

That Which Keeps on Giving - A Case of Class IV Diffuse Proliferative Lupus Nephritis in a Hispanic Woman with underlying Systemic Lupus Erythematosus

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Background: Glomerulonephritis is the primary cause of morbidity and mortality of systemic lupus erythematosus (SLE). Lupus nephritis is characterized by immune complex deposition in the mesangium leading to complement activation and hypocomplementemia. Studies show that up to 60% of adults with lupus develop renal involvement and it has been well established that Hispanic patients show poorer outcomes than Caucasians despite advances in treatment. Preserved kidney function with new-onset proteinuria should raise clinical suspicion for acute lupus nephritis. Further evaluation with a kidney biopsy is paramount in establishing a diagnosis, helping to define treatment strategy, and determining response to treatment.

Case Presentation:

A 41-yo-Hispanic-Woman with a PMH SLE without previous renal involvement, secondary Sjogren's, hypertension, heart failure and cirrhosis presented to the ER with a worsening SOB, difficulty swallowing, and anasarca over two weeks. On evaluation, the patient was hypertensive, tachypneic, had positive JVD, wheezing in lung bases, +1 pitting edema in the lower extremities and skin hyperpigmentation on the face, neck, and upper extremities. Laboratory studies revealed leukocytosis of 11.1 th/uL, Hgb 10 gm/dL, platelets 192 th/uL, Cr 0.9 mg/dL, BUN 17 mg/dL, bicarbonate 19 mmo/L, sedimentation rate 94 mm/hr, and CRP 4.5 mg/L. A urinalysis was performed, which showed 3+ proteinuria with hematuria with a subsequent protein to creatinine ratio demonstrating 2,000 mg/gm. The workup for nephritic range proteinuria revealed an ANA 10U, anti-dsDNA 2.7 IU/mL, negative ANCA, anti-cardiolipin Ab IgM <12 MPL, anti-cardiolipin Ab IgG 14 GPL, and non-reactive HIV, Hepatitis C, and Hepatitis B panels. Despite negative glomerulonephritis workup including anti-dsDNA antibody, a kidney biopsy was pursued and revealed class IV diffuse proliferative lupus nephritis with a component of thrombotic microangiopathy.

Conclusion:

Kidney biopsies are imperative when establishing a cause of new-onset proteinuria in a patient with a history of SLE. The goal of treatment is induction with immunosuppressive agents to reduce kidney inflammation promptly and prevent flares, decreasing the long-term risk of renal failure. Despite early recognition strategies and advances in treatment, Hispanic patients are likely to be diagnosed with more severe disease at presentation, specifically with class IV or V lupus nephritis. Subsequently, these patients are more likely to develop chronic renal failure compared to Caucasian patients. This case highlights the importance of screening urinalysis for proteinuria for early detection of renal involvement in patients with SLE.

References:

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