

The effect and factor analysis of potassium sodium hydrogen citrate on the formation of double J tube wall stones after ureteral stone surgery

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Abstract: Background: Double-J (DJ) ureteral stent encrustation is a frequent complication after ureteroscopic lithotripsy (URS), leading to infections, obstruction and difficult stent removal. Potassium sodium hydrogen citrate may reduce encrustation by alkalinizing urine and increasing urinary citrate excretion. **Objectives:** To evaluate the effect of potassium sodium hydrogen citrate on DJ stent encrustation and to identify biochemical, clinical and microbiological predictors following ureteral stone surgery. **Methods:** This retrospective comparative study included 110 adults undergoing URS with DJ stenting. Group A (n=55) received potassium sodium hydrogen citrate for 4 weeks, while Group B (n=55) served as control. Patients were followed for urinary parameters, complications and stent encrustation graded at removal. Multivariate regression and ROC analyses identified predictors of significant encrustation. **Results:** Grade 0 encrustation was higher in Group A (65.45% vs. 36.36%; p=0.003). Urinary pH and citrate were higher in Group A (p<0.001). Low urinary citrate, low pH and positive urine culture predicted Grade ≥ 2 encrustation. Urinary citrate showed strongest predictive value (AUC 0.821). **Conclusion:** Potassium sodium hydrogen citrate reduces DJ stent encrustation severity by modifying urinary biochemistry and may enhance postoperative outcomes.

Keywords: DJ stent complications; Potassium sodium hydrogen citrate; Stent encrustation; Ureteral stones; Urinary citrate

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INTRODUCTION

Formation of calculi in the urinary tract is called urolithiasis and it is commonly encountered in clinical settings. Such stones may exist in the kidneys, ureters, bladder or urethra and the materials making up the stones can be calcium oxalate, calcium phosphate, uric acid, or struvite. When someone has ureteral calculi, ureteroscopic surgery (URS) is commonly suggested to help remove the stone burden, relieve the obstruction and maintain kidney function. After URS, surgeons routinely place double J (DJ) ureteral stents to keep the urine draining and the ureter open to avoid urinary obstruction due to edema., these stents are placed inside the ureter with their ends curled in both the renal pelvis and in the bladder. This helps urine continue to drain and supports ureter repair. But since DJ stents stay in the bladder for a long time, they can cause several complications and one of the major complications is stent encrustation and stone growth on the wall or inside the stent (Bibby *et al.*, 2021). In severe cases, removal of heavily encrusted stents may require multiple interventions, including endoscopic or percutaneous procedures, which increase operative complexity and complication risk. Early identification and timely management are therefore essential. Stent encrustation may initially remain asymptomatic, but if neglected or misdiagnosed, it can progress to significant obstruction, recurrent infection and renal impairment (Polat *et al.*, 2017; Ulker *et al.*, 2019).

Different studies find varying levels of DJ stent encrustation, but most agree that it increases greatly as the stent remains indwelling for a long time. It is shown in studies that encrustation appears in about 9% of stents lasting up to 6 weeks, this percentage climbs to 47% to 76% for stents lasting over 6 months (Kartal *et al.*, 2018; Legrand F *et al.*, 2021). The research brings home the point that timely exchange or removal of stents is very important. The start of encrustations usually shows no symptoms, but they can become a serious problem if neglected or if it is misdiagnosed. When infection continues, patients may develop problems such as painful urination, blood in the urine, urinating more frequently, pain in the sides or recurrent fever from UTIs (Cauda *et al.*, 2017; Kadihasanoglu *et al.*, 2017). Additional factors that affect metabolism such as elevated urinary calcium levels, low urinary citrate, increased oxalate levels and limited urine volume, play a major role in people who have had nephrolithiasis before. Changes in urinary pH influence different stone-forming salts in different ways. A persistently alkaline urinary pH, especially in the presence of low urine flow, may promote precipitation of calcium phosphate and contribute to stent-related concretion formation (Mosayyebi *et al.*, 2018; Scotland KB *et al.*, 2019). Therefore, stent encrustation in DJ stents can result in worse problems than just being blocked and sometimes demands immediate medical intervention (Tsukanov *et al.*, 2018).

Potassium sodium hydrogen citrate raises urinary pH within a controlled therapeutic range, improving the solubility of uric acid and enhancing urinary citrate levels,

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both of which inhibit calcium-based crystal aggregation. Thus, appropriate alkalinization prevents uric acid crystallization and increases citrate-mediated inhibition of calcium stone formation, without excessively increasing the risk of phosphate precipitation. Hence, pre-emptively giving medication during or after surgery to patients with a high risk of forming kidney stones is well justified and works well (De Grazia *et al.*, 2019; Forbes *et al.*, 2019; Mosayyebi *et al.*, 2017).

Together with the introduction of new drugs, much study has been put into refining ureteral stents and the materials used to reduce the likelihood of encrustations, infections, or pain for patients. Modern stents are now different from the first ones because their materials, surfaces and coatings have changed a great deal. When compared with regular stents, polyurethane and silicone versions have been proven to make crystals and bacteria adhere less to the material (Scarneciu *et al.*, 2018 Mousavi *et al.*, 2024; Thongprayoon *et al.*, 2020).

