



Urinary Tract Infection and Urolithiasis: A Narrative Review of Pathophysiological Links and Clinical Implications

Dong Sup Lee

Department of Urology, St. Vincent's Hospital, The Catholic University of Korea, Suwon, Korea

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Corresponding author:

Dong Sup Lee

Department of Urology, St. Vincent's Hospital, The Catholic University of Korea, 93, Jungbudaero-ro, Paldal-gu, Suwon 16247, Korea

Email: bbangbbangi@naver.com,

lds@catholic.ac.kr

<https://orcid.org/0000-0003-0862-3205>

Urinary tract infection (UTI) and urolithiasis are closely related clinical entities that frequently coexist and influence each other's pathogenesis and clinical course. Infection-related stones, particularly struvite and carbonate apatite stones, have long been recognized as consequences of urease-producing bacterial infections. However, recent evidence suggests that the role of microorganisms—especially *Escherichia coli*—in stone disease may extend beyond traditional infection stones; in particular, biofilm formation by microorganisms may contribute to the formation and growth of calcium-based stones. This review summarizes current knowledge on the pathophysiological mechanisms linking UTI and urolithiasis, with a focus on the biochemical processes underlying infection-related stone formation, the characteristics of different stone compositions, and emerging concepts regarding bacterial involvement in metabolic stone disease. A better understanding of the interplay between UTI and urolithiasis may improve preventive strategies, guide antimicrobial and surgical decision-making, and ultimately reduce recurrence rates. This review highlights the need for an integrated clinical approach that considers both infectious and metabolic factors in patients with urinary stone disease.

Keywords: Urinary tract infections, Urolithiasis, Struvite

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HIGHLIGHTS

In the urinary tract, urease-producing microorganisms such as *Proteus mirabilis* have long been recognized as the principal drivers of struvite stone formation. However, *Escherichia coli* has been most frequently identified in struvite stones. Although complete removal of infectious stone is crucial whenever feasible, long-term strategies aimed at lowering urinary pH and reducing opportunistic microbial colonization in the urine are also required.

INTRODUCTION

The term 'apatite' is usually used when we call urinary stones, actually urinary stones composed of calcium and phosphate, and it came from Greek meaning 'cheating'

because the nature including shape and color differ from each other according to combined chemical compositions such as hydroxy group, carbonate, and so on. Chemical (or mineralogical) types of human urolithiasis are composed of calcium oxalate monohydrate (whewell-



lite), calcium oxalate dihydrate (weddellite), calcium phosphate (brushite), carbonated calcium phosphate (carbapatite), ammonium magnesium phosphate (struvite), uric acid, cystine and so on. Among those, struvite, named after Heinrich Christian Gottfried von Struve, and to the best of knowledge, its development results from bacteria producing urease. Struvite should not be considered synonymous with all infectious stones, but it could be representative for infectious stone; As Khusid et al. [1] emphasized, it is helpful for physicians to communicate each other by differentiating 'infectious stone' from 'infected stone'; an infection (infectious) stone should refer to a case where the infection precedes the formation of the stone, whereas an infected stone refers to a case where an existing stone becomes infected incidentally. This narrative review aims to summarize the current understanding of the pathophysiological relationship between urinary tract infection (UTI) and urolithiasis, with particular emphasis on infectious stone such as struvite and mixed struvite, and to discuss their clinical implications for perioperative infection control and postoperative prevention strategies.

METHODS

This narrative review was conducted using a nonsystematic literature search of PubMed and major international guideline documents, including publications from urological associations and infectious disease societies. English-language articles focusing on infectious urolithiasis, struvite stones, mixed struvite stones, perioperative infection management, and strategies for postoperative prevention were considered.

Priority was given to clinically relevant original studies, comprehensive review articles, and guideline-based evidence. When appropriate, seminal studies that contributed to the understanding of the pathophysiology and clinical management of infection-related stones were also included. The selected literature was qualitatively synthesized to summarize current concepts regarding the pathophysiology, clinical characteristics, periopera-

tive considerations, and preventive strategies for infectious urolithiasis.

