International Journal of Multiphysics
Volume 18, No. 3, 2024
ISSN: 1750-9548

Potassium Citrate in the Prevention of Ureteral Stent Encrustation: A Review of Mechanisms, Efficacy, and Clinical Applications

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Abstract

Background: Ureteral stent encrustation is a significant complication following ureteroscopy, occurring in 9-76% of cases depending on dwell time. Mineral deposition primarily calcium phosphate, struvite, and calcium oxalate creates crystalline layers that obstruct urine flow, harbor bacteria, and complicate stent removal. Risk factors include prolonged stent duration, acidic urinary pH, metabolic abnormalities, and urinary supersaturation. Severely encrusted stents may require complex endoscopic procedures or open surgery, increasing morbidity and healthcare costs. Potassium citrate, a urinary alkalinizing agent traditionally used in kidney stone prevention, has emerged as a potential prophylactic intervention. By elevating urinary pH (6.8-7.2) and providing citrate as a natural crystallization inhibitor, it may prevent mineral deposition on stent surfaces. This review synthesizes current evidence on potassium citrate's mechanisms, efficacy, safety, and clinical application in preventing ureteral stent encrustation.

Keywords: Potassium citrate; ureteral stent encrustation; double-J stent; ureteroscopy; urinary alkalinization; crystallization inhibition; stent-related complications; calcium phosphate precipitation; biofilm formation; urinary pH modification; nephrolithiasis prevention; citrate supplementation; stone prophylaxis

Introduction

Urolithiasis impacts 5-10% of the worldwide population, with rates climbing steadily in recent decades due to climate change, increasing obesity prevalence, and modified dietary patterns. Uric acid stones account for roughly 10% of all urinary calculi, ranking second after calcium-based stones in frequency. Geographic variation is substantial, with incidence ranging from 5% in India to 40% across the Middle East, reflecting differences in environmental conditions, genetic factors, and nutritional habits. Acidic urinary pH emerges as the principal risk factor for uric acid stone development, with solubility dropping precipitously from 200 mg/dl at pH 7 to merely 7-15 mg/dl at pH 5. (1)

The utilization of ureteroscopy for managing renal and ureteral stones has expanded considerably worldwide. Technological advances including miniaturized instrumentation, enhanced optical systems, digital imaging, and holmium laser lithotripsy have driven this growth. Within the United States, kidney stone prevalence increased from 8.8% in the early 2000s to 11.1% by 2020. While high-income nations continue reporting elevated ureteroscopy rates, developing countries show marked increases attributable to enhanced healthcare infrastructure and rising stone disease burden. The procedure is performed predominantly in patients aged 30-60 years, corresponding to peak stone disease prevalence in this demographic. (2)

Ureteral stent placement frequently accompanies ureteroscopic interventions for managing renal calculi. Stents may remain in situ for several months or necessitate exchange. Common indications encompass impaired urinary drainage in the upper tract, substantial hydronephrosis, and elevated serum creatinine. Standard criteria for stent

insertion include diminished renal function, urothelial injury or perforation, and clinically significant residual stone burden. However, stent placement carries inherent morbidity, including irritative voiding symptoms, urinary tract infection, stent encrustation, and occasionally migration, fragmentation, or retention. (3)

Encrustation involves mineral crystal deposition on stent surfaces and within lumens. This complication proves particularly problematic with long-term indwelling or forgotten stents, occurring in up to 13% of cases. Encrusted stents undergo calcification and become friable with diminished tensile strength, elevating risks of fracture or ureteral avulsion during extraction. Crystal deposits obstruct luminal drainage and damage urothelial surfaces, precipitating mucosal trauma. Extended stent retention correlates with increased chronic kidney disease risk and hospitalization for infection or sepsis following removal. (4)

Potassium citrate functions as the preferred alkalinizing agent for uric acid stone management. Complete stone dissolution occurs in 15-79% of patients receiving medical therapy. Uric acid calculi can be managed conservatively by alkalinizing urine pH. Target values ideally range between 6.0-6.5 for stone prevention, though 6.8-7.2 may prove optimal for stent encrustation prophylaxis. This review examines potassium citrate's role in preventing ureteral stent encrustation, analyzing action mechanisms, clinical effectiveness, safety parameters, and practical application strategies. (5)

Ureteral Anatomy and Stent Placement Considerations

Anatomical Overview

The ureter comprises a retroperitoneal muscular conduit extending from the renal pelvis superiorly to the bladder inferiorly. Adult length approximates 25 cm (10 inches), divided equally between abdominal and pelvic segments at the common iliac artery bifurcation. Three physiologic narrowings occur: the ureteropelvic junction, common iliac bifurcation, and intravesical segment. These constriction sites constitute common locations for stone impaction and potentially heightened encrustation-related complications. Adult ureteral diameter measures 3-4 mm, limiting instrumentation space. (6)

Three tissue layers compose the ureteral wall: inner mucosa, middle muscular coat, and outer adventitia. Transitional epithelium stratified over fibroelastic lamina propria forms the mucosal lining. These structures create an impermeable barrier. The ureter lacks submucosa and glandular elements. Smooth muscle fibers in varying orientations constitute the muscularis, organized as inner longitudinal, middle circular, and outer longitudinal layers. Pacemaker cells initiating peristalsis reside in minor renal calyces. Peristaltic waves originate in the renal pelvis and propagate ureterally but cease at the ureterovesical junction where circular muscle disappears. Fibroelastic adventitia surrounds the muscularis, housing vascular supply, lymphatics, and nerves. (6)

