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Kevin E. Meyers, Helen Liapis, and Mohamed G. Atta

Erratum

829 Correction

 eJournal club provides a timely and interactive electronic journal club experience by offering a forum in which CJASN readers have the opportunity to converse with the featured study authors. Visit ejc.cjasn.org to learn more.

On the Cover

What's the diagnosis? A 56-year-old man with type-2 diabetes mellitus developed malaise, fever, altered sensorium, jaundice and AKI. The patient recalled a tick bite 4-weeks prior while hiking. The patient was febrile, tachypneic and oligo-anuric (<100 ml/24 hours) despite >3 liters of crystalloid. He was jaundiced with lung crackles and mild hepatomegaly. Multiple laboratory abnormalities included hyponatremia, anion gap metabolic acidosis, increased serum creatinine, indirect hyperbilirubinemia, anemia, and thrombocytopenia (with elevated LDH and depressed haptoglobin). RBC smear demonstrated 36% parasitemia. IgM and IgG for *Babesia microti* were positive. Chest roentgenogram demonstrated diffuse bilateral interstitial edema. Automated urinalysis demonstrated a specific gravity of 1.014, pH 5.5, large blood, 3+ protein, and 1+ leukocyte esterase. Protein: creatinine ratio was 2.5 mg/mg creatinine. Urine sediment revealed multiple muddy brown granular casts and large macrophages with multiple inclusions (upper left and right panels). Urine was sent for Papanicolaou stain, which confirmed the cells as macrophages with numerous RBC fragments. Giemsa stain of the urine demonstrated *Babesia* inclusions in the RBC cytoplasm (lower left panel). Macrophages were actively phagocytosing RBCs (lower right panel). The patient was treated with RBC exchange transfusions and plasmapheresis, and combination anti-microbial agents. Despite this, he remained oligo-anuric and underwent hemodialysis. The parasite burden decreased to undetectable within ten days of plasmapheresis and anti-microbial therapy. Despite this, the patient remained dialysis-dependent at 3 weeks and underwent a kidney biopsy. Acute interstitial nephritis characterized by lymphocytes, macrophages, plasma cells and rare small granuloma was seen. Isolated macrophages, containing red blood cell fragments, were seen in the tubular lumen and macrophages were identified in the interstitium using CD68-KP1 immune histochemistry stain. In this case, examination of the urine sediment provided early insight into inflammatory kidney involvement by the parasite. Giant urinary macrophages with RBC fragments were noted on microscopy of the spun sediment (upper panels), which led to demonstration of macrophages actively phagocytosing parasitized RBCs (lower right panel). *Babesia* ring forms within urinary RBCs confirmed the presence of parasitized RBCs within the urine (lower left panel). As macrophages participate in surveillance and phagocytosis of cellular debris and pathogens in the kidney parenchyma and play a role in innate immunity and chemokine and cytokine-induced signaling, they ultimately help eradicate microbes within the kidney. It is likely that the immune reaction responsible for the granulomatous AIN in our case was initially generated by *Babesia*. (Images and text provided by Randy L. Luciano, MD, and Mark A. Perazella, MD Yale University School of Medicine, New Haven, Connecticut)