MECHANISMS OF DEVELOPMENT OF INFECTIOUS UROLITHIASIS

Urease producing *Enterobacteriaceae* such as *Proteus* spp., *Klebsiella* spp. *Pseudomonas* spp., *Morganella* spp., *Citrobacter* spp., and so on [2] can hydrolyze urea into ammonia (NH_3) where ammonia transforms into ammonium (NH_4^+) and hydroxide (OH^-) in response with water, subsequently raising pH [3], which facilitates the bonding of ammonium with magnesium and phosphate as 1:1:1 ratio eventually creating struvite. Ureolysis also can create carbonate (CO_3^{2-}) which can be mixed with calcium and phosphate turning into carbapatite. Struvite or carbapatite can emerge everywhere of urinary tract in cases that the above conditions are met, which differs from the biomineralization of brushite, weddellite and whewellite that theoretically initiates stone formers to be visualized from amorphous spherules at the duct of Belini [4].

Regarding bacteria isolated from infectious stones, *Escherichia coli* has been reported as the most frequently identified organism among various bacterial species [5]. Parkhomenko et al. [2] reported that non-urease-producing bacteria accounted for 47% or more of struvite-containing stones, with *E. coli* identified in 33.3% of cases. Furthermore, Nevo et al. [6] observed that *E. coli* was more commonly associated with mixed struvite stones, defined as stones containing a calcium oxalate component with less than 25% struvite, whereas *Proteus* spp. and *Klebsiella* spp. predominated in pure struvite stones.

Despite these observations, plausible mechanistic explanations for struvite formation involving non-urease-producing microorganisms have not been clearly established. In particular, no definitive mechanism has been demonstrated by which *E. coli* directly generates ammonium from urea to promote struvite formation. A recent study proposed that mixed struvite stones (calci-

um oxalate stones combined with struvite) may reflect sequential infections, in which an initial infection by urease-producing bacteria is followed by *E. coli* colonization. This hypothesis was supported by differences in supersaturation levels of calcium oxalate and calcium phosphate between pure struvite and mixed struvite stones, suggesting a potential role of metabolic biomineralization in the calcium oxalate component of mixed stones [7].

In this context, Taabodi et al. [8] reported that *E. coli* can utilize ammonium to produce urea, which may act as a negative feedback mechanism for ammonium accumulation. This finding suggests that *E. coli* is unlikely to generate ammonium directly from urea; instead, the isolation of *E. coli* from ammonium-rich stones may reflect secondary infection following an initial urease-producing bacterial infection. Taken together, these findings support an association between *E. coli* and mixed struvite stones but do not establish a causal role.

Separately, Mikami et al. [9] demonstrated that *E. coli* can produce ammonia under specific experimental conditions, such as growth in Luria–Bertani medium. In that study, ammonia production was enhanced in a *ki-*vd**-overexpressing strain via the citric acid cycle, while ammonia assimilation into glutamine was reduced in a *glnA*-deleted strain. These experimental findings raise the possibility that ammonia generation by *E. coli* may occur under amino-acid-rich conditions, potentially facilitating struvite formation when urinary pH is relatively high and phosphate and magnesium are available as substrates; however, whether such mechanisms operate *in vivo* within the urinary tract remains uncertain.

Biomineralization processes involving weddellite and whewellite have been widely described, with gradients in Gibbs free energy and calcium-to-phosphate ratios contributing to diagenetic phase transitions [4]. Obstructive urological conditions, such as hydronephrosis, hydrocalycosis, or calyceal diverticula, have also been associated with calcium-based stone formation, accompanied by increased urinary calcium and decreased citrate levels [10,11]. Nevertheless, the relationship between infectious stones and calcium oxalate

components remains incompletely understood. Although some studies have suggested that the presence of *E. coli* may be associated with calcium oxalate crystallization *in vivo* [12,13], no definitive microarchitectural or chemical mechanisms have been identified.

Therefore, based on the currently available evidence, it is not possible to determine whether the calcium oxalate component observed in infectious stones, particularly mixed struvite stones, arises primarily from host-driven biomineralization processes or from bacterial mineral-regulating effects. At present, the most plausible finding is an association between mixed struvite stones containing calcium oxalate and *E. coli* infection, which should be interpreted as hypothesis-generating rather than evidence of causation.