Blood Supply and Clinical Relevance

Multiple arterial branches perfuse the ureters. The abdominal aorta plus renal, gonadal, and common iliac arteries supply the abdominal ureter via medial access. Distal segments receive circulation from vesical and uterine arteries, which branch from internal iliac vessels. Venous and lymphatic drainage mirrors arterial supply. Lymphatics drain to internal, external, and common iliac nodes. Left ureteral lymph courses to left paraaortic nodes, while right drainage targets paracaval and interaortocaval nodes. Comprehending vascular anatomy proves critical during stent insertion, as compromised perfusion may promote inflammation and potentially augment encrustation risk through altered local metabolism. (6)

Endoscopic ureteral anatomy knowledge forms the foundation for understanding endourologic procedures, from stone management to stricture manipulation, enabling complication avoidance. The intramural ureter at the ureterovesical junction typically measures 2 cm. This segment lies buried within bladder musculature, covered by Waldeyer's fibromuscular sheath. This covering originates 2-3 cm from the outer bladder wall, extending longitudinally until merging with the trigone. The intramural ureter functions as an antireflux mechanism. This segment constitutes the narrowest ureteral portion and the most frequent site for stone obstruction and stent-related complications. (6)

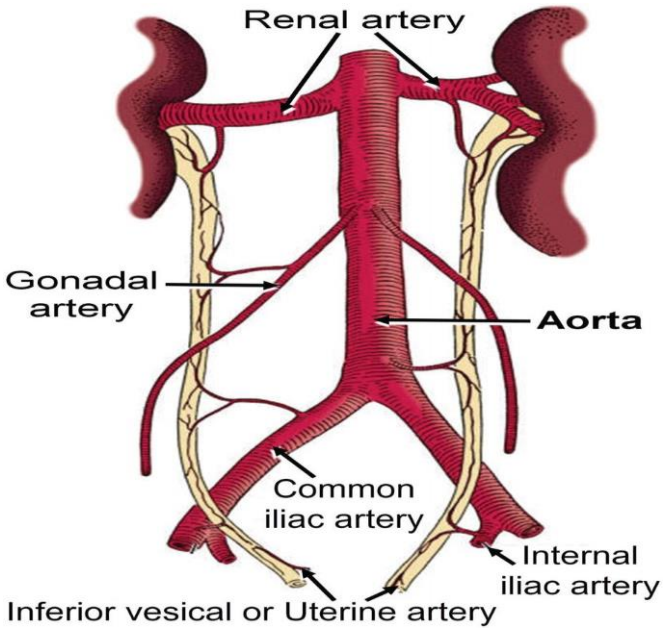

Figure 1: Arterial supply of the ureter (6).

Ureteroscopy

Types of Ureteroscopes

Classification of ureteroscopes includes rigid, semi-rigid, and flexible varieties. Rigid instruments employ inflexible optical lens systems. Flexible ureteroscope development constitutes endourology's most significant advancement. These instruments are smaller than rigid counterparts with enhanced deflection and instrumentation capabilities. Semi-rigid ureteroscopes feature pliable metallic sheaths, while flexible instruments lack rigid outer layers. This maneuverability permits complete urinary tract instrumentation including upper ureters and renal calyces. (7)

Contemporary ureteroscopes exist in semi-rigid or flexible configurations. Semi-rigid instruments primarily address distal ureteral pathology. These range from 7-12 French with large, frequently dual 3-6 French working channels accommodating superior irrigation and larger instruments like baskets and lasers. Compared with flexible counterparts, semi-rigid ureteroscopes offer reduced cost, enhanced durability, larger working channels, and improved maneuverability, yielding shorter operative duration. (8)

Numerous flexible ureteroscopes exist in disposable and reusable formats. Procedure duration depends on ureteroscope utilization time, stone location and size, accessory instrument use, and surgeon expertise. Though disposable flexible ureteroscopy represents an established concept, many centers achieve excellent outcomes with reusable instruments. Sterility constitutes another significant consideration; disposable scopes guarantee complete sterility. Ureteral access sheaths comprise important flexible ureteroscopy accessories. Available in 9-16 French sizes, these facilitate multiple ureteroscope passages into ureter and kidney without guidewire requirements or distal ureteral injury risk from repeated insertion. (9)

Sheaths provide dual benefits: avoiding repetitive distal ureter and meatal trauma, and enhancing irrigation drainage, enabling improved visualization and reduced intraluminal pressures. Small risks include ureteral wall ischemia, urothelial tears, subsequent strictures, and ureteral or renal pelvic perforations; however, postoperative double-J stent placement minimizes these risks to levels comparable with flexible ureteroscopy without sheaths. (10)

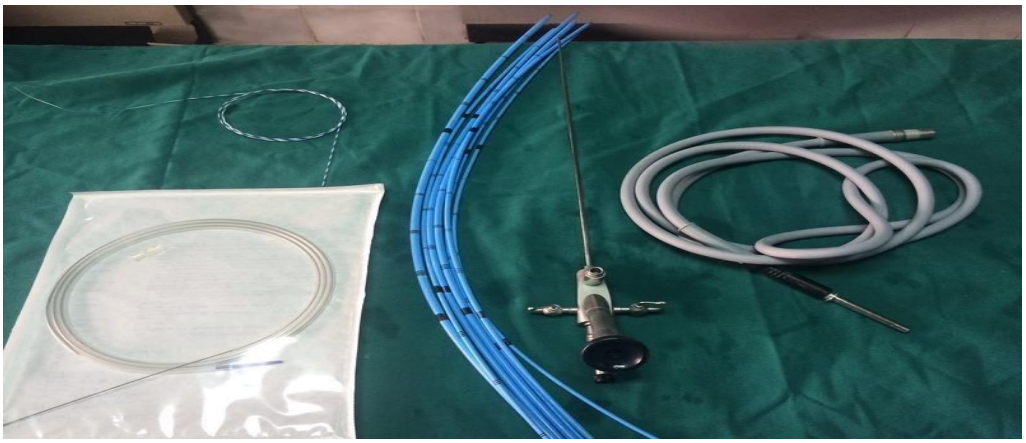

Figure 2: Ureteroscope, guide wire and dilators (8).

