

Case Report

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Hypertensive Cardiogenic Shock in a Child with Acute Poststreptococcal Glomerulonephritis: An Unusual Presentation - A Case Report

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Introduction

Acute poststreptococcal glomerulonephritis (APSGN) is characterized by an abrupt onset of hematuria, edema, hypertension, oliguria, and diminished renal function after group A hemolytic streptococcus infection [1]. In the pediatric age group, acute poststreptococcal glomerulonephritis (APSGN) accounts for approximately 80% of the cases of acute nephritic syndrome (ANS) [1,2]. In developed countries, the incidence is estimated to be 0.3 cases per 1,00,000 children per year according to Italian Biopsy Registry.

Acute poststreptococcal glomerulonephritis (APSGN) is one of the most common renal diseases resulting from a prior infection with group A β -hemolytic streptococcus. Manifestations of acute poststreptococcal glomerulonephritis ranges from subclinical infections to life threatening conditions. Typical clinical features of the disease include an acute onset with gross hematuria, edema, hypertension and moderate proteinuria (acute nephritic syndrome) 1 to 2 weeks after an antecedent streptococcal pharyngitis or 3 to 6 weeks after a streptococcal pyoderma. Patients with APSGN sometimes exhibit unusual clinical manifestations, which may lead to diagnostic delay or misdiagnosis of the disorder. Cardiogenic shock is uncommon but potentially fatal initial manifestations of APSGN. There are very few reports of cardiogenic shock as the initial manifestation of APSGN. In patients presenting with cardiogenic shock, without a clear etiology, APSGN should be considered. We report a 07 year old boy presenting with cardiogenic shock as initial manifestations of APSGN.

Keywords: Glomerulonephritis; Shock, Cardiogenic; Hypertension; Infections; Streptococcal.

Running Title: Hypertensive Cardiogenic Shock in a Child with PSGN

In developing countries, the number of APSGN cases ranges between 9.5 and 28.5 new cases per year [3].

APSGN patients may develop heart failure as a complication of hypertension – initially left-sided heart failure followed by right-sided heart failure and pulmonary edema [4,5]. Other potential complications include acute kidney injury, encephalopathy, rapidly progressive glomerulonephritis, acidosis, hyperkalemia, hyperphosphatemia and hypocalcemia [3,6].

One percent of the patients develop chronic kidney disease in the long term [3]. Patients with APSGN sometimes exhibit atypical or unusual clinical manifestations like posterior reversible leukoencephalopathy syndrome, thrombotic microangiopathy, gallbladder wall thickening, uveitis, subglottic edema, and unusual clinical courses like minimal urinary abnormalities and recurrence [6].

Shock can occur in heart failure patients but very rare in APSGN patients. Acute post streptococcal glomerulonephritis is not a common cause of cardiogenic shock. We present a case of APSGN who developed cardiogenic shock as a rare presentation.

Case Report

A 7-year-old boy, the 1st child of his parents, was reasonably well until 10 days ago. When he came to Bangabandhu Sheikh Mujib Medical University, he complained about the swelling of the whole body for 10 days which started from the face and then became generalized. It was associated with scanty cola colored urine for the same duration. His alert mother also complained of respiratory distress for the last 1 day which was so severe in nature that he could not utter any words but was not associated with cough. Dyspnea was aggravated on the lying posture but had no diurnal variation. The mother also stated that the boy had a history of some pustular skin lesions on the chest, back, and left foot about one month back which healed with itching and desquamated over the next 20 days. He had no history of vomiting, headache, convulsion, fever, dysuria, sore throat, chest pain, rash, arthritis, or any feature of vasculitis. There was no history of any drug or poison intake that could cause this condition. His past, developmental, and family history were unremarkable. On examinations, he was semiconscious with a Pediatric Glasgow coma scale score of 11/15 (E3 V4M5), cyanosed, dyspneic, and puffy faced. His pulse was not palpable, BP was not recordable, and capillary refill time was 5 seconds. His respiratory rate was 70 per minute. His temperature was subnormal 36°C and his jugular venous pressure was elevated. His skin was cool and he was edematous. The partial pressure of oxygen was 88% and the heat coagulation test for protein was one plus. Anthropometrically, he was age appropriate. Skin examination revealed some old healed skin lesions on the chest, back, and on the left foot. On cardiovascular system examination, his heart rate

was 170 per minute; a gallop rhythm and basal crepitation were auscultated with no murmur. On gastrointestinal system examination, we found ascites evidenced by shifting dullness. The liver was enlarged 7.5 cm from the right costal margin along its long axis which was firm and tender. The rest of the physical examinations were unremarkable.

Immediate investigation showed serum creatinine=1.1 mg/dl, blood urea=76.0 mmol/L, Na=139.2 mmol/L, K=6.03 mmol/L, Cl=112 mmol/L, TCo2=12 mmol/L, and random blood sugar=7.1 mmol/L. Chest X ray revealed features of pulmonary edema with cardiomegaly. Complete blood count showed Hb=9.7 gm/dl and total count of white blood cells=19,000 (81% neutrophils). The peripheral blood film revealed moderate normochromic normocytic anemia with neutrophilic leukocytosis. Routine urine analysis showed 0-2 pus cells/HPF, 6-8 RBCs/HPF, 1+ protein, and RBC casts with >30% dysmorphic RBCs. Blood culture showed no growth and the echocardiography report was normal.