TREATMENT STRATEGY OF INFECTIOUS UROLITHIASIS

1. Preoperative Management of Complicated UTI With Urolithiasis

The prevalence of febrile UTI has been reported as only 25% among struvite-related stones [14]. If febrile UTI was notified in patients with urolithiasis without any information about microorganisms at presentation, blood and urine culture should be performed immediately before administration of broad-spectrum antibiotics which was known at least as effective as 80% according to regional antibiotics monitoring system [15]. Then, percutaneous nephrostomy (PCN) catheter or ureteric catheter should be placed if any evidence of delayed drainage such as hydronephrosis was identified. Xu et al. [16] preferred PCN placement in febrile patients with urolithiasis because ureteric stent placement had higher failure rate than PCN insertion, and might increase renal pelvis pressure during and after the procedure which could allow reflux of infection sources into blood, aggravating the inflammatory response. If patients have ureter stone only, and infection control was completely done, antegrade fluid irrigation with natural dropping of normal saline from PCN to open channel of ureteroscope can

extremely reduce operation time during semirigid ureteroscopic lithotripsy [17]. However, ureteric stent placement may be beneficial in patients with bleeding tendency, or in those with small stones in multiple calyces who are candidates for retrograde intrarenal surgery (RIRS) rather than percutaneous nephrolithotomy because preoperative ureteric stent placement facilitates the advancement of ureteral access sheath during RIRS, sometimes enables to choose wider one [18]. After control of acute infection with PCN insertion, antegrade ureteric stent placement would be better for those patients before several days prior to RIRS, in cases of previously noted narrow ureter, kinked ureteral course or large prostate [19].

According to Korean Society of Endourology and Robotics, empiric antibiotics are recommended in urolithiasis before endoscopic surgery but not before shock wave lithotripsy once infection is not suspicious [20]. However, the duration of usage of antibiotics for asymptomatic patients with bacteriuria remained unclear. According to recent report, the duration of antibiotic administration did not affect postoperative UTI in asymptomatic patients with positive urine culture where groups with prophylactic use of antibiotics regardless of duration was significantly reduced postoperative UTI than a group without antibiotics though [21]. Therefore, all patients with highly suspicious infection, empirical antibiotics should be used at least for several days before operation, based on culture report if available.

2. Intraoperative Management of Infectious Urolithiasis

Over the past decade, the engineering advancements in urological equipment, such as flexible ureteroscopes and related disposable devices, have been remarkable. From a urological endoscopic perspective, the consideration of postoperative UTIs can be summarized with two key words; that are 'intrarenal pressure' and 'residual stone (or stone free).' Consensus of upper limit of intrarenal pressure during endoscopic surgery has been suggested as 40 cmH₂O [22]. To maintain intrarenal pres-

sure under this point, a ureteral access sheath is useful, as it can distribute the pressure load through the sufficient space between the scope and the sheath, minimizing pyelovenous backflow of infectious sources, as mentioned above.

Martínez-Piñeiro et al. [23] found that the vast majority of regrowing stone was arisen from residual stone after surgical treatment of struvite. Regrowing of struvite could be observed even within 6 months [24]. Moreover, antibiotics penetration into the matrix of the stone is limited because of its nature as biofilm scaffold [25]. Therefore, surgeons should do their best to ensure no residual stones are left behind.

3. Postoperative Management of Infectious Urolithiasis

Now that citrate has a nature of dissolution for calcium oxalate crystals, it has been already used as a preventive medicine for recurrent calcium oxalate stones [26,27]. However, citrate could be used as energy source in some bacteria such as *Klebsiella pneumoniae*; even in *E. coli* in the presence of sufficient co-substrates such as glucose, lactose, pyruvate, or glycerol, in which it could be fermented anaerobically in the presence of citrate transporter and citrate lyase [28]. It is important for physicians including endourologists to know that (1) *E. coli* is versatile in terms of aerobic and anerobic respiration [29], (2) more hypoxic, more distal to renal cortex [30], (3) some *E. coli* could utilize citrate following acquired adaptation [31]. Therefore, citrate should be considered as a preventive medicine only with non-high pH, without recently proven struvite/carbapatite-related stones and without microorganisms proven by urine culture.

If infectious stone was confirmed by stone analysis, then close monitoring is mandatory using urine culture, urinary pH, and 24-hour urine collection. According to recent report, half of patients would have same microorganisms even at 6 months after stone removal, and sustained detection of urinary microorganisms was an important risk factor for early stone regrowth, emphasizing oral immunization which could reduce sustained col-

onization of microorganisms in urinary tract [24]. Basically, struvite and related stones have relatively high urinary pH; so, using methionine maybe beneficial [32]. According to European Association of Urology guideline [33], methionine is recommended 200–500 mg 1–3 times per a day. Acidifying agents such as acetohydroxamic acid and ammonium chloride were also indicative in cases of infectious stone, however these drugs cannot be available in many regions including south Korea. Twenty-four hour urine collection may offer beneficial information to patients who undertook surgical removal of infectious stone. However, concentration of each cation or anion increased with time after surgical removal of the stone, therefore the test should be performed at least a few months later after operation [34]. Although there is lack of study using drugs lowering calcium excretion or other mineral excretion in patients with infectious stones, conducting this kind of study would be beneficial to elucidate a mechanism for mixed struvite.