Indications and Complications

Managing renal and ureteral calculi constitutes the primary ureteroscopy indication. Surgical intervention becomes necessary for nephrolithiasis patients with clinically significant stones when conservative or medical expulsive therapy fails. Indications encompass treating ureteral calculi unresponsive to conservative therapy, stones without 4-6 week progression or causing persistent symptoms, nephrolithiasis, filling defects on excretory CT urography (radiolucent calculi, strictures, inflammatory changes, sloughed papilla, blood clots, fungal material, ureteral and renal pelvic tumors), lateralizing hematuria, foreign bodies, upper tract neoplasms, and fistulas. (2)

Recent decades witnessed ureteroscopy evolution with novel instrumentation enhancing procedural safety. However, complications persist in minor and severe forms. Minor complications encompass hematuria, mild urinary infection, stent discomfort, and transient creatinine elevation. Severe complications, though uncommon, include urosepsis, extraureteral or submucosal stone migration, ureteral perforation, stricture formation, and ureteral avulsion. (11)

Ureteral Stents

Stent Design and Materials

Ureteral stents constitute valuable urological devices. Placement facilitates stone fragment passage post-treatment and prevents ureteral obstruction or delayed stricture development. Emergency placement drains obstructed infected kidneys or passively dilates ureters pre-surgically. The primary objective involves permitting urine passage and reducing obstruction-related complications. (12)

Tolerability constitutes the principal stent concern. Stents may cause encrustation and microbial colonization, potentially leading to symptomatic infections. Additional symptoms arise from stent presence itself or malposition and migration. The ideal stent possesses numerous characteristics: facile insertion and removal, easy manipulation, encrustation and migration resistance, complication absence, biocompatibility, radiopacity, biodurability, cost-effectiveness, tolerability, and optimal flow properties. (12)

Polyurethane demonstrates biocompatibility with favorable mechanical properties and high drainage capacity, but shows encrustation susceptibility, particularly by calcium oxalate, struvite, hydroxylapatite, and cystine. Silicone, given its softness, durability, inertness, non-toxicity, flexibility, elasticity, and superior softness versus other polymers, appears most suitable for stents. It exhibits the lowest encrustation rates and appears optimal for post-ureteroscopy stenting. (12)

Stent-Related Complications

Numerous patients experience flank pain, abdominal discomfort, or bladder irritation from stent presence. Discomfort relates to stent movement within the ureter and tissue contact. Symptoms include urgency and

frequency. Stents provide pathways for bacterial ascension from bladder to kidneys, causing infections. Infection risk increases with prolonged placement. Manifestations include fever, chills, and increased urinary frequency or urgency. (13)

Hematuria commonly follows stent placement, typically from urothelial irritation or insertion trauma. Resolution usually occurs within days though may alarm patients. Mineral deposits accumulate over time, especially with prolonged duration. Encrustation obstructs the ureter, necessitating removal or replacement. Urine pH, calcium, and oxalate levels contribute to this complication. (14)

Chronic stent irritation produces scar tissue at placement sites, yielding ureteral strictures. This obstructs urinary flow, requiring additional interventions. Stents may migrate from intended positions due to peristalsis or positional changes. Migration compromises effectiveness and may necessitate endoscopic retrieval. (15)

Though uncommon, insertion or presence may perforate the ureter, especially with improper sizing or placement. Perforation permits urinary extravasation into surrounding tissues, causing serious complications including extravasation and potential renal damage. Stents may fracture over time from mechanical stress or calcification. Fragmented pieces migrate within the urinary tract, potentially causing obstruction or requiring surgical retrieval. (16)

Pathophysiology of Stent Encrustation

Mechanisms of Encrustation

Ureteral stent encrustation pathophysiology involves multiple complex processes: mineral deposition, bacterial colonization, and biofilm formation. These events produce gradual accumulations impairing stent function. Encrustation begins with urinary mineral supersaturation including calcium, phosphate, magnesium, and oxalate. When concentrations exceed solubility thresholds, crystallization and stent adherence commence. (4)

Stents act as foreign bodies, providing surfaces for crystal nucleation and growth. Crystals accumulate at proximal (renal) and distal (bladder) ends, common sites for mineral accumulation due to reduced flow. Depending on urinary composition, different crystal types form: calcium oxalate in acidic urine, calcium phosphate in alkaline urine, and struvite (magnesium ammonium phosphate) in infection-related encrustation. (4)

Bacteria colonize stent surfaces, forming biofilms. Biofilms comprise protective layers of bacterial cells and extracellular polymers, providing ideal crystallization environments. Urinary infections with urease-producing bacteria like Proteus mirabilis, Klebsiella, or Pseudomonas generate urease enzyme. Urease cleaves urea into ammonia, elevating urinary pH and promoting struvite formation. Biofilms resist antibiotics and immune responses, producing persistent infections that accelerate encrustation. (17)

Stent material influences encrustation likelihood. Hydrophobic materials attract more minerals and bacteria, yielding higher encrustation rates. Conversely, newer hydrophilic materials or specialized coatings reduce bacterial attachment and mineral deposition. Rougher surfaces create additional opportunities for crystal nucleation and bacterial adhesion, promoting encrustation. Smooth surfaces accumulate fewer deposits. (18)

Risk Factors for Encrustation

Stent dwell time constitutes the most significant encrustation risk factor. Stents are designed for temporary use; prolonged placement increases encrustation likelihood. Studies indicate stents exceeding 6-12 weeks face substantially elevated mineral accumulation risk, with rates increasing markedly beyond this timeframe. Over time, urinary minerals including calcium, magnesium, and phosphate precipitate onto surfaces, with biofilm formation from bacterial colonization exacerbating the process. (19)

