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On the Cover

What’s the diagnosis? A 43-year-old man with a history of human immunodeficiency virus presented to the hospital with abdominal pain and weakness. He was not on Highly Active Antiretroviral Therapy (HAART). He had acute kidney injury with a serum creatinine of 2.2 mg/dl without prior history of chronic kidney disease. His initial urinalysis was significant for 30 milligrams per deciliter of proteinuria, 3-5 red blood cells per high powered field and 20-50 white blood cells per high powered field. A chest computed tomography scan revealed a cavitary lung mass. He was persistently febrile. Lumbar puncture was performed and was positive for Cryptococcus antigen. Amphotericin was started for treatment of cryptococcal meningitis and presumed pulmonary involvement. The patient decompensated and was intubated for hypoxic respiratory failure. Renal function continued to worsen and a biopsy was performed. He was started on renal replacement therapy. Renal biopsy showed severe tubulointerstitial injury. Organisms consistent with Cryptococcus were present in the glomeruli (Figure 1), interstitium and tubules. The renal parenchyma was markedly distorted, making it difficult to identify glomeruli. Electron microscopy revealed no subepithelial, intramembranous, subendothelial, or mesangial deposits. Slides stained positive with a special mucicarmine stain (Figure 2). A mucicarmine stain is generally used to identify organisms with polysaccharides in the cell wall, characteristic of Cryptococcus neoformans.

He had no history of antecedent exposure to non-steroidal anti-inflammatory medications, beta lactam antibiotics or sulfonamides. He had no history of systemic lupus erythematosus or sarcoidosis. Human immunodeficiency virus associated nephropathy (HIVAN) was considered but the patient did not have the nephrotic syndrome. No electron dense deposits were present on electron microscopy to suggest human immunodeficiency virus immune complex mediated glomerulonephritis (HIVICK). The patient was receiving amphotericin, a well described nephrotoxin, but due to the presence of acute kidney injury at initial presentation coupled with the glomerular and tubulointerstitial changes, his renal injury was attributed to cryptococcal infection.

The patient continued on renal replacement therapy with no recovery of renal function. He died related to complications from disseminated cryptococcal infection two weeks after presentation.

Cryptococcus can lead to meningitis, especially in immunocompromised patients. Renal involvement is rare. To our knowledge, these findings have not previously been reported. (Images and text provided by Sandy Gibson MD, Mid Atlantic Nephrology Associates, Richmond, Virginia and Nadia Yousef MD, Todd Gehr MD and Jason Kidd MD, Virginia Commonwealth University School of Medicine, Richmond, Virginia.)