Infective Endocarditis-Associated Glomerulonephritis versus Cryoglobulinemic Glomerulonephritis: An Unfortunate Clinical Overlap

Yesha K. Shah DO  
*Lehigh Valley Health Network*, Yesha.Shah@lvhn.org

Emilee E. Kurtz DO  
*Lehigh Valley Health Network*, Emilee.Kurtz@lvhn.org

Sharon E. Maynard MD  
*Lehigh Valley Health Network*, Sharon_E.Maynard@lvhn.org

Mohammad Saqib MD  
*Lehigh Valley Health Network*, Mohammad.Saqib@lvhn.org

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Introduction
Infective endocarditis (IE) occurs in 30-60% of patients with Staphylococcus aureus bacteremia and carries a mortality rate of 40-50%. The most common comorbidities are cardiac valve disease, intravenous drug use, and hepatitis C. Glomerulonephritis due to infective endocarditis most commonly presents with AKI and complicated staphylococcal tricuspid valve infection. The most common pattern of glomerular injury is necrotizing and crescentic glomerulonephritis. Cryoglobulinemia (CG) occurs when the serum contains cryoglobulins, either single or mixed, which precipitate at low temperatures. This can result in an immune complex mediated, small-to-medium vasculitis that most commonly affects the kidneys, presenting as a mesangio proliferative glomerulonephritis. Common known infectious causes of cryoglobulinemia are Hepatitis C and HIV. However, IE-associated glomerulonephritis can also present as an immune mediated phenomena and present as a mesangio proliferative glomerulonephritis similar to that seen in cryoglobulinemic glomerulonephritis. The case below discusses a patient with a history of intravenous drug use who was discovered to have Hepatitis C.

Case Presentation
44-year-old male with past medical history of polysubstance use presented to an outside hospital with foot pain and fever. Blood cultures demonstrated MSSA bacteremia. Transesophageal echocardiogram was performed with findings of tricuspid valve endocarditis. He was initiated on Oxacillin and Daptomycin with subsequent transfer to our hospital for further evaluation by cardiothoracic surgery. Unfortunately, his renal function declined significantly during the course of his hospital stay – initially his creatinine was 0.7 (baseline) with progressively worsening renal function to a peak creatinine of 5.59 after eight days. Urinalysis demonstrated proteinuria, hematuria, positive leukocyte esterase, WBCs, RBCs and 2+ bacteria. Protein/creatinine ratio was 3.34 mg/dL. C3 and C4 complement levels were assessed and found to be low at 17 mg/dL and 9.5 mg/dL respectively. ANA was positive. Cryoglobulin qualitative screening was positive. PR3 was positive at 28 RU/mL and MPO was negative at <9 RU/mL. Rheumatoid factor, GBM autoantibody were checked and negative. Additionally, given the patient’s history of intravenous drug use, an HIV and hepatitis panel were checked with the former negative and the latter positive. Viral load was positive with HCV RNA 4.62 million. Cryoprecipitates were checked given the aforementioned positive results with findings of positive IgG, IgA, and IgM. Renal biopsy was ultimately completed three days after transfer in order to confirm the diagnosis and guide further management. The biopsy came back with the following results: “electron dense deposits, mesangial, global, subendothelial segmentsa, subepithelial and intramembranous, segmental, foot process effacement. Results consistent with diffuse proliferative GN, most consistent with cryoglobulenic glomerulonephritis.”

Discussion
This case highlights a diagnostic dilemma and demonstrates the importance of a thorough evaluation due to the overlapping features of IE and mixed cryoglobulinemia.

REFERENCES