J DJ stents are prone to growing calculi and this depends on several factors related to biology, chemistry and physics. The main problem is urine becoming stagnant which usually occurs at coiled parts of the stent or those parts that touch the lining of the ureter. Long periods of urine stasis cause more time for solutes to stay together which boosts the chance for crystals to form. Also, growth of bacteria in the stent such as *Proteus*, *Klebsiella* or *Pseudomonas* increases urine pH by changing urea into ammonia. This environment makes it easy for magnesium ammonium phosphate (struvite) and carbonate apatite to develop in stent encrustations found in infections (Alblowi *et al.*, 2022; Sorokin *et al.*, 2017). The bacteria form a sticky layer that holds urine fluid and attracts minerals which get deposited there. Besides, when left in the body, the DJ stent may provide sites for growing stones. When there are tiny bumps on the surface, manufacturing flaws or the stent material deteriorates, these become places where crystals develop irregularly. Because the stent surface is rough, crystals gather, grow larger and end up forming big encrustations. This points to how urinary chemistry, microorganisms in the urinary tract and the biophysics of stents influence each other. For this reason, controlling the solute levels, pH and preventing infections in urine may become important to prevent stent-connected stones (Kamal *et al.*, 2024, Safday *et al.*, 2021).

There is a mounting number of scientific studies showing that potassium sodium hydrogen citrate helps reduce encrustation on DJ stents. When compared to standard hydration, patients who get this drug after surgery have lower chances of forming stones in their ureteral stent, according to research (Stamatelou and Goldfarb, 2023; Shastri *et al.*, 2023). Because of these results, some surgical centres now include pharmacologic citrate therapy as part of the care plan for patients undergoing urological

stone treatments. Nevertheless, because of shortcomings like small sizes, mixed groups of patients and unequal follow-up strategies, existing studies do not cover as many aspects of PICOTS (Ripa *et al.*, 2022). Besides, other important factors that may help therapy, such as when and how long citrate is given, the pH range in the urine, total urine output and how strictly patients stick to their medications, are yet to be fully investigated. So, there is a need for studies that look at groups of patients sorted by how their metabolism responds to the treatments, as this would help evaluate which treatments work best in which patients. Also, variations in the type of stent used, how long the stent is and how it is inserted may add confusion to future studies. Since the selection of patients and the therapeutic approach are not well defined, the benefits of potassium sodium hydrogen citrate for avoiding stent encrustation have not been fully realized (Alelign and Petros, 2018).

Because of these changes, urinary alkalinization and citrate might be mostly helpful for these groups. Also, the common re-occurrence of stones and high chances of stent problems in these regions explain the benefit of proactive prevention measures. Latest regional studies strongly agree that citrate salts decrease stone recurrence and help change metabolic markers involved in stone formation (Jia *et al.*, 2024; Kamal *et al.*, 2020, Al Darrab *et al.*, 2020, Cabo *et al.*, 2023).

Specific factors such as optimal dosing regimen, timing of initiation relative to stent placement, duration of therapy and patient adherence need to be thoroughly investigated. Additionally, biochemical parameters including changes in urinary citrate concentration, pH modulation and 24-hour urine composition should be assessed to correlate therapeutic effects with metabolic outcomes. By addressing these gaps in knowledge, the medical community can develop a robust, evidence-based approach in incorporating potassium sodium hydrogen citrate into the postoperative management of patients with DJ stents (Wang and Wang, 2024). However, there remains a lack of controlled studies evaluating how changes in urinary citrate and pH specifically influence DJ stent encrustation when stent dwell time and surgical technique are standardized. Additionally, few studies have conducted multivariate and ROC-based analyses to identify independent biochemical predictors of encrustation. The present study addresses these gaps and aims to contribute to this body of evidence by evaluating both the effect of the agent on stent wall stone formation and identifying clinical and biochemical factors that influence treatment outcomes.

MATERIALS AND METHODS

The Department of Urology at a teaching hospital for tertiary care was the setting for this retrospective, observational and comparative study. The goal was to

study whether the use of potassium sodium hydrogen citrate affected the formation of stones that stay attached to the ureter after ureteroscopic lithotripsy (URS). Besides, this study examined a wide range of clinical, biochemical and microbiological reasons that may cause stent encrustation. The Institutional Ethics Committee gave ethical approval before the study started. Every patient who joined the study was informed regarding what the study hoped to achieve, how it would be done, what outcomes might result and what dangers they could face. The informed consent provided by every patient gave them the option to withdraw at any moment and still receive the usual level of care.

Sample size calculation

The sample size was calculated to detect a minimum expected difference of 25% in moderate to severe stent encrustation rates between the citrate and control groups. With a power of 80% and a two-sided α of 0.05, the required sample size was 50 patients per group. To compensate for possible dropouts, 55 patients were recruited into each group.

There were 110 participants in the study, all adults aged 18 to 70 with radiopaque ureteral stones who were set for elective ureteroscopy, with a stent placed afterward. All patients took part in the study by recruiting as they became eligible and having detailed clinical, imaging and laboratory evaluations.

Inclusion criteria

People in the study were adults 18–70 years old who had a confirmed diagnosis of only one unilateral ureteral stone (near the kidney, in the middle or near the bladder) obtained with non-contrast computed tomography of the kidney and bladder. The patients were set up for URS with DJ stenting and all had normal renal function (creatinine below 1.5 mg/dl and eGFR over 60 mL/min/1.73 m²) (Mosayyebi *et al.*, 2018; Scotland *et al.*, 2019). Anyone included in the study had to have a negative urine culture plus be willing to follow the required treatments and testing.

Exclusion criteria

Patients were excluded if they had bilateral stones or stones affecting multiple renal units requiring multiple procedures, as well as those with ureteral stents or nephrostomy tubes, active urinary tract infection or urosepsis, or chronic kidney disease stage 3 or higher. People with problems affecting the liver, those with uncontrolled diabetes, those born with or having acquired urological complications (including pelvi-ureteric junction obstruction and ureteral strictures), pregnant or lactating women, those known to be sensitive to citrate compounds and those with a history of diseases that cause metabolic stones (like cystinuria or primary hyperoxaluria) were not considered for this study. Anyone who had recently used

citrate or thiazide treatment was not enrolled in the research.