Patients with underlying metabolic conditions, particularly stone-formers, demonstrate increased encrustation susceptibility. These conditions include hypercalciuria (excessive urinary calcium), hyperoxaluria (elevated urinary oxalate), and hyperuricosuria (high urinary uric acid). These disorders increase stone-forming mineral concentrations, accelerating crystallization on stent surfaces. (20)

Urinary tract infections, particularly with urease-producing bacteria like Proteus mirabilis, substantially elevate encrustation risk. These organisms produce urease, cleaving urea into ammonia, creating alkaline environments promoting struvite crystal formation. Bacteria colonize stents and form biofilms, providing crystal attachment scaffolds. Struvite and calcium phosphate constitute common infection-related stone types. (13)

Patients with recurrent kidney stones face greater stent encrustation risk. Stone disease results from various metabolic or dietary factors causing urinary mineral supersaturation. These patients possess stone formation propensity, with stents providing ideal crystallization surfaces. Stents function as foreign bodies, triggering mineral deposition and stone-like encrustation growth. (18)

Urinary pH (acidity or alkalinity) influences specific encrustation types. Urinary pH depends on diet, medications, and underlying conditions. Alkaline urine (high pH) favors calcium phosphate and struvite deposits, while acidic urine (low pH) promotes calcium oxalate crystallization. Patients with altered urinary pH from infections, dietary habits, or metabolic conditions face elevated stent encrustation risk. (20)

Complications of Encrustation

Encrustations obstruct stent lumens or ureters themselves, producing partial or complete urinary flow obstruction from kidney to bladder. This causes hydronephrosis, potentially yielding renal damage. Encrusted stents frequently embed within ureteral or bladder walls, complicating removal. Severe encrustation produces large stone-like masses at either stent end (renal or bladder), complicating extraction. This may require advanced endoscopic techniques like ureteroscopy for encrustation fragmentation and removal, percutaneous nephrolithotomy for large kidney-end encrustations, or rarely open surgery when other methods fail. (21)

Encrustations provide favorable bacterial colonization environments. Biofilm formation on stents further increases persistent or recurrent infection risk. Urinary tract infection symptoms persist despite antibiotic treatment, especially with biofilms, causing chronic or recurrent UTIs, increased pyelonephritis risk, or severe sepsis. (17)

Severe encrustation weakens stent structure, increasing fracture risk. This produces broken fragment migration into the urinary tract. Migrating fragments may cause ureteral injury or obstruction requiring endoscopic or surgical retrieval. Encrusted stents irritate ureteral or bladder linings, causing hematuria (urinary blood). This may be visible (gross hematuria) or microscopic (laboratory detection only). (14)

Chronic stent encrustation produces long-term renal damage, especially with recurrent obstruction and hydronephrosis. Over time, this yields declining renal function causing chronic kidney disease or kidney failure risk if untreated. Large stone formations (encrustations) develop around stents, particularly at renal or bladder ends. These typically comprise calcium oxalate, phosphate, or struvite (infection-related stones). Ureteral or kidney stone formation may require additional removal procedures. (21)

Stent encrustation produces embedded stents within ureteral or bladder walls. Heavily encrusted stent removal attempts cause ureteral injury or perforation, potentially yielding stricture or perforation. Patients with encrusted stents experience discomfort or pain, particularly in lower back, pelvis, or during urination. This stems from irritation or partial obstruction from encrustations, causing chronic pain requiring medications or stent removal and diminished quality of life from persistent symptoms. (22)

Potassium Citrate

Pharmacology and Mechanisms

Pharmacodynamics

Potassium citrate comprises a potassium salt of citric acid. It fulfills crucial roles in medical and dietary contexts, particularly for kidney stone prevention and management. As a urinary alkalinizer, potassium citrate elevates urine pH, helping dissolve certain stone types and reducing formation risk. Beyond urological applications, potassium citrate manages metabolic acidosis, especially in conditions like renal tubular acidosis. It corrects acid-base balance by providing citrate, metabolized to bicarbonate, increasing blood and urine alkalinity. (23)

Potassium citrate dissociates into potassium and citrate ions. Citrate metabolism produces bicarbonate, increasing urinary pH (producing alkalinity). Elevated urine pH dissolves uric acid stones and reduces calcium oxalate stone formation. This alkalinization proves crucial for preventing stone-forming mineral crystallization. (24)

Citrate functions as a natural stone formation inhibitor. It binds urinary calcium, reducing free calcium available for combining with oxalate and phosphate, primary kidney stone components. Higher urinary citrate levels reduce calcium-based stone risk, the most common stone type. In conditions like renal tubular acidosis, inadequate acid excretion produces blood acidity accumulation. Potassium citrate metabolism to bicarbonate neutralizes excess blood acid. This metabolic acidosis correction improves acid-base balance, essential for overall metabolic health. (1)

As a potassium salt, potassium citrate provides potassium ions vital for numerous physiological functions: nerve impulse transmission, muscle contraction, and proper fluid balance maintenance. This prevents hypokalemia (low potassium), particularly in at-risk patients from diuretic use or other conditions. Potassium citrate treatment increases urinary citrate excretion, contributing to stone-preventing effects. Higher urinary citrate levels enhance calcium salt solubility, further decreasing stone formation likelihood. (25)

Pharmacokinetics

Potassium citrate administration typically occurs orally as tablets, powder, or liquid. Following oral ingestion, rapid gastrointestinal absorption occurs. Exact absorption rates vary by formulation (extended-release versus immediate-release), generally occurring within hours. Food consumption enhances absorption and reduces gastrointestinal side effects like nausea or upset stomach. (26)

