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Case Report

Post-Infectious Glomerulonephritis Associated with Necrotizing Pneumonia

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Abstract

Post-Streptococcal Glomerulonephritis (PSGN) in children is a well-documented entity that typically presents weeks after an infectious process by Group A Streptococcus (GAS). The case presented in this report serves as an example of Acute Glomerulonephritis (AGN) unrelated to streptococcal skin or throat infection. We present a case of a 5-year-old female with an episode of necrotizing pneumonia with concomitant findings suggestive of AGN who was managed conservatively. This case emphasizes that respiratory and renal findings should always raise concerns and require further evaluation.

Keywords: Acute Glomerulonephritis; Hematuria; Necrotizing Pneumonia; Post-Infectious Glomerulonephritis

Introduction

Post-Streptococcal Glomerulonephritis (PSGN) has been well documented after skin or pharyngeal infections [1]. However, there are only a limited number of cases of pneumonia-associated acute glomerulonephritis. A literature review yielded 15 cases of this rare entity in the last 16 years [2-4]; most cases required supportive measures only. In this review, we present a case of necrotizing pneumonia with concomitant acute glomerulonephritis.

Case Report

A five-year-old female presented to the emergency department with a two-day history of fever, vomiting, decreased appetite, and moderate to severe abdominal pain. The patient's, mother also noted brown urine for the past two days. During her initial work-up, she was found to have right consolidation pneumonia and was subsequently admitted to the inpatient unit for further management. On physical exam, she was afebrile, oxygen saturation 94% at 0.25L of Oxygen via nasal cannula and respiratory rate in the forties. She was pale and mildly dehydrated. Her cardiovascular exam was significant for tachycardia of 131 bpm, regular rhythm, no murmurs, normal S1 and S2. Chest exam showed decreased air exchange on the right mid-t- lower base. No wheezing or crackles. Left side with good air exchange, no added sounds. Abdominal exam revealed tenderness of epigastrium and right upper quadrant with no mass palpation or rebound. No CVA tenderness. Extremities and face with mild edema. Chest X-ray on admission showed a right-sided and probably left-sided pneumonia with moderate right pleural effusion. CT of the chest done without contrast revealed necrotizing pneumonia involving mainly right-sided lung, and suggestive of streptococcus pneumonia. Ultrasound of the kidneys revealed swelling but no hydronephrosis.

Laboratory studies showed a white blood cell count of 13,490/mm^3, hemoglobin of 10.5 g/dL, hematocrit of 31.5 g/dL, platelets of 219,000/mm^3, and C-reactive protein of 24 mg/dL. Further workup revealed BUN 25 mg/dL, creatinine of 0.9 mg/dL, total protein 5.2, and albumin 2.7. Macroscopic urine analysis showed gross orange color, 2+ protein, 2+ leukocyte esterase, specific gravity of 1.012, and large blood. Microscopic analysis revealed WBC of 89, RBC 305, no bacteria, 4 hyaline casts and 11 squamous epithelial cells. Renal workup was significant for low C3 and C4 complement levels of 28 mg/dL and 15 mg/dL respectively, creatinine in urine of 42, protein in urine of 329, ANA screen was positive with a ANA titer of 1:40 and speckled pattern, Anti DNase B antibody levels were <78 units/mL, Anti-Glomerular Basement Membrane (GBM) antibody of 5, proteinase-3 antibodies were <3.5 units/mL, and myeloperoxidase antibodies <9.0. Urine culture was negative throughout hospital course. On admission, she was started on ceftriaxone and clindamycin for pneumonia. Throughout the course of her hospitalization, she was weaned-off the oxygen as tolerated, fever trended down, WBC trended to normal, CRP decreased from 24.28mg/dL to 6.25mg/dL, and creatinine prior to discharge was 0.4mg/dL with a BUN of 4 mg/dL. Total protein level and Albumin continued to decrease down to 4.8 and 2.1 respectively. Protein in urine and leukocyte esterase at mid-hospital stay was 3+ for both, with urine WBC of 137. Her blood pressure became