SPECIFIC CONDITIONS

1. Hyperparathyroidism

Hyperparathyroidism is a strong predisposing condition for calcium phosphate stone, named brushite. Although hyperparathyroidism is not a classic infectious-stone condition, it is clinically relevant because carbonate apatite may overlap with infection-related phosphate stone phenotypes and may complicate etiologic interpretation. Daudon et al. [35] found that the prevalence of carbapatite (66.9%) is twice more than brushite in hyperparathyroidism. Therefore, we should not be prejudiced that phosphorus leak in hyperparathyroidism always is a preceding condition to brushite. Vice versa, as soon as recognizing carbapatite, screening for hyperparathyroidism would be clever. In a study, the relative risk for renal stone episode was 40 in primary hyperparathyroidism and the risk gradually decreased with times over 10 years after parathyroidectomy [36].

2. Patients With Neurogenic Deficit and Infectious Stones

Increased incidence of UTI in patients with neurological deficit has been widely recognized. UTI and catheterization in neurogenic bladder patients have been suggested to be responsible for development of bladder stone in which carbapatite and struvite were revealed as most common stone components [37]. In addition, bacteriuria is a risk factor for urolithiasis including renal stone as it is obviously recognized with previous studies [38]. In clinical practice, patients with neurological deficit usually have cardio-pulmonary morbidities such as atelectasis, aspiration, arrhythmia and so on [39], minimizing of the frequency for operation in their lives is of great importance. Therefore, in patients with neurological deficit, not only bladder stone, but renal stone also should be monitored regularly with appropriate preventive medicines. Although the advantage of low-dose prophylactic antibiotics has been shown, the method may be applied limitedly and cautiously, regarding selection of antimicrobial resistance [40].

INTEGRATIVE STRATEGIES FOR UNDERSTANDING INFECTIOUS UROLITHIASIS

To provide a structured overview of the diverse experimental findings and clinical observations discussed in this narrative review, the key concepts and proposed mechanisms linking UTI and urolithiasis are summarized in Table 1. This synthesis highlights the multifactorial nature of infection-related stone formation, encompassing microbial characteristics, urinary biochemical conditions, and host-related factors. Importantly, it also distinguishes between mechanisms supported by established evidence and those that remain associative or hypothesis-generating, thereby offering a strategic framework for interpretation and for identifying priorities for future research.

Table 1. Summary of preventive and monitoring strategies for infectious stones

Options	Comments
Avoidance of dehydration	Lowering urinary supersaturation of minerals
Removal of all residual stones, if possible	Infectious stones grow rapidly around a nidus and may act as a barrier to antibiotic penetration.
Checking parathyroid hormone	Especially when carbonate apatite is identified on stone analysis.
Low-dose prophylactic antibiotics	Should be used cautiously and limited due to the risk of antimicrobial resistance.
Oral immunotherapy	May decrease the postoperative detection rate of microorganisms, especially <i>Escherichia coli</i>
Lowering urine pH	L-methionine, acetohydroxamic acid, ammonium chloride
Thiazide	The effect is not clearly established in infectious stone, but thiazides may reduce urinary calcium excretion and potentially contribute to reduced biomineralization of calcium-based stones.
Citrate	May not be recommended; however, it can be considered only when urine pH is appropriate (≤ 6.0), no microorganisms are detected, and there is no recent history of infectious stones.

CONCLUSION

UTI and urolithiasis are closely interconnected through complex pathophysiological mechanisms involving urease-producing organisms, urinary biochemical alterations, and biomineralization processes. Struvite stones and mixed infection stones represent clinically important entities that often require both surgical management and careful infection control. Understanding the microbiological and biochemical mechanisms underlying infection-related stones is essential for optimizing perioperative management and preventing recurrence. In addition to complete stone removal, appropriate antibiotic therapy, metabolic evaluation, and long-term preventive strategies play key roles in patient outcomes. Future research should further clarify the mechanisms of mixed infection stones and develop evidence-based strategies for preventing recurrence, particularly in patients with recurrent infection or complex stone disease.

NOTES

- **Author Contribution:** All contributions to this paper were made by the single author (DSL).
- **ORCID**
Dong Sup Lee: 0000-0003-0862-3205

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