Study groups and intervention

When URS and stenting were successfully done, patients were randomly assigned to either group by a computer. Group A (n=55) started receiving potassium sodium hydrogen citrate granules by mouth (10 mEq twice per day with water after meal, for a total of four weeks) from the second day after surgery. Another group of patients (Group B, n=55) had routine care after surgery but did not get urinary agents to raise their urine pH (Pearle *et al.*, 2014; EAU Guidelines Panel, 2025). Both groups were advised to consume a minimum of 2.5 litres of water daily and were educated on dietary modifications, with emphasis on the importance of adequate hydration. The dosing regimen (10 mEq twice daily) was selected based on previously published clinical protocols for urine alkalinization in urolithiasis patients (Leslie and Sajjad, 2024; Mayo Clinic, 2025).

Surgical procedure and stenting protocol

All patients underwent semi-rigid ureteroscopic lithotripsy using a 6/7.5 Fr ureteroscope under spinal or general anaesthesia. Stone fragmentation was performed using a holmium: YAG laser, with energy settings ranging between 0.8–1.2 J and frequency between 8–12 Hz. Fragment retrieval was performed selectively, with smaller fragments left for spontaneous passage. After ensuring adequate clearance and haemostasis, a polyurethane DJ stent (5 Fr, 24–26 cm) was placed under fluoroscopic guidance (Fig. 1). Stent dwell time was uniformly maintained at 28 ± 2 days for all patients and stent removal was scheduled accordingly.



Fig. 1: Post-ureteroscopic radiograph showing early encrustation of double J stent wall

Parameters recorded

No patient was excluded from the study and all had a set of parameters recorded. Information collected for each participant included their age, sex, body mass index (BMI), history of stone disease and included details about their medical family history and current health problems like hypertension, diabetes, gout, or the metabolic syndrome. Parameters related to stones were size, whether the stone was in the left or right ureter, its position in the ureter (up, in the middle or down), its density in Hounsfield units (HU) and its volume measured on NCCT. Surgeons reported if the kidney was completely clear of stones and if they had to perform additional procedures. The type of stone was checked following surgery when possible. Compared to the results before surgery, investigations were repeated 4 weeks before the stent was removed. Interpreted values were: serum creatinine, uric acid, calcium, phosphate, electrolytes, complete blood count and fasting glucose. Spot urine pH, urine routine microscopy, urine culture and sensitivity and a 24-hour urine collection for calcium, oxalate, citrate, uric acid, magnesium and volume were part of the urine analysis. Second urine pH, 24-hour urinary analysis and urine culture were performed at the time of follow-up (refer to Figs. 2, 3).



Fig. 2: Advanced encrustation and stone formation along the double J stent loop and shaft.

Stent-related data included the duration of stent indwelling (in days), and patient-reported symptoms such as flank pain, dysuria, hematuria, and lower urinary tract symptoms (LUTS). Any complications encountered during stent removal were recorded.

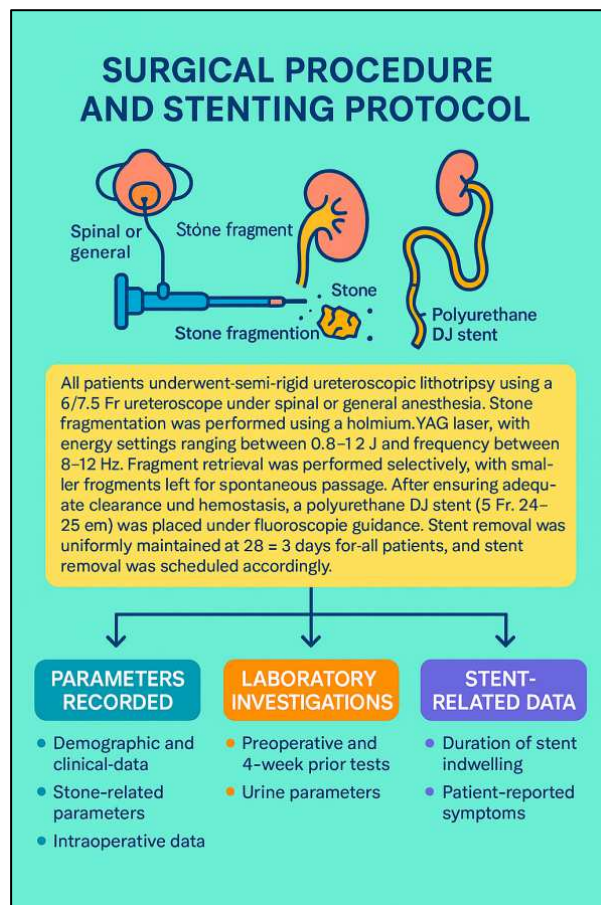


Fig. 3: Postoperative biochemical and urine evaluations performed at 4-week follow-up prior to stent removal.

Stent encrustation evaluation

Upon removal, each DJ stent was thoroughly rinsed with sterile saline and subjected to gross visual examination under magnification for colour change, surface irregularities and mineral deposits. Encrustation was graded using the Griffith Visual Scale, where Grade 0 indicated no visible encrustation, Grade 1 indicated mild deposits involving less than 25% of the surface, Grade 2 indicated moderate deposits involving 25-50% of the surface and Grade 3 represented severe encrustation involving more than 50% of the surface or luminal obstruction (Griffith *et al.*, 1978; Kawahara *et al.*, 2012). Selected stents underwent light microscopy and scanning electron microscopy (SEM) for surface characterization. Where sufficient deposits were found, compositional analysis of encrustations was performed using Fourier-transform infrared spectroscopy (FTIR).