mildly elevated during the latter part of her hospital stay, ranging from 113-132/ 77-90, at the 95 percentiles, but required no medication. Clinically, the patient started improving by hospital day 6 when gross hematuria and edema resolved. Patient was in the hospital for a total of 10 days and she was discharged home on oral antibiotics and conservative measures. One-month follow-up in the outpatient clinic showed normalized levels of C3 138mg/dL and C4 24 mg/dL, creatinine 0.34 mg/dL, albumin 4.3, ANA negative and CBC within normal limits. Her UA was remarkable for mild hematuria but no proteinuria. Her blood pressure was at 90 percentiles for age (Table 1).

	On presentation	Prior to discharge	One-month follow-up
CBC	WBC 13,490/mm^3,		Within normal limits
	Hemoglobin of 10.5 g/dL,		Within normal limits
	Hematocrit of 31.5 g/dL,		Within normal limits
	Platelets of 219,000/mm^3,		Within normal limits
C-reactive protein	24 mg/dL	6.25 mg/dL	
CMP	BUN 25 mg/dL	4 mg/dL	
	Creatinine of 0.9 mg/dL	0.4 mg/dL	0.34 mg/dL
	Total protein 5.2	4.8	
	Albumin 2.7	2.1	4.3
Macroscopic UA	Orange color		
	Protein 2+	3+	0
	Leukocyte esterase 2+	3+	0
	Specific gravity of 1.012		
	Large blood		Mild
Microscopic UA	WBC of 89	137	
	RBC 305		
	Bacteria 0		
	Hyaline casts 4		
	Squamous epithelial cells 11		
Nephrology work-up	C3 28 mg/dL		138 mg/dL
	C4 15 mg/dL		24 mg/dL
	Creatinine in urine of 42		
	Protein in urine of 329		
	ANA (+), 1:40, speckled pattern		(-)
	Anti DNase B antibody <78 µ/mL		
	GBM antibody 5		
	Proteinase-3 antibodies <3.5 µ/mL		
	Myeloperoxidase antibodies <9.0.		

Table 1: Shows patient's data.

Discussion

PSGN typically occurs after an infection with Group A streptococcus (*S. pyogenes*) involving the skin or throat. PSGN is most commonly seen in pediatric patients between the ages of 3-6 years old, and more commonly in males than females. There have been several mechanisms postulated that explain kidney damage following a streptococcal infection: cross-reactivity of antibodies against the Glomerular Basement Membrane (GBM), deposition of immune complexes, and/or deposition of streptococcal antigens on GBM with further antibody binding. In any case, there is an activation of the complement cascade with further inflammatory response. The clinical course of the disease is dictated by the ability to clear this inflammatory response. In current literature, there are not many cases describing AGN following a lung infection. This is the case of a 5-year-old Female who had an episode of necrotizing pneumonia during which time she presented with gross hematuria and edema with remarkable labs showing low C3 and C4 levels, transient elevated creatinine and blood pressure, proteinuria, and hematuria. Given this clinical picture and laboratory findings, she was diagnosed with glomerulonephritis secondary to infectious pneumonia suggestive of streptococcal infection. Kidney biopsy was not performed due to normalizing complement levels during her follow-up period. She required no further medical management and her kidney injury self -resolved [5-9].

Due to its rarity the incidence of this pneumonia-related complication is unknown and further investigation is required. However, a clinical picture such as the one described, should always alert the physician of possible renal injury in order to monitor and trend blood pressure and creatinine levels. Furthermore, fluid management and avoidance of nephrotoxic drugs is important to prevent further kidney damage. Although watchful-waiting with supportive measures approach is the standard of care in PSGN [1], there have been some cases reported where anti-hypertensive medication and dialysis are required to compensate for decrease renal function, and should always remain a consideration [4].

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