

Treatment of the Infected Stone

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KEYWORDS

• Struvite • Calcium carbonate apatite • Staghorn • Obstructive pyelonephritis • Kidney stones

• Urinary tract infection

KEY POINTS

- Infection stones result from urease-producing bacteria and are struvite and/or calcium carbonate apatite in composition.
- Optimal management of infection stones is complete stone removal, and failure to achieve complete stone clearance results in a high recurrence rate.
- Obstructive pyelonephritis is a urologic emergency and can result in urosepsis and death.
- Emergent decompression with retrograde ureteral stent placement or percutaneous nephrostomy tube (PCNT) placement and broad-spectrum antibiotics are imperative to treating patients with obstructive pyelonephritis.

INTRODUCTION

An infected kidney stone can refer to stones that form because of urinary tract infections (UTIs) with urease-producing bacteria, secondarily infected stones of any composition, or stones obstructing the urinary tract leading to pyelonephritis. Most commonly, kidney stones that form secondary to urease-producing bacteria are composed of struvite or calcium carbonate apatite, and presentation is frequently incidental and generally nonemergent. Secondarily infected metabolic stones have also been described. These stones are frequently colonized with non-ureaseproducing bacteria and often have discordant culture results compared with the lower urinary tract. Obstructive pyelonephritis secondary to urinary tract calculi is considered a urologic emergency, and immediate treatment is indicated to avoid serious complications, including urosepsis and death. Given the difference in pathophysiology and treatment approach, these entities are discussed separately.

Infection Stones

Infection stones are most commonly composed of magnesium ammonium phosphate (ie, struvite) and/or calcium carbonate apatite. These stones result from chronic infections with ureaseproducing bacterial pathogens and frequently form large branched stones known as staghorn calculi. The incidence of infection stones has overall decreased during the last 30 years, likely due to improved medical care. They are more common in women (10%-11% vs 4% in men) and elderly patients.^{1,2} The pathogenesis of struvite and calcium carbonate apatite stone formation is presented in Fig. 1.^{3,4} Urease from bacteria splits urea into ammonia and carbon dioxide. Ammonia reacts with water to become ammonium and hydroxide ions, which creates an alkaline milieu. In this alkaline environment, the ammonium combines with magnesium, phosphate, and water to create magnesium ammonium phosphate stones. The carbon dioxide eventually breaks down to carbonate, which combines with calcium and phosphate to

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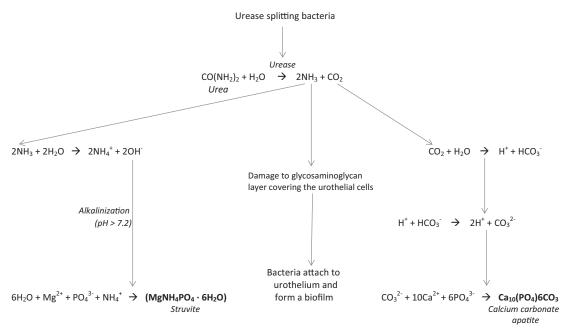


Fig. 1. Pathogenesis of infection stones. Urease from bacteria splits urea (ie, carbamide) into ammonia and carbon dioxide. The ammonia combines with water to produce ammonium and hydroxide. Hydroxide results in an alkalosis of the urine leading to the formation of magnesium ammonium phosphate (ie, struvite). The ammonia also damages the glycosaminoglycan layer causing urothelial damage, allowing the bacteria to attach to the urothelium and form a biofilm. Carbon dioxide complexes with water to form bicarbonate and then carbonate. Carbonate combines with calcium and phosphate forming calcium carbonate apatite.

form calcium carbonate apatite stones. The most common urease-producing bacterial pathogens are Proteus spp, Klebsiella spp, Providencia spp, Morganella morganni, and Staphylococcus aureus.^{5,6} Infection stones are commonly asymptomatic or present with UTIs, flank or abdominal pain, fevers, gross hematuria, or less commonly with sepsis or renal insufficiency.⁷ Patients with indwelling catheters, neurogenic bladder, and urinary diversion have the highest risk of developing infection stones due to chronic bacterial colonization. The natural history of these stones is associated with progressive morbidity and mortality, with the 10-year mortality rate reported at 28% with nonsurgical management versus 7% with surgical treatment.8

Secondarily infected stones, which are nonstruvite and non-calcium carbonate apatite stones associated with infection, have been described.⁹ In a series of 125 patients undergoing percutaneous nephrolithotomy (PCNL), de Cógáin and colleagues⁹ found that 24 (23%) of 106 patients with nonstruvite stones had positive stone cultures. A history of neurogenic bladder was associated with positive stone culture in both patients with infected nonstruvite and struvite stones in this series. Nonurease-producing bacteria, including *Escherichia coli* and *Enterococcus* spp, are the predominant organisms colonizing these metabolic stones.9,10 Whether these stones form and become secondarily infected or whether these stones result from a nidus of infection that propagates stone formation is unclear. Theories for how bacteria could be a nidus for nonstruvite and non-calcium carbonate apatite stones include kidney cell injury and inflammation potentiating crystal retention, alteration of the microenvironment by bacterial metabolic activity, or biofilm that acts as a matrix for stone growth.⁹ In general, sending a sample for stone culture should be considered for all patients undergoing PCNL to help target antibiotic therapy in the event of a postoperative infection. Patients should make appropriate dietary modifications and receive specific medical therapy based on metabolic studies to prevent recurrent urolithiasis. Further studies are necessary to elucidate the exact role and clinical significance of bacteria in these stones.

Obstructive Pyelonephritis

Pyelonephritis is an infection of the kidney with typical presentation including but not limited to fever, flank pain, and irritative lower urinary tract symptoms. Obstructive pyelonephritis is a complicated UTI and considered a urologic emergency because of the significant risk of morbidity and Download English Version:

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