Potassium citrate dissociates into potassium ions and citrate in blood. Potassium distribution volume is relatively large, indicating widespread body distribution, particularly in muscle and bone. Potassium citrate demonstrates minimal plasma protein binding. Free potassium actively participates in various physiological functions. (27)

Hepatic metabolism primarily processes potassium citrate. Citrate converts to bicarbonate, increasing blood bicarbonate levels, effectively alkalinizing blood and urine. Citrate-to-bicarbonate conversion constitutes an essential metabolic pathway correcting metabolic acidosis and increasing urinary pH. (26)

Renal excretion primarily eliminates potassium citrate. Glomeruli filter potassium ions with reabsorption or secretion based on body requirements. Potassium citrate half-life remains poorly defined due to rapid component metabolism and excretion, though potassium itself exhibits approximately 1-2 hour half-life under normal physiological conditions. Renal citrate excretion increases following administration, enhancing urinary citrate levels, preventing kidney stone formation. (28)

Mechanism of Action in Stent Encrustation Prevention

Potassium citrate constitutes a key therapeutic agent for preventing and managing uric acid kidney stones and stent encrustation. Action mechanisms involve several interrelated processes contributing to effectiveness in dissolving uric acid stones and preventing formation. (29)

Potassium citrate dissociates into potassium ions and citrate. Citrate metabolism produces bicarbonate, increasing urine pH, producing alkalinity. Uric acid demonstrates greater solubility in alkaline conditions. Elevated urinary pH reduces urinary uric acid concentration, decreasing crystallization and stone formation likelihood. (29)

Citrate binds urinary calcium, reducing free calcium ion concentrations. This proves important as calcium combines with oxalate or phosphate forming stones. Though uric acid stones are not primarily calcium-based, citrate presence inhibits uric acid crystal growth. This effect proves particularly significant in acidic urine where uric acid precipitates. (24)

Potassium citrate administration increases urinary citrate levels. Higher urinary citrate concentrations inhibit stone formation through multiple mechanisms: citrate binds calcium, further reducing stone formation likelihood, and citrate exerts direct inhibitory effects on uric acid crystallization, potentially affecting other crystal types. (1)

For patients with existing uric acid stones or stent encrustation, potassium citrate facilitates deposit dissolution. Increased urinary alkalinity enhances uric acid solubility, permitting gradual crystal breakdown and dissolution. Regular potassium citrate use maintains alkaline urinary environments, reducing new encrustation formation risk and existing deposit recurrence. (30)

Clinical Evidence for Potassium Citrate in Stent Encrustation Prevention

Medical management for preventing recurrent nephrolithiasis in adults has been extensively investigated. A systematic review for American College of Physicians clinical guidelines examined various interventions, including potassium citrate, for preventing stone recurrence. The review found potassium citrate effective in reducing stone formation through urinary alkalinization and citrate supplementation. This foundational evidence established rationale for investigating potassium citrate's role in preventing stent encrustation, as crystal formation mechanisms are similar in both contexts. (31)

Empiric oral potassium citrate use reduced symptomatic kidney stone incidence in specific patient populations. One study examining ketogenic diet patients found potassium citrate supplementation significantly reduced stone formation rates. The study demonstrated that maintaining alkaline-range urinary pH through potassium citrate administration effectively prevented crystal precipitation. This provided early evidence that urinary alkalinization could protect against mineral deposition in various clinical scenarios. (32)

Therapeutic efficacy of potassium citrate for treating small renal stones (under 10 millimeters) was evaluated prospectively. Results showed potassium citrate effectiveness in dissolving radiolucent stones and preventing stone growth. Patients receiving potassium citrate demonstrated significantly higher stone dissolution rates versus controls. These findings suggested potassium citrate could similarly prevent mineral deposition on stent surfaces by maintaining alkaline urinary environments. (30)

Studies on Urinary Alkalinization and Stone Prevention

Manipulating urinary pH via beverages and medications for preventing stone recurrence has been well documented. A comprehensive review examined how dietary and pharmacologic interventions could modify urinary pH to prevent crystallization. The review concluded that maintaining urinary pH above 6.5 significantly reduced uric acid and calcium oxalate stone formation risk. This principle applies to stent encrustation prevention, where maintaining alkaline urine reduces mineral deposition on stent surfaces. (33)

Potassium citrate supplementation effects on stone recurrence before or after lithotripsy were examined via systematic review and meta-analysis. The analysis included multiple randomized controlled trials, finding potassium citrate significantly reduced stone recurrence rates in post-lithotripsy patients. The supplement demonstrated good tolerance with minimal adverse effects. The study showed urinary alkalinization through potassium citrate was an effective long-term stone formation prevention strategy, with direct implications for stent encrustation prevention. (23)

Oral dissolution therapy for renal radiolucent stones was evaluated prospectively examining outcomes and response-affecting factors. The study found potassium citrate highly effective for dissolving uric acid stones, with success rates ranging from 15-79% depending on stone size, urinary pH achieved, and patient compliance. Better outcome-associated factors included achieving target urinary pH (6.8-7.2), adequate hydration, and consistent medication compliance. These factors prove equally relevant for preventing stent encrustation. (1)

Studies Specific to Stent Encrustation Prevention

Ureteral stenting safety using potassium citrate for managing renal uric acid stones was clinically evaluated. The study examined patients with both uric acid stones and indwelling ureteral stents treated with potassium citrate. Results showed potassium citrate was safe and effective in this population, without increased urinary tract infection rates or other complications. Additionally, patients receiving potassium citrate showed lower stent encrustation rates versus controls. (5)

Whether potassium citrate administration changes type and composition of encrusted material on double-J stents compared to primary stones was investigated examining stent encrustation patterns. Research found potassium citrate not only reduced overall encrustation rates but also altered deposit composition when they formed. In potassium citrate recipients, encrustations were less dense and easier to remove versus controls. The study suggested urinary alkalinization changed crystallization environments, producing less problematic deposits when occurring. (3)