Outcome measures

The main outcome of the research was the occurrence and severity (grade) of stent encrustation in the intervention and control groups. The secondary objectives encompassed alterations in urine parameters following treatment, namely urinary pH and citrate concentrations, along with their

association with the extent of encrustation. The study aimed to evaluate the correlation between metabolic risk factors and encrustation, the occurrence of stent-related LUTS, infections and obstruction, while identifying independent predictors of encrustation by multivariate statistical modelling.

Statistical analysis

All gathered data were put into Microsoft Excel 2021 and analysed utilising SPSS version 26.0 (IBM Corp., Armonk, NY). Categorical variables, including sex, presence of encrustation and incidence of symptoms were represented as frequencies and percentages and compared across groups utilising the Chi-square test or Fisher's exact test. Continuous variables such as urine pH, citrate concentrations and stone dimensions were reported as mean \pm standard deviation (SD) or median with interquartile range (IQR), contingent upon their distribution. Group comparisons were performed utilising the independent samples t-test for normally distributed data or the Mann-Whitney U test for skewed data. Correlations between urine parameters and stent encrustation grades were evaluated using Pearson or Spearman correlation coefficients. A multivariate logistic regression model was utilised to ascertain independent predictors of stent encrustation, including all factors with a p-value <0.10 from univariate analysis into the final model. A p-value of less than 0.05 was deemed statistically significant.

RESULTS

Baseline demographic and clinical characteristics

The demographic attributes of both research cohorts were analogous, indicating effective randomisation. The average age of patients in the citrate group was 44.20 ± 10.60 years, whereas it was marginally greater in the control group at 45.80 ± 9.80 years (Table 1). Nonetheless, this disparity was not statistically significant ($p = 0.420$), suggesting age uniformity across both groups. The male-to-female distribution was virtually equal, with Group A exhibiting a ratio of 36:19 and Group B a ratio of 34:21 ($p = 0.684$), indicating an absence of gender bias. The BMI, a possible determinant in metabolic disorders and stone formation, was comparable between the groups (25.40 ± 3.20 kg/m² vs. 25.70 ± 3.50 kg/m²; $p = 0.610$), excluding obesity as a confounding variable. A same percentage of patients in Group A (49.09%) and Group B (47.27%) had a prior history of urinary stone disease ($p = 0.839$), indicating equivalent recurrence risk in both groups. The familial history of nephrolithiasis was comparably dispersed (25.45% in Group A vs 29.09% in Group B; $p = 0.671$), indicating an equivalent genetic risk. The occurrence of common comorbidities, including hypertension (23.64% vs. 27.27%; $p = 0.665$), diabetes mellitus (16.36% vs. 18.18%; $p = 0.800$) and gout/metabolic syndrome (9.09% vs. 10.91%; $p = 0.744$), exhibited no significant differences between the groups. The mean systolic blood pressure was

124.30 ± 10.40 mmHg in Group A and 125.20 ± 9.80 mmHg in Group B, whereas the diastolic pressure was 78.50 ± 8.20 mmHg compared to 77.90 ± 7.90 mmHg, respectively. Neither systolic ($p = 0.601$) nor diastolic readings ($p = 0.690$) exhibited statistical significance. The mean fasting blood glucose levels were comparable in both groups (94.40 ± 11.30 mg/dL in Group A vs 95.20 ± 10.90 mg/dL in Group B; $p = 0.770$), indicating metabolic similarity.

Stone characteristics and surgical parameters

The stone-related features and intraoperative factors were effectively comparable between the two research groups. The average stone size in the citrate group was somewhat lower (9.40 ± 1.70 mm) than in the control group (9.60 ± 1.90 mm), however the difference lacked statistical significance ($p = 0.580$). The distribution of stone locations exhibited a balanced pattern: upper ureter stones were found in 32.73% of Group A compared to 30.91% in Group B ($p = 0.837$), mid-ureter stones were 27.27% in Group A and 29.09% in Group B and distal ureter stones were 40% in both groups. The p-value of 0.837 represents the comparison of upper ureter stones between the two groups only, not a comparison across all three stone locations. The mean stone density, expressed in HU, was 925.00 ± 150.00 in Group A and 937.00 ± 158.00 in Group B ($p = 0.687$), signifying comparable levels of stone hardness and resistance to fragmentation. Stone removal after ureteroscopy was practically complete in both groups 94.55% in the citrate group and 92.73% in the control group ($p = 0.711$)—indicating comparable procedural effectiveness.

Intraoperative laser parameters were nearly equivalent, with Group A using 0.98 ± 0.12 J of energy at a frequency of 10.20 ± 1.80 Hz, in contrast to Group B's 0.99 ± 0.15 J and 10.10 ± 1.60 Hz (Table 2). The variations were not statistically significant ($p = 0.813$ for energy, $p = 0.790$ for frequency), indicating consistency in surgical approach. The average duration of indwelling DJ stents was standardised in both groups: 28.20 ± 1.60 days in Group A and 28.10 ± 1.80 days in Group B ($p = 0.827$), therefore excluding stent dwell time as a possible confounding variable in encrustation results.

