Abstract Form

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Project Title:	Poststreptococcal glomerulonephritis following Group A Streptococcus pyogenes wound infection with bacteremia

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction: The occurrence of AKI is commonly seen during hospitalizations with an estimated 1 in 5 adults (Per KDIGO definition) developing AKI and occurring in up to 50% of all critically ill patients. In the acute setting, rapid declines in renal function are often associated with immediate insults however a broader initial differential should not be excluded. The following describes a case of delayed AKI due to poststreptococcal glomerulonephritis (PSGN) following a group A streptococcus (GAS) wound infection with bacteremia.

Case Report: 61-year-old male with a past medical history of type 2 diabetes complicated by Charcot's neuroarthropathy, peripheral vascular disease, dyslipidemia, and hypertension was admitted for right lower extremity (RLE) cellulitis for 3 days. He has had prior admissions due to RLE infections including right heel osteomyelitis necessitating right calcaneal debridement and graft application with wound vacuum-assisted closure (VAC) device placement. Physical exam was remarkable for RLE swelling and warmth with mild erythema and serosanguinous fluid drainage surrounding the wound VAC site. Initial labs were significant for leukocytosis with WBC of 22.2, BUN of 45, creatinine (Cr) of 1.54, CRP of 30.32, ESR of 100, and procalcitonin of 4.06. MRI of the RLE noted osteomyelitis at the calcaneus with fracture. Blood cultures obtained were positive for Group A Streptococcus pyogenes. A bedside debridement was performed with wound cultures also positive for streptococcus pyogenes, pseudomonas aeruginosa, and rare Enterobacter cloacae. Treatment was started with IV Unasyn then transitioned to PO levofloxacin with an expected 6-week course however changed to IV cefepime due to concern for tendon rupture of the right shoulder. Infectious markers resolved and lab values returned to normal limits. Starting on hospital day 27, routine labs noted new worsening renal function with Cr tripling from 1.04 to 3.31 prompting nephrology consult. Initial differentials included ATN due to episodes of relative hypotension however no improve was seen following blood pressure normalization or adequate fluid resuscitation. Renal ultrasound was largely unremarkable. UA was significant for 3+ blood and 2+ protein. Urine studies obtained exhibited a fractional excretion of sodium of 1.2% suggesting intrinsic renal disease. Estimated 24hr urinary protein excretion was calculated to 2.5g/day suggesting nephritic syndrome. Serologic studies obtained found decreased C3 (78mg/dl) and elevated Streptolysin O Ab. (443IU/ml). CT guided biopsy of the left kidney was performed and demonstrated subacute postinfectious glomerulonephritis superimposed on background diabetic nephropathy, hypertensive nephrosclerosis with approximately 30% of global glomerulosclerosis, and moderate tubulointerstitial scarring without active crescent glomerular injury identified. A trial of prednisone 80mg daily was briefly initiated but discontinued due to lack of improvement. Ultimately HD was required to be initiated with Cr peak of 5.64 and was continued at discharge. On follow-up, it was found that the patient's renal function recovered to baseline (Cr. 1.07) and HD was no longer required 4 weeks post-discharge.

Discussion: PSGN is a delayed complication due to nephritogenic strains of group A beta-hemolytic streptococcus, propagated by an immune complex-mediated inflammatory response (type III hypersensitivity reaction). Latency of onset varies based on initial GAS infection with PSGN traditionally presenting 1-3 weeks after pharyngitis or more commonly up to 6 weeks following impetigo or skin infections. Clinical manifestations span a spectrum of symptoms including asymptomatic elevation in serum Cr, microscopic hematuria, proteinuria, generalized edema, hypertension, cola colored urine, hypertensive encephalopathy, and fulminant acute nephritic syndrome. Due to the rapid increase in accessible treatment, incidence of PSGN has significantly decreased with approximately <30 cases per 100,000 individuals annually and over 95% of reported cases occurring in developing countries. Although prerenal disease or ATN can contribute to up to 60% of AKI cases, it is important that clinicians retain an initially broad list of differentials when approaching patient's presenting with an AKI especially when encountering a patient with an infection well known to cause PSGN.