AKI in a Patient with Urinary Tract Infection, Urinary Crystals, and a Bladder Stone

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KIDNEY360 3: 790–792, 2022. doi: https://doi.org/10.34067/KID.0006112021

Case Description
A 56-year-old man with a history of diabetes mellitus type 2 for 13 years, depression, and neurogenic bladder who underwent suprapubic catheter placement 2 years ago presented to the hospital with altered mental status and lower abdominal pain for 2 days. His initial vital signs revealed a BP of 90/60 mm Hg, heart rate of 110 beats per minute, respiratory rate of 22 breaths per minute, and temperature of 37.3°F. Physical exam demonstrated suprapubic tenderness. Laboratory studies revealed the following: white blood cells of 14,690 per μl (3000–11,200 per μl), hemoglobin of 11.0 g/dl (12.5–15.9 g/dl), serum creatinine of 3.71 mg/dl (baseline creatinine 1.6 mg/dl), BUN of 104 mg/dl (6–23 mg/dl), serum bicarbonate of 10 mmol/L (22–29 mmol/L), serum anion gap of 25 mmol/L (7–16 mmol/L), whole blood lactate of 5 mmol/L (0.5–1.7 mmol/L), and serum osmolality of 335 mOsm/kg (275–295 mOsm/kg). Urinalysis revealed a pH of 9.0 with hematuria and pyuria. Examination of the urine sediment by light microscopy demonstrated cofilin lid-shaped crystals consistent with triple phosphate crystals (Figure 1A). Under polarized light, the crystals were positively birefringent; crystals were blue when parallel to the light, and yellow when perpendicular to the light (Figure 1B). Computed tomography scan of the abdomen without contrast showed a thickened bladder wall with a large, calcified bladder stone (Figure 2), with no evidence of calculi in the kidneys or ureters. The patient was started on intravenous fluids and antibiotics covering urease-producing bacteria, and a 4.5-cm, yellow-tan, calcified stone was removed emergently by cystoscopy. Urine culture later grew Klebsiella oxytoca and calculus analysis demonstrated a magnesium ammonium phosphate component. On day 3 of hospitalization, the patient clinically improved, with serum creatinine returning to his baseline, and he was discharged on a 14-day course of cefdinir.

Discussion
Triple phosphate crystals and stones are composed of magnesium ammonium phosphate and are typically seen in the setting of bacteria that produce ammonia, resulting in increased urine pH. Some bacteria, like Proteus mirabilis, K. pneumoniae, or Corynebacterium species produce urease which breaks down urinary urea into carbon dioxide and ammonia. The latter combines with water to produce ammonium hydroxide. The net result is increased ammonium in an alkaline urine. Triple phosphate stones are more common in women and in patients with high risk of developing urinary tract infections, such as those with a neurogenic bladder, urinary diversion, or indwelling Foley catheter (1,2). Patients usually present with urinary tract infection symptoms and/or urosepsis. Triple phosphate stones should be expected in patients with alkaline urine (urine pH >8.0) who have triple phosphate crystals in their urine sediment. Further imaging with computed tomography or renal ultrasound is needed to identify the calculus. Sending urine culture and starting empirical antibiotics covering urease-producing bacteria is crucial. However, a surgical approach is usually required for infectious source control. Repeat imaging in 3 months post-treatment is recommended to assess for the presence of any residual stone fragments (3). Most experts advise 3–6 months of prophylactic antibiotics for all patients with triple phosphate stones. Urease inhibitors are not commonly used due to their significant, but reversible, side effects, including gastrointestinal symptoms, bone marrow suppression, hemolytic anemia, and liver dysfunction (4).

Teaching Points
- Triple phosphate crystals and stones are formed by the crystallization of magnesium, ammonium, and phosphate in alkaline urine as a result of urease-producing bacteria.
- Diagnosis is made by visualization of positively birefringent, cofilin lid-shaped crystals on urine microscopy, abdominopelvic imaging, and analysis of stone composition.
- Urine culture and prompt initiation of antibiotics targeting urease-producing bacteria is crucial to prevent worsening infection, although definitive treatment often includes surgical management.

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Disclosures

M. Hanouneh reports serving on a speakers bureau for AstraZeneca. B.G. Jaar reports receiving honoraria from the American Board of Internal Medicine–Nephrology; serving as a scientific advisor for, or member of, the American Board of Internal Medicine, BMC Nephrology, BMC Medicine, CJASN, and National Kidney

Figure 1. Urine sediment by light microscopy. (A) The arrows point to coffin lid–shaped crystals consistent with triple phosphate crystals. (B) Urine sediment by light microscopy under polarized light. The arrows point to positive birefringent triple phosphate crystals; crystals were blue when parallel to the light and yellow when perpendicular to the light.

Figure 2. Computed tomography scan of abdomen without contrast. The arrows point to a thickened bladder wall with a large, calcified bladder stone.
Foundation; and having other interests in/relationships with UpToDate (receiving royalties as an author). The remaining author has nothing to disclose.

Funding
None.

Acknowledgments
Informed consent was obtained from the patient.

Author Contributions
M. Hanouneh provided supervision; M. Hanouneh and B.G. Jaar reviewed and edited the manuscript; and C.F. Schretlen wrote the original draft.

References

Received: September 22, 2021 Accepted: March 3, 2022