Laboratory and urine biochemical parameters at pre-treatment, 4 weeks and 6 weeks

The metabolic response to potassium sodium hydrogen citrate treatment was apparent across several parameters. Serum creatinine levels were consistent in both groups over time, exhibiting no significant intergroup difference at 4 weeks (0.90 ± 0.10 mg/dL in Group A vs. 0.91 ± 0.10 mg/dL in Group B; $p = 0.740$), suggesting that renal function was maintained throughout the study duration and was unaffected by citrate therapy. The urinary pH in the citrate group significantly increased from a baseline of 5.60 ± 0.30 to 6.80 ± 0.50 at 4 weeks, followed by a little

decrease to 6.70 ± 0.40 at 6 weeks. Conversely, Group B had a little increase in urine pH from 5.70 ± 0.40 to 5.90 ± 0.40 at 4 weeks, remaining relatively stable at 5.85 ± 0.40 by 6 weeks. The intergroup difference at 4 weeks was statistically significant ($p < 0.001$), indicating that potassium sodium hydrogen citrate efficiently alkalinised the urine. A significant elevation in 24-hour urine citrate was noted in the citrate group, increasing from 280.00 ± 90.00 mg to 520.00 ± 110.00 mg at 4 weeks, followed by a modest reduction to 500.00 ± 100.00 mg at 6 weeks. The control group had a little rise from 270.00 ± 95.00 mg to 295.00 ± 100.00 mg at 4 weeks, staying relatively stable at 290.00 ± 95.00 mg at 6 weeks. The disparity in urinary citrate across the groups after 4 weeks was statistically significant ($p < 0.001$), indicating the biochemical effectiveness of the intervention. Urinary calcium levels in Group A diminished somewhat from 210.00 ± 45.00 mg to 200.00 ± 48.00 mg at 4 weeks and subsequently to 198.00 ± 46.00 mg at 6 weeks. Group B had a marginal rise from 215.00 ± 50.00 mg to 218.00 ± 55.00 mg, followed by a little decrease to 216.00 ± 54.00 mg. Nonetheless, this alteration was not statistically significant ($p = 0.582$), suggesting a little impact of citrate treatment on urine calcium levels. Concerning urinary oxalate, Group A exhibited a reduction from 34.00 ± 8.00 mg to 30.00 ± 9.00 mg at 4 weeks and 29.00 ± 8.00 mg at 6 weeks. Group B had relatively steady levels (33.00 ± 7.00 mg to 32.00 ± 10.00 mg and thereafter 32.00 ± 9.00 mg), with no significant difference detected between the groups at 4 weeks ($p = 0.401$). In the citrate group, urinary uric acid levels exhibited a small reduction from 520.00 ± 80.00 mg to 500.00 ± 90.00 mg and subsequently to 495.00 ± 85.00 mg. Conversely, Group B had little variations, recording values of 510.00 ± 85.00 mg, 515.00 ± 95.00 mg and 510.00 ± 90.00 mg at the three respective time periods. The alterations were not statistically significant ($p = 0.553$), indicating that citrate exerted a negligible influence on uric acid excretion under these circumstances. Urinary magnesium levels in Group A rose from 84.00 ± 10.00 mg to 88.00 ± 12.00 mg at 4 weeks and marginally increased to 89.00 ± 11.00 mg at 6 weeks, whereas Group B exhibited relative stability, changing from 85.00 ± 9.00 mg to 86.00 ± 11.00 mg and remaining at 86.00 ± 10.00 mg thereafter. The intergroup difference at 4 weeks was not significant ($p = 0.320$), indicating that magnesium excretion was not influenced by the therapy (Table 3).

For clearer interpretation, the biochemical parameters at 4 weeks were directly compared between Group A and Group B, as shown in the reformatted table 3. In addition to inter-group comparisons, intra-group changes from baseline to 4 weeks were analysed. Group A demonstrated significant increases in urinary pH and citrate from baseline to 4 weeks ($p < 0.001$ for both), whereas Group B showed no significant intra-group change over the same interval.

Stent-related symptoms and complications

Symptoms associated with stents were typically more prevalent in the control group than in the citrate group, but several differences did not achieve statistical significance. Flank discomfort was seen in 21.82% of patients in Group A and 34.55% in Group B ($p = 0.137$), suggesting a potential but non-significant trend towards symptom alleviation in the citrate group. Dysuria was seen in 32.73% of Group A and 43.64% of Group B ($p = 0.231$), indicating a numerical decrease but without statistical significance. Haematuria, whether microscopic or gross, was observed in 18.18% of patients in the citrate group compared to 23.64% in the control group ($p = 0.492$). Additionally, LUTS, such as frequency and urgency, were reported by 29.09% in Group A against 38.18% in Group B ($p = 0.294$). The results indicate that although symptoms were somewhat less prevalent in the intervention group, the changes lacked statistical significance.

Fever episodes were seen in 7.27% of the citrate group compared to 14.55% in the control group ($p = 0.218$), potentially indicating a diminished inflammatory response or infection rate associated with lower encrustation, however not statistically significant. The difficulty of stent removal was significantly greater in the control group (12.73%) than in the citrate group (3.64%), nearing statistical significance ($p = 0.081$), possibly associated with increased stent encrustation without citrate treatment. Notably, the incidence of stent-associated urinary tract infection (UTI) was significantly reduced in the citrate group (5.45%) vs to the control group (16.36%), with a p-value of 0.048. This statistically significant difference corroborates the preventive function of potassium sodium hydrogen citrate in diminishing encrustation and bacterial colonisation, thereby reducing the incidence of urinary tract infections (Table 4).

