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What's the diagnosis?

Case description:

A 69-year-old man with cirrhosis, CAD with heart failure (preserved EF), aortic stenosis/aortic insufficiency, hypertension, abdominal aortic aneurysm, and stage 3B CKD of unclear etiology, Gilbert's disease, and chronic macrocytic pancytopenia (bone marrow biopsy revealed non-caseating granulomas with negative staining for micro-organisms and cancer cells) developed acute kidney injury (AKI) during hospitalization for workup of progressive weakness. Prior to the increase in serum creatinine, the patient was exposed to piperacillin-tazobactam for fever, which was subsequently diagnosed as influenza. The patient had transient hypotension but nephrotoxic medications were not administered prior to AKI. Exam revealed normal vital signs, mild hypervolemia, and absence of rash. Electrolytes were normal while serum creatinine was 3.2 mg/dL. Corrected serum calcium was 11.5 mg/dL. Ultrasound of the kidneys revealed 11.4 cm right kidney with mild echogenicity a few simple cysts and 11.7 cm left kidney with mild echogenicity. No hydronephrosis or stones were seen. Urinalysis revealed the following: SG 1.006, pH 8.0, 1+ protein, 2+ blood, 1+ leukocyte esterase. Urine microscopy revealed isomorphic erythrocytes 5-10/high-power field and leukocytes 5-10/high-power field but no casts or crystals. A kidney biopsy was undertaken for AKI, which revealed a trifecta of findings.

Image Description:

Image 1: The left panel reveals a diffuse inflammatory infiltrate consisting of lymphocytes, and macrophages along with non-caseating granulomas with multi-nucleated giant cells (Hematoxylin and Eosin stain). Stains for various micro-organisms and a diagnosis of sarcoidosis with kidney involvement was made.

Image 2: The middle panel demonstrates calcium-phosphate crystal deposition within tubules or nephrocalcinosis (Hematoxylin and Eosin stain). These crystals are not birefringent when polarized and stain positively with the von Kossa stain.

Image 3: The right panel shows biconcave cholesterol crystal clefts within the arterioles and glomerular capillaries (Hematoxylin and Eosin stain). The clefts are actually footprints of the cholesterol crystals, which are removed during the fixative process.

Teaching Points:

This case demonstrates the various processes that can affect the kidneys and lead to both acute and CKD. Sarcoidosis likely played a role in causing the underlying CKD and may have worsened with further infiltration of the kidney with some contribution from hypercalcemia. An elevated ACE level was also noted in the patient, making this case consistent with an atypical presentation of sarcoidosis with primarily bone marrow and kidney involvement. The nephrocalcinosis noted on biopsy likely developed as a result hypercalcemia and hypercalciuria from underlying sarcoidosis. The cholesterol crystal clefts in the arterioles and glomerular capillaries likely reflect atheroemboli spontaneously released from the abdominal aortic aneurysm, which subsequently lodge in the vasculature of the kidneys. Atheroembolic disease of the kidneys, which is now relatively rare, can present with acute, subacute or CKD along with other systemic manifestations (central nervous system, eye, gastrointestinal tract, feet/toes, skin, etc.) from atheroemboli to multiple organs. The patient was treated with prednisone 60 mg/d and hypercalcemia resolved and kidney function improved to serum creatinine 1.6 mg/dL over the next several weeks.

(Images and text provided by Naomi Shin, MD, Yale University School of Medicine; Gilbert W. Moeckel, MD, PhD, Department of Pathology, Yale University School of Medicine; and Mark A. Perazella, MD, Section of Nephrology, Yale University School of Medicine.)