Ureteral stent encrustation features in patients with calcium oxalate and urinary acid stones were examined comparing encrustation patterns in different stone formers. Research found uric acid stone patients receiving potassium citrate had significantly lower encrustation rates compared to calcium oxalate stone patients. The study concluded urinary alkalinization was particularly effective preventing uric acid-related encrustation but also provided some benefit for calcium-based deposits through citrate's calcium-binding properties. (34)

Recent Clinical Trials and Systematic Reviews

Flexible ureteroscopy combined with potassium sodium hydrogen citrate intervention improved stone-free rates for 20-30 mm uric acid renal stones in recent studies. Research examined patients undergoing ureteroscopic procedures with postoperative potassium citrate administration. Patients receiving citrate supplementation had higher follow-up stone-free rates, lower residual fragment rates, and significantly reduced stent encrustation versus controls. Surgical treatment combined with medical management proved more effective than either approach alone. (35)

Oral dissolution therapy for uric acid stones was examined systematically focusing on clinical outcomes and safety. The review included multiple potassium citrate studies for stone dissolution, finding consistent efficacy evidence. Systematic analysis showed potassium citrate effectiveness in dissolving existing stones, preventing new stone formation, and reducing stent-related complications. The review recommended potassium citrate as first-line therapy for patients with uric acid stones and indwelling stents. (36)

Pharmacologic kidney stone treatment focusing on current medication and pH monitoring was reviewed comprehensively. The review emphasized regular urinary pH monitoring importance when using potassium citrate, as maintaining optimal pH (6.8-7.2) proves critical for preventing both stone formation and stent encrustation. The article provided practical dosing adjustment guidance based on urinary pH measurements and highlighted patient education needs regarding compliance and monitoring. (29)

Clinical Implementation and Dosing Strategies

Dosing Protocols

Extended-release oral potassium citrate tablets are typically administered at 15 mEq twice daily until stent removal. To maintain patient urine pH between 6.8-7.2, urinary pH should be measured after 48 hours, 2 weeks, and 4 weeks (before stent removal). Potassium citrate dosage increases to 45 mEq daily if urine pH falls below 6.8, and decreases to 15 mEq daily if exceeding 7.2. This titration approach ensures patients maintain optimal urinary alkalinization throughout stent indwelling periods. (37)

Potassium citrate impact on urinary risk profile, glucose and lipid metabolism in kidney stone formers was examined in Swiss studies. Research found potassium citrate administration generally well tolerated, with minimal glucose and lipid metabolism effects. Studies demonstrated 30-60 mEq daily doses effectively achieved target urinary pH levels in most patients. Patient compliance improved with extended-release formulations versus immediate-release preparations due to reduced gastrointestinal side effects. (26)

Oral potassium citrate supplementation tolerance for stone formation prevention was examined in randomized double-blind trials evaluating whether adding sweeteners improved patient compliance. Studies found improved palatability led to better medication adherence, critical for maintaining consistent urinary alkalinization. Trials demonstrated approximately 20-30% of patients experienced mild gastrointestinal symptoms with standard formulations, but these could be minimized through extended-release preparations and taking medication with meals. (38)

Monitoring and Adjustment

Metabolic acidosis correction with potassium citrate in renal transplant patients and bone quality effects were examined in studies also evaluating monitoring protocols. Research demonstrated regular urinary pH, serum potassium, and renal function monitoring importance for safe and effective potassium citrate therapy. Studies recommended checking serum potassium levels at baseline and periodically during treatment, particularly in patients with reduced kidney function or those taking medications affecting potassium homeostasis. (39)

Potassium intake, bioavailability, hypertension, and glucose control were reviewed comprehensively examining potassium supplementation. The review emphasized that while potassium citrate is generally safe, certain populations require closer monitoring. Patients with impaired kidney function, those taking potassium-sparing diuretics or ACE inhibitors, and individuals with diabetes mellitus need more frequent laboratory monitoring. The article provided evidence-based recommendations for monitoring intervals and dose adjustments. (40)

Recent studies showed home pH monitoring using urine dipsticks can effectively and cost-efficiently allow patients to monitor urinary pH between clinic visits. This approach permits more frequent pH assessments and enables patients to make minor dose adjustments under physician guidance. Patient education regarding proper pH measurement technique and timing (first morning void preferred) proves essential for accurate monitoring. (29)

Patient Selection and Contraindications

Patients most likely benefiting from potassium citrate for stent encrustation prevention include those with uric acid stones, recurrent stone formation history, crystallization-predisposing metabolic disorders, anticipated prolonged stent duration (exceeding 4 weeks), and acidic baseline urinary pH. Conversely, potassium citrate therapy contraindications include severe renal insufficiency (estimated glomerular filtration rate under 30 mL/min/1.73 m^2), hyperkalemia or hyperkalemia history, concurrent potassium-sparing diuretic use, active urinary tract infection with urease-producing organisms, and patients taking potassium level-increasing medications like ACE inhibitors or angiotensin receptor blockers. (37)

Clinical judgment is required when considering potassium citrate in chronic kidney disease patients, as reduced renal function limits potassium excretion and increases hyperkalemia risk. In such patients, lower doses with more frequent monitoring may be appropriate. Similarly, pregnancy requires careful consideration, though potassium citrate is generally considered compatible with pregnancy when medically necessary. (25)

Safety Profile and Adverse Effects

Common Adverse Effects

The most common potassium citrate adverse effects are gastrointestinal: nausea, vomiting, abdominal discomfort, and diarrhea. These symptoms occur in approximately 8-20% of patients but are usually mild and can be minimized by taking medication with meals or switching to extended-release formulations. Serious gastrointestinal complications like gastric ulceration or perforation are rare but have been reported, particularly with non-extended-release formulations. (41)