Stent encrustation grading and predictive parameters

The grading of DJ stent encrustation revealed a significant protective effect of potassium sodium hydrogen citrate against moderate to severe encrustation. In Group A (citrate), 36 patients (65.45%) had Grade 0 encrustation, compared to only 20 patients (36.36%) in Group B (control), a statistically significant difference ($p = 0.003$). This demonstrates that two-thirds of the citrate group had stents completely free of encrustation, whereas this was true for just over one-third of the control group. Mild encrustation (Grade 1, <25% surface) occurred in 21.82% of Group A and 30.91% of Group B, while moderate encrustation (Grade 2, 25–50%) was seen in 9.09% of the citrate group versus 20.00% in controls. Severe encrustation (Grade 3, >50% surface area or stent obstruction) occurred in only 2 patients (3.64%) in the citrate group, compared to 7 patients (12.73%) in the control group. Although not all grades individually reached statistical significance, the overall trend clearly favored the citrate group in reducing encrustation severity.

The analysis of predictive urinary factors showed that, when patients from both groups were pooled according to encrustation severity, those with no encrustation (Grade 0) had a significantly higher mean urinary pH (6.70) compared to those with Grade 2 or 3 encrustation (5.90), with a p -value < 0.01 . These findings reinforce the direct correlation between low urinary pH and hypospitraturia with increased stent encrustation risk.

Urine culture positivity was significantly more common in patients with moderate to severe encrustation—only 1 of 36 (2.78%) in the citrate group vs. 6 of 33 (18.18%) in the control group had positive cultures ($p = 0.048$). This highlights the role of bacterial colonization and biofilm formation in promoting mineral deposition on the stent surface. Additionally, LUTS were reported in 3 patients (8.33%) with Grade ≥ 2 encrustation in Group A versus 9 patients (27.27%) in Group B ($p = 0.041$), suggesting that symptomatic stents are more likely to have moderate/severe mineral buildup, possibly due to local irritation or infection (Table 5).

Multivariate logistic regression analysis for predictors of moderate to severe stent encrustation (Grade ≥ 2)

This table 6 delineates independent variables of moderate to severe DJ stent encrustation by multivariate logistic regression analysis. The likelihood of developing Grade ≥ 2 encrustation was 2.87 times greater in participants from the control group than in those from the citrate group (95% CI: 1.12–7.41; $p = 0.028$). This highlights the preventive effect of potassium sodium hydrogen citrate, even after accounting for confounding variables. A urinary pH of less than 6.0 correlated with a heightened likelihood of encrustation (AOR = 3.22; 95% CI: 1.38–7.48; $p = 0.007$), demonstrating strong sensitivity (81.25%) and satisfactory specificity (70.73%). Low urine citrate (< 400 mg/24h) emerged as a robust independent predictor (AOR = 3.91; 95% CI: 1.62–9.43; $p = 0.002$), exhibiting the highest sensitivity (84.37%) and specificity (74.14%) among the variables assessed. A positive urine culture independently forecasted encrustation (AOR = 2.74; $p = 0.049$), demonstrating high specificity (83.90%) but lower sensitivity (53.13%), indicating that not all encrustation instances were linked to infection; nonetheless, when infection was present, the predictive value was substantial. The presence of LUTS neared significance (AOR = 2.35; $p = 0.078$), indicating a trend but not establishing it as a definite independent predictor. Diabetes mellitus did not demonstrate a statistically significant correlation (AOR = 1.61; $p = 0.359$), suggesting it may not independently influence encrustation risk in this cohort.

ROC curve analysis of predictive parameters for stent encrustation (Grade ≥ 2)

The ROC analysis provided diagnostic performance metrics for each of the main predictors of encrustation. Urinary citrate at 4 weeks showed the highest

discriminative power, with an AUC of 0.821 (95% CI: 0.736–0.906). A cut-off of < 400 mg/24h yielded 84.37% sensitivity and 74.14% specificity ($p < 0.001$), confirming this as the strongest predictor of significant encrustation. Urinary pH at 4 weeks had a slightly lower AUC of 0.793 (95% CI: 0.705–0.882), with an optimal cut-off value of < 6.1 yielding 81.25% sensitivity and 70.73% specificity ($p < 0.001$). This reinforces the value of urine alkalization as a preventive strategy. Positive urine culture showed moderate accuracy (AUC = 0.681), with high specificity (83.90%) but lower sensitivity (53.13%), consistent with regression findings. Presence of LUTS had the lowest AUC (0.643), reflecting moderate diagnostic value (sensitivity 65.62%, specificity 67.80%), but still statistically significant ($p = 0.034$), suggesting that symptomatic patients warrant closer monitoring (Table 7, Fig. 4).

DISCUSSION

The demographic and clinical characteristics of the two groups were well aligned in this investigation, validating successful randomisation. The average age was 44.20 ± 10.60 years in Group A and 45.80 ± 9.80 years in Group B, which aligns with the populations examined in research by Prochaska *et al.* (2018) and Rodriguez *et al.* (2021), who documented mean ages between 42 and 47 years in stone-forming cohorts. The comparable gender pattern (65.5% males in Group A versus 61.8% in Group B) aligns with the male preponderance commonly noted in urolithiasis, as reported by Ferraro *et al.* (2021)

The current study indicated a BMI of roughly 25.5 kg/m² in both groups, signifying a range from normoweight to overweight. This aligns with the findings of Arivoli *et al.* (2024), who reported a BMI range of 24–26 kg/m² in patients receiving DJ stenting following ureteroscopic stone extraction. Additionally, comorbidities such as hypertension and diabetes mellitus were seen in 23.64% and 16.36% of the citrate group, respectively, consistent with the 20–30% prevalence documented in studies by Lewandowski *et al.* (2020) among recurrent stone formers. Both groups had comparable stone dimensions (9.40 ± 1.70 mm vs. 9.60 ± 1.90 mm) and anatomical placements, with a majority located in the distal ureter (40.0% in each group). This reflects the pattern shown by Triozzi *et al.* (2023) and Ferraro *et al.* (2021), wherein distal ureteral stones constituted 38–42% of patients undergoing ureteroscopy. The average stone density in our study (925–937 HU) aligns with the range linked to calcium oxalate stones, corroborated by Marangella *et al.* (2021), who documented mean densities of 900–1000 HU for mixed oxalate-phosphate stones.

