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On the Cover

What is the Diagnosis?

A 74-year-old man with hypertension and type 2 diabetes presented for evaluation of AKI. He denied any symptoms, and physical exam was unremarkable. Laboratory data revealed serum creatinine of 3.31 mg/dl (baseline 1.5 mg/dl). Urinalysis showed >100 isomorphic red blood/HPF. Twenty-four-hour urine protein was 0.5 g; there was negative serology workup, including serum free light chains ratio, urine and serum electrophoresis with immunofixations. Kidney biopsy showed fibrillary glomerulonephritis and moderate parenchymal fibrosis. Further imaging studies for malignancy screening were negative. The patient was diagnosed with idiopathic fibrillary glomerulonephritis and received rituximab 1000 mg every 2 weeks for a total of two doses. At 6 months follow-up, serum creatinine returned to baseline.

Image Description:

Light microscopy showed mesangial matrix expansion and thickening of the capillary walls (left image). Immunohistochemical staining for DNA-J heat-shock protein family member B9 was positive in the mesangium and along capillary walls (middle image). Electron microscopy revealed randomly arranged fibrils in the mesangium with a mean diameter of 17.3 nm (right image).

Teaching Points:

Fibrillary glomerulonephritis manifestations include hematuria, proteinuria, and AKI. The diagnosis can be confirmed by kidney biopsy with pathogenic staining for DNA-J heat-shock protein family member B9 and recognizing random fibrillar deposits in the mesangium and glomerular capillary walls, which are often 16 to 24 nm in diameter on electron microscopy. Secondary causes include malignancies, monoclonal gammopathy, autoimmune diseases, hepatitis C, and HIV. If a secondary cause is established, treatment of the underlying cause is warranted. The optimal treatment for idiopathic fibrillary glomerulonephritis in patients with abnormal kidney function or nephrotic range proteinuria is not well established. However, results from some studies support the use of rituximab.

(Images and text provided by Faten Aqeel, Johns Hopkins School of Medicine, Department of Medicine, Division of Nephrology, Baltimore, Maryland; Mohamad Hanouneh, Johns Hopkins School of Medicine, Department of Medicine, Division of Nephrology, Baltimore, Maryland, and Nephrology Center of Maryland, Baltimore, Maryland; Avi Z Rosenberg, Department of Pathology, Johns Hopkins Medical Institutions, Baltimore, Maryland; Bernard G. Jaar, Johns Hopkins School of Medicine, Department of Medicine, Division of Nephrology, Baltimore, Maryland, and Nephrology Center of Maryland, Baltimore, Maryland and Johns Hopkins Bloomberg School of Public Health, Department of Epidemiology, Baltimore, Maryland and Johns Hopkins Medical Institutions, Welch Center for Prevention, Epidemiology, and Clinical Research, Baltimore, Maryland)