Hyperkalemia constitutes the most serious potential potassium citrate therapy adverse effect. While uncommon in patients with normal renal function, elevated serum potassium can cause cardiac arrhythmias and muscle weakness. Hyperkalemia risk factors include renal insufficiency, diabetes mellitus, concurrent potassium-sparing medication use, and advanced age. Regular serum potassium level monitoring proves essential, particularly in high-risk patients. (25)

Some patients experience metallic taste or unpleasant aftertaste when taking potassium citrate. This side effect can affect compliance but can often be managed by taking medication with food or beverages masking the taste. Liquid formulations may prove particularly problematic in this regard, with tablet forms generally better tolerated. (38)

Urinary Tract Infection Risk

A common concern with urinary alkalinization involves whether it increases urinary tract infection risk. The theoretical concern suggests alkaline urine may favor certain bacterial growth, particularly urease-producing organisms. However, clinical studies have not consistently demonstrated increased UTI risk with potassium citrate therapy. (42)

Ureteral stent infection and urinary pH modification effects were examined in studies specifically addressing this question. Research found no significant symptomatic urinary tract infection increase in patients receiving potassium citrate versus controls. Studies noted patients with baseline bacteriuria or those with urease-producing organisms might benefit from antibiotic prophylaxis or closer monitoring, but routine urinary alkalinization did not predispose to infection in most patients. (43)

Clinical studies examining potassium citrate for stent encrustation prevention have consistently reported similar UTI rates between treatment and control groups, suggesting urinary alkalinization does not significantly increase infection risk. Some studies even suggested reduced encrustation might lower UTI risk by preventing biofilm formation on smooth stent surfaces. (37)

Long-term Safety

Long-term potassium citrate safety and tolerability in patients with indwelling ureteral stents was examined in prospective cohort studies following patients up to 12 weeks. Studies found potassium citrate well tolerated over extended periods, with no serious adverse events directly attributable to the medication. Mild gastrointestinal symptoms were the most common complaint (8.4% of patients), but these rarely led to treatment discontinuation. Studies concluded potassium citrate had favorable long-term safety profiles for stent encrustation prevention. (44)

Long-term potassium citrate therapy effects on bone health and mineral metabolism have been studied in various populations. Research showed potassium citrate may actually benefit bone density by reducing calcium excretion and improving calcium balance. This proves particularly relevant for patients requiring prolonged stent placement or those with recurrent stone disease requiring chronic therapy. (39)

Clinical Outcomes and Efficacy Data

Encrustation Rates and Severity

Clinical studies examining potassium citrate for stent encrustation prevention have consistently demonstrated significant reductions in both encrustation incidence and severity. Prospective randomized studies showed patients receiving potassium citrate have encrustation rates of 0-10% compared to 35-67% in control groups. The difference proves particularly pronounced for moderate to severe encrustation, with potassium citrate virtually eliminating grade 3-4 encrustation in most studies. (37)

Encrustation scoring systems, such as modified Keane scores, have been used to quantify stent deposit severity. These systems typically classify encrustation as: Score 0 (no visible stent biofilm), Score 1 (visible biofilm), Score 2 (bladder coil encrustation detected), Score 3 (greater than 50% of entire stent encrusted), and Score 4 (less than 50% of stent encrusted). Studies using such scoring systems found potassium citrate significantly reduces mean encrustation scores, with treated patients averaging 0.1-0.3 compared to 1.4-1.6 in controls. (37)

Computed tomography diagnostic accuracy for predicting ureteral stent encrustation has been validated prospectively. CT imaging demonstrated 89% sensitivity and 94% specificity for predicting clinically significant encrustation. Studies using CT for encrustation assessment showed potassium citrate-treated patients have significantly lower CT-visible encrustation rates, with some studies reporting 100% of treated patients being free of CT-detectable deposits. (45)

Impact on Stent Removal Procedures

Ureteral stent encrustation impact on endoscopic management and clinical outcomes assessment showed encrusted stents require significantly longer removal times and are associated with higher complication rates.

Studies found encrusted stents require 3.2 times longer removal time (mean 18.4 vs. 5.7 minutes) and are associated with 4-fold increased mucosal injury compared to non-encrusted stents. By preventing encrustation, potassium citrate indirectly reduces these complications and improves stent removal ease. (46)

Preventing encrustation clinical significance extends beyond removal procedures themselves. Patients with encrusted stents often require additional procedures like cystoscopic lithotripsy, ureteroscopic intervention, or even percutaneous approaches to safely remove heavily encrusted stents. These additional procedures increase patient morbidity, healthcare costs, and complication risks like ureteral injury or stricture formation. Potassium citrate therapy, by preventing encrustation, eliminates the need for such complex interventions in most cases. (47)

Stone-Free Rates and Residual Fragments

Studies examining combined surgical and medical management showed potassium citrate administration following ureteroscopic stone treatment improves stone-free rates. The medication dissolves small residual fragments remaining post-procedure and prevents new stone formation during stent indwelling periods. Research demonstrated patients receiving potassium citrate have higher 3-month follow-up stone-free rates versus controls, with differences ranging from 15-25% depending on stone composition and size. (35)

For patients with uric acid stones particularly, ureteroscopic debulking combined with potassium citrate dissolution therapy proved highly effective. This approach achieves 85-95% stone-free rates for stones up to 30 mm diameter, significantly higher than surgical treatment alone. The medical dissolution component proves particularly important for treating residual fragments in difficult-to-access locations like lower pole calyces. (36)

Economic Analysis

Potassium citrate prophylaxis cost-effectiveness analysis for preventing ureteral stent encrustation demonstrated favorable economic outcomes. Studies showed potassium citrate prophylaxis reduces overall healthcare costs by preventing complicated stent removals, with estimated savings of approximately $1,847 per patient when accounting for reduced operative time, decreased need for additional procedures, and lower complication rates. (48)