Table 1: Baseline demographic and clinical characteristics (n = 110)

Parameters	Group A (Citrate, n=55)	Group B (Control, n=55)	p-value
Mean age (years)	44.20 ± 10.60	45.80 ± 9.80	0.420
Male: Female ratio	36:19	34:21	0.684
Mean BMI (kg/m ²)	25.40 ± 3.20	25.70 ± 3.50	0.610
History of stone disease (%)	27 (49.09%)	26 (47.27%)	0.839
Family history of stones (%)	14 (25.45%)	16 (29.09%)	0.671
Hypertension (%)	13 (23.64%)	15 (27.27%)	0.665
Diabetes mellitus (%)	9 (16.36%)	10 (18.18%)	0.800
Gout / Metabolic syndrome (%)	5 (9.09%)	6 (10.91%)	0.744
Mean systolic BP (mmHg)	124.30 ± 10.40	125.20 ± 9.80	0.601
Mean diastolic BP (mmHg)	78.50 ± 8.20	77.90 ± 7.90	0.690
Mean fasting glucose (mg/dL)	94.40 ± 11.30	95.20 ± 10.90	0.770

Table 2: Stone characteristics and surgical parameters

Parameters	Group A (Citrate, n=55)	Group B (Control, n=55)	p-value
Mean stone size (mm)	9.40 ± 1.70	9.60 ± 1.90	0.580
Stone location (%)			
- Upper ureter	18 (32.73%)	17 (30.91%)	0.837
- Mid ureter	15 (27.27%)	16 (29.09%)	
- Distal ureter	22 (40.00%)	22 (40.00%)	
Mean stone density (HU)	925.00 ± 150.00	937.00 ± 158.00	0.687
Complete stone clearance (%)	52 (94.55%)	51 (92.73%)	0.711
Laser energy used (J)	0.98 ± 0.12	0.99 ± 0.15	0.813
Laser frequency (Hz)	10.20 ± 1.80	10.10 ± 1.60	0.790
Mean stent indwelling time (days)	28.20 ± 1.60	28.10 ± 1.80	0.827

Table 3: Laboratory and urinary biochemical parameters at baseline, 4 weeks and 6 weeks

Parameters	Group A (Citrate) baseline	Group B (Control) baseline	Group A 4 weeks	Group B 4 weeks	p-value† (4 weeks)	Group A 6 weeks	Group B 6 weeks	p-value‡ (6 weeks)
Serum creatinine (mg/dL)	0.88 ± 0.09	0.86 ± 0.08	0.90 ± 0.10	0.91 ± 0.10	0.740	0.92 ± 0.10	0.92 ± 0.10	0.980
Urinary pH	5.60 ± 0.30	5.70 ± 0.40	6.80 ± 0.50	5.90 ± 0.40	<0.001	6.70 ± 0.40	5.85 ± 0.40	<0.001
Urinary citrate (mg/24 h)	280 ± 90	270 ± 95	520 ± 110	295 ± 100	<0.001	500 ± 100	290 ± 95	<0.001
Urinary calcium (mg/24 h)	210 ± 45	215 ± 50	200 ± 48	218 ± 55	0.582	198 ± 46	216 ± 54	0.601
Urinary oxalate (mg/24 h)	34 ± 8	33 ± 7	30 ± 9	32 ± 10	0.401	29 ± 8	32 ± 9	0.378
Urinary uric acid (mg/24 h)	520 ± 80	510 ± 85	500 ± 90	515 ± 95	0.553	495 ± 85	510 ± 90	0.566
Urinary magnesium (mg/24 h)	84 ± 10	85 ± 9	88 ± 12	86 ± 11	0.320	89 ± 11	86 ± 10	0.298

Intra-group analysis revealed that Group A experienced statistically significant increase in urinary pH and citrate levels from baseline to 4 weeks (p < 0.001), whereas no significant intra-group changes were observed in Group B.

Table 4: Stent-related symptoms and complications

Parameters	Group A (Citrate, n=55)	Group B (Control, n=55)	p-value
Flank pain (%)	12 (21.82%)	19 (34.55%)	0.137
Dysuria (%)	18 (32.73%)	24 (43.64%)	0.231
Hematuria (%)	10 (18.18%)	13 (23.64%)	0.492
LUTS (Frequency, urgency) (%)	16 (29.09%)	21 (38.18%)	0.294
Fever episodes (%)	4 (7.27%)	8 (14.55%)	0.218
Difficulty in stent removal (%)	2 (3.64%)	7 (12.73%)	0.081
Stent-associated UTI (%)	3 (5.45%)	9 (16.36%)	0.048

Table 5: Distribution of double-j stent encrustation grades and associated urinary predictors

