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Case Report Open Access

Decreased Urine Output and Vomiting in a 4 Year Old Boy

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Abstract

Globally, post infectious glomerulonephritis is a common cause of acute glomerular disease. Persistent hypocomplementemia is known to be associated with dysregulation of the complement alternative pathway. Experimental evidence regarding the effect of viral illness in case of acute glomerular disease are very limited. The following report illustrates a unique presentation of post infectious glomerulonephritis associated with viral infection and discussion of C3 glomerulonephritis.

Keywords: Post infectious glomerulonephritis; Epstein Barr virus; C3 glomerulonephritis; Post streptococcal glomerulonephritis; Rhinovirus; Parainfluenza 1; Enterovirus

Introduction

Post infectious glomerulonephritis (PIGN) is the most common cause of acute glomerular disease in children worldwide. The global burden of disease had been above 450,000 cases annually. However the incidence has decreased over time with lower incidence of pyoderma and widespread use of antibiotics [1-3]. PIGN primarily affects children ages 3-12 years of age and is more prevalent in males than females. Within the subset of PIGN the most common infectious cause is a Group A Streptococcal infection (GAS), either pyoderma or pharyngitis. The onset of glomerulonephritis varies with the site of infection; occurring 1-2 weeks after skin infection and 3-6 weeks after pharyngitis [2].

Post streptococcal glomerulonephritis (PSGN) is caused by immune complex build up in the glomeruli however the exact mechanism for the formation of these complexes remains unclear. The most believed theory is that GAS antigens deposit on the glomerular basement membrane with subsequent binding to the antibodies resulting in immune complex formation [1,4].

The most common presenting clinical features include gross hematuria, edema, and hypertension. However, this triad of features is not required to make a diagnosis of PIGN. In this patient, he was not hypertensive or edematous which is likely due to his acute moderate dehydration preventing fluid overload. For the diagnosis of PSGN, one would expect low C3 with normal C4, confirmed recent GAS infection via either culture or elevated ASO titers and elevated BUN and creatinine. Often the FeNa is <1% which is normally associated with prerenal disease as opposed to glomerulonephritis. The urine protein to creatinine ratio is elevated. A biopsy is not indicated in PSGN unless associated with prolonged hypocomplementemia or persistence of hypertension and abnormal renal function.

Recently there is controversy regarding the diagnosis of PSGN versus C3 glomerulonephritis (C3G). It has been shown that persistent hypocomplementemia is associated with dysregulation of the complement alternative pathway and there is evidence that the 'atypical' PIGN with delayed recovery of hypocomplementemia and severe persistence of renal injury progressing to ESRD may actually be more consistent with C3G [4,5]. Unfortunately, the only way to distinguish between PIGN and C3G is kidney biopsy, which is not commonly indicated and available for patient with PIGN. Based on limited number of renal biopsy results, patients with PIGN have shown to have C3 and immunoglobulin deposition while C3G patients show only C3 with little or no immunoglobulin deposition. On electron microscopy PIGN shows subepithelial hump deposits while C3G shows no deposits [5,6]. Nevertheless, there is need for additional research of the complement alternative pathway function in setting of acute glomerulonephritis.

There has been limited research on the effect of viral illness on PIGN. EBV most commonly causes tubulointerstitial disease rather than glomerular injury [2]. Yet, there are two case reports that show EBV associated with endocapillary and mesangioproliferative glomerulonephritis [7].

Our patient was diagnosed with post infectious glomerulonephritis possibly secondary to previous group A streptococcal infection due to elevated ASO titer and transient low C3 level; however specific etiology remains unclear due to concurrence of five other confirmed, active infections. His respiratory viral panel was positive for rhinovirus, enterovirus and parainfluenza one. In addition, an EBV panel showed an active infection and clinical examination along with imaging studies confirmed a right upper lobe acute bacterial community acquired pneumonia. All of these may have contributed to his post infectious glomerulonephritis.

Case Presentation

A 4-year old boy was hospitalized with history of vomiting and decreased urine output. Patients' mother stated that he stopped eating solid food two days prior to admission and then started vomiting with all liquids 24 hours prior to admission. She also noted decreased urine output for 24 hours. He had associated diffuse, abdominal pain. He had a fever of 102°F at home which resolved with acetaminophen. Three days prior to admission he complained of sore throat, cough and rhinorrhea and was brought to his pediatrician's office. A rapid group A streptococcal antigen test and culture were negative.