Based on history, clinical examination, and relevant investigations, we diagnosed the case as acute post streptococcal glomerulonephritis with cardiogenic shock due to hypertensive heart failure. The patient was so sick that we considered pneumonia septic shock in differential diagnosis. We immediately rushed to the patient and kept the patient in the propped-up position. We administered high flow (5L/min) oxygen through a face mask and 20ml/kg normal saline (200 ml) over half an hour very cautiously but since the patient did not improve, we started dopamine at 5 µg/kg/min, dobutamine 10 µg/kg/min, and other supportive measures like keeping the patient NPO, intravenous fluid, bladder catheterization, IV antibiotic Cefepime, IV sodium bicarbonate for correction of acidosis, IV calcium gluconate, and nebulization with salbutamol for the correction of hyperkalemia. Subsequently, we increased the dobutamine dose. The patient improved after 5 hours evident by a normal peripheral temperature. Oxygen saturation improved to 92%, his pulse became palpable and his respiratory rate slowed down to 47/min. His urine output was about 200 ml but BP was still unrecordable. We started nasogastric tube feeding and increased the dobutamine dose to 20 µg/kg/min. After 2 hours, his pulse was palpable at 150 beats/min and his blood pressure was 130/100 mmHg. We stopped IV fluid, dopamine, and dobutamine. Subsequently heart failure was managed with furosemide 5mg/kg and blood pressure was controlled with

oral nifedipine. Gradually, the boy improved and after 4 days, he was stable with a normal heart rate, blood pressure, and diuresis followed by a reduction in edema. After that, intravenous medications were stopped and nasogastric tube feeding was discontinued. Subsequent investigation showed a spot urinary protein to creatinine ratio of 1.75. Serum albumin was 28 gm/L and serum calcium was 6.4 mg/dl. HBs antigen was negative, ASO was 1160 IU/ml (normal <200IU/ml), and C3 was 0.184 mg/dl (normal: 0.9-1.8 gm/L). USG revealed normal size kidneys. His renal function tests on the 4th day of admission revealed serum creatinine was 0.7 mg/dl and blood urea was 34 mg/dl. He was discharged on the 9th day of admission. On follow-up after 2 weeks, the child was asymptomatic with his blood pressure well controlled. His vital signs including pulse, blood pressure, and respiratory rate were normal after 3 weeks without any medications. Eight weeks after hospital admission, his serum complement C3 level became normal (110 mg/dL).

Discussion

In APSGN, the glomerular filtration rate is decreased, leading to the activation of the renin-aldosterone system and subsequent salt and water retention that result in edema and hypertension. Also, the capacity to excrete salt and water is usually diminished, leading to expansion of the extracellular fluid (ECF) volume [7]. The expanded ECF volume is responsible for edema, hypertension, anemia, and circulatory congestion. Hypertension is present in 60–80% of the children with APSGN. Cardiogenic shock is uncommon but a potentially fatal initial manifestation of APSGN. APSGN may result in heart failure as a complication of hypertension – initially left-sided heart failure followed by right-sided heart failure and pulmonary edema simulating pneumonia or myocarditis, and a very severe cardiogenic shock [5]. In heart failure, often called congestive heart failure (CHF), the heart is unable to pump blood at a rate commensurate with the requirements of the metabolizing tissues or can do so only at an elevated filling pressure. Although usually caused by a slowly developing intrinsic deficit in myocardial contraction, a similar clinical syndrome is present in some patients with heart failure caused by conditions in which the normal heart is suddenly presented with a load that exceeds its capacity (e.g., fluid overload, acute

myocardial infarction, acute valvular dysfunction), or when ventricular filling is impaired [8].

APSGN patients may sometimes present with clinical and radiologic signs of pulmonary edema. While dyspnea is a presenting complaint in only 5% of the patients, the evidence of congestive heart failure was found in half of the children in one early series [9].

Shock is a life-threatening state that occurs when the delivery of oxygen and nutrients is insufficient to meet tissue metabolic demands. The cardiogenic shock, a low-output state, is characterized by an elevated ventricular filling pressure, a low cardiac output, systemic hypotension, and evidence of end organ hypoperfusion [10]. This failure may be due to depressed myocardial contractility, arrhythmias, volume overload, or diastolic dysfunction [8]. Physical examination findings consistent with a diagnosis of cardiogenic shock are tachypnea, an S3 gallop, wheezing or rales, dyspnea or cough, cyanosis, diaphoresis, hepatomegaly, jugular vein distention (JVD), and peripheral edema [10]. Chest radiography reveals cardiomegaly and pulmonary venous congestion [5] which were present in our case. Unlike other forms of shock in which the central venous pressure (CVP) is low, the CVP is elevated above 10 cm H₂O in cardiogenic shock. Initially, it may be impossible to differentiate cardiogenic shock from septic shock. Empiric treatment for possible septic or cardiogenic shock should not be delayed [11]. We treated the patient for both septic and cardiogenic shock.

This presentation in APSGN is uncommon. A review of the literature did not reveal any previous reports. A child presenting with heart failure and APSGN should be considered in the differential diagnosis along with other causes of shock. A high degree of suspicion and prompt treatment can prevent fatal consequences.

Conflict of Interest

None declared

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