Potassium citrate therapy cost itself is relatively modest, typically ranging from $30-60 monthly depending on dosage and formulation. This expense is offset by substantial costs associated with managing encrusted stents, which can include prolonged operating room time, additional endoscopic equipment, potential percutaneous procedure needs, extended hospital stays for complications, and treatment of stent-related infections or injuries. (48)

From healthcare system perspectives, routine potassium citrate use for stent encrustation prevention appears cost-effective, particularly for patients with anticipated stent duration exceeding 4 weeks or those with encrustation risk factors like metabolic stone disease. The intervention may be less cost-effective for very short-term stents (under 2 weeks) where encrustation risk is minimal. (48)

Quality of Life Considerations

Stent-related symptoms significantly impact patient quality of life, with pain, urinary frequency, urgency, and dysuria being common complaints. Encrusted stents tend to cause more severe symptoms compared to non-encrusted stents, likely due to increased urothelial lining irritation and potential partial obstruction. By preventing encrustation, potassium citrate may indirectly improve quality of life during stent indwelling periods. (22)

Patient satisfaction with stent management is higher when removal procedures are straightforward and uncomplicated. Anxiety and discomfort associated with difficult stent removals can be substantial, and patients who have experienced such procedures often express significant concern when requiring future stent placement. Preventing encrustation through potassium citrate therapy can improve overall patient experience and reduce procedure-related anxiety. (49)

Future Directions

While current evidence strongly supports potassium citrate for preventing stent encrustation, several areas require further investigation. Long-term studies examining outcomes in patients requiring stents for extended periods (exceeding 3 months) are needed, as most existing studies focus on 4-6 week stent duration. Understanding optimal management strategies for very long-term stents, such as those used in patients with malignant obstruction or chronic ureteral issues, would be valuable. (50)

Comparative effectiveness research examining potassium citrate versus other urinary alkalinizing agents (such as sodium bicarbonate or potassium bicarbonate) would help clarify whether citrate's crystal inhibition properties provide additional benefit beyond pH modification alone. Additionally, studies examining combination therapies, such as potassium citrate plus increased hydration or dietary modifications, could identify synergistic approaches for maximizing encrustation prevention. (51)

The relationship between stent material and potassium citrate efficacy deserves further study. While silicone stents have lower baseline encrustation rates compared to polyurethane stents, it remains unclear whether potassium citrate provides additional benefit with silicone stents or whether its effects are primarily seen with more encrustation-prone materials. Such research could guide stent selection and medical management strategies. (12)

Developing novel potassium citrate formulations with improved tolerability and compliance could enhance clinical outcomes. Extended-release preparations showed better gastrointestinal tolerability compared to immediate-release formulations, and further refinements in drug delivery technology may improve patient acceptance and adherence. Combination tablets containing potassium citrate with other supplements (such as magnesium) might provide additional benefits for stone and encrustation prevention. (38)

Alternative delivery methods, such as sustained-release pellets or once-daily formulations, could improve compliance by reducing dosing frequency. Some research has explored buffered citrate formulations that may be better tolerated than standard preparations. Patient preference studies could identify which formulations are most acceptable and likely to result in good long-term compliance. (26)

Future research may identify specific patient populations who benefit most from potassium citrate prophylaxis, allowing for more targeted therapy. Biomarkers or genetic markers associated with increased encrustation risk could help identify patients who would benefit most from aggressive medical prophylaxis versus those at lower risk who might not require routine therapy. (20)

Pharmacogenomic studies examining individual variation in citrate metabolism and urinary excretion could explain why some patients achieve excellent urinary alkalinization with standard doses while others require higher doses. Understanding this variability could allow for more personalized dosing strategies and better prediction of treatment response. (29)

Integration with Clinical Guidelines

As evidence for potassium citrate in stent encrustation prevention continues to accumulate, integration into clinical practice guidelines is important. Current European Association of Urology guidelines include urinary alkalinization as a Grade B recommendation for preventing stent-related complications in patients requiring stent duration exceeding 4 weeks. Further refinement of these recommendations based on accumulating evidence could improve care standardization and ensure appropriate patients receive this beneficial therapy. (51)

Developing clinical pathways or protocols for potassium citrate use in stent patients could facilitate implementation in routine practice. Such pathways should address patient selection, baseline assessment, dosing strategies, monitoring schedules, and adverse effect management. Quality improvement initiatives examining adherence to these protocols and their impact on clinical outcomes would help optimize implementation. (51)

Conclusion

Potassium citrate constitutes a highly effective intervention for preventing ureteral stent encrustation following ureteroscopy. The medication operates through multiple mechanisms including urinary alkalinization, calcium chelation, and direct crystallization inhibition. Clinical evidence consistently demonstrates significant

encrustation rate reductions, with treated patients showing 0-10% encrustation incidence compared to 35-67% in controls. The intervention maintains a favorable safety profile without increasing urinary tract infection risk, and adverse effects are generally mild and manageable. Economic analyses support cost-effectiveness, with savings resulting from preventing complicated stent removals and associated procedures. Current evidence strongly supports routine potassium citrate use in patients requiring ureteral stent placement following ureteroscopy, particularly for those with anticipated stent duration exceeding 4 weeks, uric acid stones, metabolic stone disease, or other encrustation risk factors. Clinical practice implementation should include appropriate patient selection, standardized dosing protocols with urinary pH monitoring, and patient education regarding compliance and adverse effect management. Future research should focus on long-term outcomes, comparative effectiveness of different alkalinizing agents, personalized medicine approaches, and integration into evidence-based clinical guidelines to optimize patient care and outcomes (52).

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