On examination patient had vital signs and measurements as follows; heart rate 130 beats/min, respiratory rate 20 breaths/minute,

blood pressure 101/70 mm Hg with a height of 108 cm (80th percentile) and weight 19.3 kg (85th percentile). The positive physical examination findings included diminished air entry in right upper lung field with mild crepitation, his abdomen was non-tender, and there was no hepatosplenomegaly. He had no rash, edema, pallor or pharyngeal erythema/exudates, but had mild clear rhinorrhea.

Patient had chest and abdominal radiography performed which showed a large consolidation in the right upper lobe with a small right pleural effusion. There was a mass-like density in the right upper quadrant of the abdomen with displacement of the adjacent bowel loops (Figure 1).

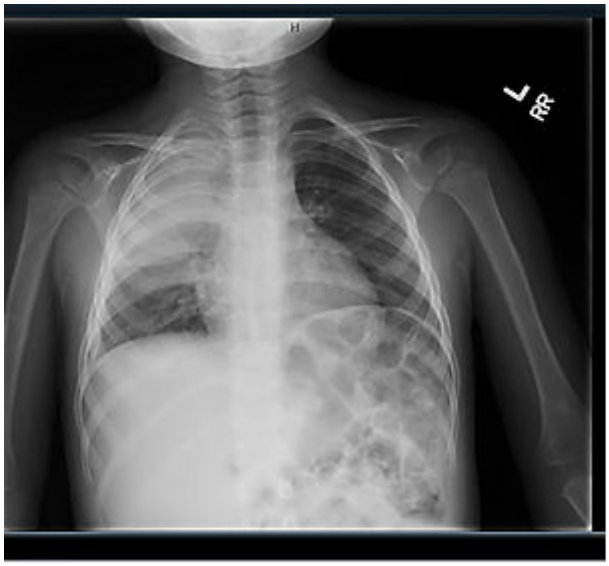

Figure 1: X-ray images showing large consolidation along with a small right pleural effusion.

Initial laboratory evaluation was significant for C-reactive protein of 257 mg/dL, albumin of 2.4 gm/dL leukocytosis of 25.6 × 103/μL, with 8% bands and 71% neutrophils. Basic metabolic panel results were as follows; sodium 126 mMol/L potassium 4.3 mMol/L chloride 91 mMol/L, bicarbonate 18 mMol/L, blood urea nitrogen (BUN) 31 mg/dL, creatinine 0.98 mg/dL. Liver enzymes were within normal limits. Further laboratory testing revealed the diagnosis.

Discussion

Because the abdominal radiograph was concerning for right upper quadrant mass, an abdominal ultrasound was performed which showed mild hepatomegaly and echogenic appearing and slightly enlarged kidneys with debris in the bladder. Urinalysis obtained because of oliguria and azotemia and results showed: urine red in color with 1+ ketones, SG 1.025, 3+ blood, 3+ protein, 2+ leukocyte esterase, nitrite positive, 50-100 white blood cells, and 2+ bacteria. Repeat urinalysis 4 hours later also showed 20-50 hyaline casts. Additional urine studies showed sodium 43 mMol/L, protein 304mg/dL, creatinine 90mg/dL, thus Fractional excretion of Na (FeNa)<0.3% and UPC>3.0. To complete the evaluation, complement levels were measured and showed a normal C4 level with low C3 level. Patient had elevated anti-streptolysin A (ASO) titers. Due to hepatomegaly Epstein Barr Virus (EBV) antibody panel was checked and was positive for an active EBV infection; however hepatitis panel was negative. Lastly, a respiratory viral panel was positive for rhinovirus, enterovirus and parainfluenza 1. Blood and urine cultures were negative.

Treatment was primarily supportive, aside from ampicillin for acute community acquired bacterial pneumonia. Fluids were initially restricted to 1.0L/day, approximately 1300 mL/m^2/day and he did not need any antihypertensive medicine during hospitalization because he remained normotensive. After 24 hours of intravenous antibiotics and fluids, he was tolerating liquids and a regular diet. He was discharged home with instructions for fluid restrictions and close nephrology follow up. At his 12 week follow up appointment he showed full recovery with normal renal function and normal C3 level.

Conclusion

Primary management for post infectious glomerulonephritis is supportive. The sequelae of PIGN present early on and are normally self-limiting. Patients require frequent reevaluation, especially when hypertensive. Diuretic medication may be started for hypertension and/or edema. The most commonly used diuretics are thiazide or loop diuretics until resolution of symptoms. High dose corticosteroids may be considered in patients severe enough to require renal biopsy; however this is not a commonly required [1]. Patients should be followed regularly until normalization of complement levels and return of normal renal function.

Acknowledgement

The authors wish to thank the patient for his consent to publish features of his case, the identity of the patient remains protected.

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