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AKI in a Patient with Cerebral Toxoplasmosis

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Clinical Images in Nephrology and Dialysis

Case Description

A 59-year-old man, on mycophenolate mofetil and prednisone for long-standing polymyositis, presented to the emergency department with a 2-day history of flashes in his left eye. His vital signs and physical examination were unremarkable. Brain magnetic resonance imaging revealed multiple rim-enhancing lesions with perilesional edema. An extensive workup, including cerebrospinal fluid analysis, was unremarkable except for positive serum toxoplasma IgG antibody level. Mycophenolate mofetil was discontinued. Oral sulfadiazine (6 g/d) and pyrimethamine (75 mg/d) were initiated for suspected cerebral toxoplasmosis. After 2 days, the patient developed sudden onset flank pain and gross hematuria.

His serum creatinine level acutely increased to 2.0 from 0.6 mg/dl (reference range 0.6–1.2 md/dl). Abdominal computed tomography scan revealed a stone in the inferior pole of the right kidney (Figure 1A, white arrow) and left-sided hydroureteronephrosis (Figure 1A, red arrow) due to another at the left vesicoureteral junction (Figure 1B, white arrow). The patient denied a history of kidney stones. Cystoscopy revealed an orange-colored stone (Figure 1C) with floating crystalline debris. A percutaneous nephrostomy tube was placed after unsuccessful left ureteral stenting. Spectroscopic analysis of the extracted stone confirmed acetylated 2-sulfanilamidopyrimidine compound. Sulfadiazine was replaced with clindamycin and urine was alkalinized to pH 7.5. At 3-month follow-up, serum creatinine returned to baseline.

Drug-induced renal calculi represent 1%–2% of the total number of stones analyzed in specialized laboratories (1). Sulfa medications were first implicated in nephrolithiasis after their introduction in the early 1940s (2). Sulfadiazine with pyrimethamine is the preferred drug regimen in the treatment of central nervous system toxoplasmosis. The dosage of sulfadiazine is typically between 4 and 8 g/d, with the potential risk of formation of insoluble calculi in the urinary tract, often bilaterally (3). Predisposing factors for the development of calculi include daily administration of the medication, low urinary pH, low urine volume, and urinary stasis. The critical urine pH that maintains crystal solubility is reported being 7.15 (4). Examination of urine sediment under the microscope has been a useful tool to assess the morphology of crystals. Crystals can be detected even in the absence of AKI. The most common morphology of sulfonamide crystals includes needle-shaped crystals, rosettes, or an appearance resembling "shocks of wheat." Infrared spectroscopy is useful to characterize the calcification (5). Aggressive hydration and, if needed, use of sodium bicarbonate to alkalinize urine are recommended when taking sulfonamides to minimize the risk of crystal formation and subsequent AKI.

Teaching Points

- The low solubility of sulfadiazine and its metabolites in the acidic urine, along with high urine concentration, predispose for crystal formation that has been described to resemble shocks of wheat.
- A high index of suspicion for medication-induced nephrolithiasis merits consideration to enable quick diagnosis and timely drug discontinuation to

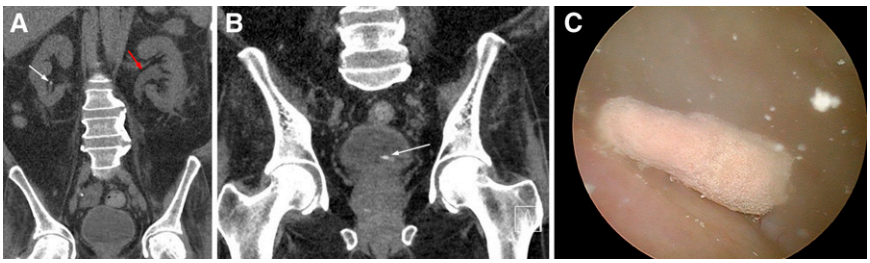

Figure 1. | **Abdominal computed tomography and cystoscopy images.** Abdominal computed tomography scan revealed (A) a stone in the inferior pole of the right kidney (white arrow) and left-sided hydroureteronephrosis (red arrow) due to (B) another at the left vesicoureteral junction (white arrow). (C) Cystoscopy revealed an orange-colored stone with floating crystalline debris.

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prevent unnecessary morbidity and health care utilization.

Author Contributions

S. Kuppachi reviewed and edited the manuscript; and J. Patel wrote the original draft of the manuscript.

Disclosures

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