cases of APSGN in Infants.

Characteristics	Volti 1993	Bingler 2006	Kari 2013	Kasim Ali 2024
Age (Months)/Gender	8 / F	14 / M	14 / F	11 / F
Country	Italy	USA	Saudi Arabia	Israel
Previous History	Otitis and pharyngitis	Not specified	URTI	Viral tonsillitis
Presentation	Oliguria, dark urine, palpebral edema, vomiting	Gross hematuria 1 week following mild illness with palpable temperature	4 days of progressive periorbital puffiness, edema in lower limbs, tea-colored urine	Fever, gross hematuria, decreased intake, weakness
BP	125/70	88/40	110/70 (HTN)	108/67
Initial Urine Test	Proteinuria 300 mg, Hematuria 80-100, Several casts	Blood +3, Protein +3, 60 WBC	Protein > 300 mg/dl, Numerous RBCs, negative WBC	Protein 500 mg/dl, RBC 250 cells/µL, WBC 75 cells/µL
Culture	Throat culture GAS type 12	Throat culture GAS	Negative	Sterile

Table 2: Laboratory Values in Reported Cases of APSGN in Infants.

Lab Values	Volti 1993	Bingler 2006	Kari 2013	Kasim Ali 2024
WBC (K/UL)	Normal	14.8	8.7	12
Hemoglobin (g/dl)	Normal	9.6	9.1	10.5
Platelets (K/UL)	NA	708	1038	330
C3 (mg%)(55-120)	33	35	16.9	18
C4 (mg%)(16-38)	11	22	21.6	24
ASO (IU/l)	450	470	3320	30
Creatinine (mg/dl)(0.2-0.4)	0.77	0.5	0.36	0.82
Urea (mg%)	68	17	14	122
Albumin (g/dl)	NA	2.7	2.1	3.4
Total Protein (g/dl)	NA	6.1	6.4	6.3
LDH (U/l)	NA	NA	NA	617
CRP (mg/dl)	NA	NA	NA	1.64
ESR (mm/h)(0-10 mm/h)	55	>140	NA	49

Table 3: Additional Clinical Information and Management in Reported Cases.

Additional Info	Volti 1993	Bingler 2006	Kari 2013	Kasim Ali 2024
US Findings	NA	Bilateral nephromegaly	NA	Bilateral nephromegaly
Treatment	NA	NA	Prednisone 2mg/kg/d 4 weeks, then tapering down	Supportive treatment
Additional Labs	IgG2 250 (300-3270)	IgG 1260, IgA 202, IgM 79	24 hr urine collection -- nephrotic range proteinuria (2.5 g)	IgG 1038, IgA 59.9, IgM 123,anti-DNase B 380

The management of APSGN in infants follows the same principles as in older children, focusing on supportive care and careful monitoring of fluid balance, blood pressure, and renal function **[30].** In our case, the patient experienced transient mild edema and weight gain but did not necessitate any specific interventions beyond close observation. This aligns with the generally favorable prognosis of APSGN in children, even in atypical presentations **[23].** The literature reveals a range of treatment approaches. Notably, Kari documented a case of 14 a 14-month-old baby where prednisone was employed as part of the therapeutic regimen **[29].** Long-term follow-up is crucial in cases of infantile APSGN due to the limited data on long-term outcomes in this age group. While the majority of children with APSGN recover completely, there is ongoing debate about potential long-term sequelae, particularly in terms of hypertension and chronic kidney disease **[31,32].** Follow-up data from all four cases indicated improvement in clinical and laboratory parameters, emphasizing that our patient's complete recovery by 18 months post-incident is encouraging; however, continued vigilance is warranted.

Conclusion

This case report underscores the importance of considering APSGN in infants presenting with hematuria, proteinuria, and acute kidney injury, even in the absence of documented streptococcal infection. The diagnosis can be challenging in this age group due to its rarity and atypical presentation in this age group. Our experience emphasizes the need for a high index of suspicion when evaluating urinary abnormalities in very young children, particularly for hematuria and nephrotic syndrome. This case contributes to the limited literature on APSGN in infants and highlights the need to consider this diagnosis across all pediatric age groups, potentially leading to a reevaluation of age-based assumptions in APSGN diagnosis and management.

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