Parameters	Group A (Citrate, n=55)	Group B (Control, n=55)	p-value
Stent Encrustation Grade			
Grade 0 (No encrustation)	36 (65.45%)	20 (36.36%)	0.003
Grade 1 (<25% surface)	12 (21.82%)	17 (30.91%)	
Grade 2 (25–50% surface)	5 (9.09%)	11 (20.00%)	
Grade 3 (>50% surface / obstruction)	2 (3.64%)	7 (12.73%)	
Mean urinary pH at 4 weeks (Grade 0 vs ≥ 2)	6.70 \pm 0.40	5.90 \pm 0.40	<0.01
Urinary citrate at 4 weeks (mg/24 h) (Grade 0 vs ≥ 2)	510 \pm 100	310 \pm 95	<0.01
Urine culture positivity – Grade ≥ 2 (%)	1 (2.78%)	6 (18.18%)	0.048
LUTS incidence – Grade ≥ 2 (%)	3 (8.33%)	9 (27.27%)	0.041

Table 6: Multivariate logistic regression analysis for predictors of moderate to severe stent encrustation (Grade ≥ 2)

Predictor variables	Adjusted odds ratio (AOR)	95% Confidence interval (CI)	p-value	Sensitivity (%)	Specificity (%)
Control group (vs. citrate group)	2.87	1.12 – 7.41	0.028	78.13	69.51
Urinary pH < 6.0	3.22	1.38 – 7.48	0.007	81.25	70.73
Urinary citrate < 400 mg/24h	3.91	1.62 – 9.43	0.002	84.37	74.14
Positive urine culture	2.74	1.01 – 7.42	0.049	53.13	83.90
Presence of LUTS	2.35	0.91 – 6.08	0.078	65.62	67.80
Diabetes mellitus	1.61	0.58 – 4.49	0.359	43.75	61.02

Table 7: ROC curve analysis of predictive parameters for stent encrustation (Grade ≥ 2)

Predictor variables	AUC (95% CI)	Optimal cut-off value	Sensitivity (%)	Specificity (%)	p-value
Urinary citrate at 4 wks (mg/24h)	0.821 (0.736–0.906)	<400	84.37	74.14	<0.001
Urinary pH at 4 wks	0.793 (0.705–0.882)	<6.1	81.25	70.73	<0.001
Positive urine culture	0.681 (0.577–0.786)	Presence of growth	53.13	83.90	0.013
LUTS (Frequency/Urgency) Presence	0.643 (0.532–0.753)	Symptomatic vs None	65.62	67.80	0.034

The findings of the present study demonstrated a clear reduction in stent encrustation among patients treated with potassium sodium hydrogen citrate. This aligns with the known biochemical role of citrate in inhibiting calcium-based crystal aggregation and modifying urinary pH. Rather than repeating the numerical results, these findings collectively support the concept that biochemical optimization is a key determinant of stent performance. The protective effect remained significant even after adjusting for confounders, reinforcing the metabolic contribution to encrustation risk.

Significantly, total stone removal was attained in 94.55% of Group A and 92.73% of Group B, aligning with the success rates documented by Rodriguez *et al.* (2021) and Prochaska *et al.* (2018), who reported clearance rates ranging from 90% to 96% utilising contemporary laser lithotripsy. The consistency in stent stay length (about 28 days) and laser settings across all groups meant that differences in results could be solely ascribed to the pharmaceutical intervention. The study's principal biochemical achievement was the significant increase in urine citrate and pH in the therapy group.

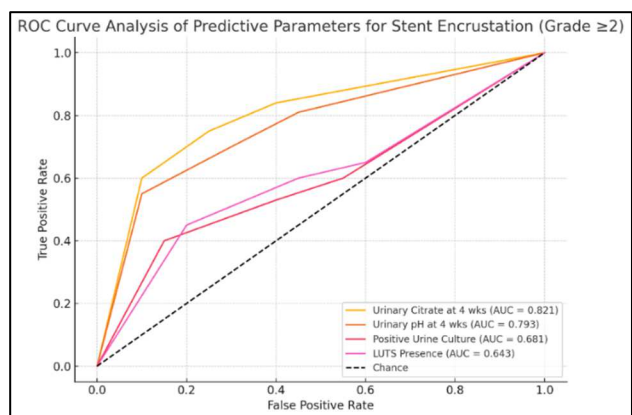


Fig. 4: ROC curve analysis of predictive parameters for stent encrustation (Grade ≥ 2)

At 4 weeks, urinary citrate elevated to 520.00 ± 110.00 mg/24h from a baseline of 280.00 ± 90.00 mg/24h and pH climbed to 6.80 ± 0.50 . The results are significantly greater than those documented by Bua *et al.* (2019), who noted mean urine citrate elevations of around 150–200 mg/24h with potassium citrate treatment. The enhanced response seen in this study may be attributed to the application of potassium sodium hydrogen citrate, which might have synergistic buffering effects. Conversely, the control group exhibited no variation in urine citrate levels (270.00 to 295.00 mg/24h), corroborating the observations of Royaux *et al.* (2001), which indicated that dietary alterations alone may not substantially impact hypocitraturia. The citrate group exhibited a notable elevation in urinary pH relative to the control group (6.80 vs. 5.90; $p < 0.001$), corroborating the results of Xu *et al.* (2018), who emphasised citrate's function in neutralising acidic urine and improving uric acid solubility. Although urine calcium and oxalate levels exhibited a small decline in the therapy group, the changes were not statistically significant. This conclusion aligns with the meta-analysis by Lin *et al.* (2022), which determined that citrate treatment predominantly modifies urinary citrate and pH, although had less direct influence on calcium and oxalate unless accompanied by dietary restrictions or thiazides. Urinary magnesium in the treatment group exhibited a small rise (from 84.00 to 88.00 mg/24h), however not substantially. Given that magnesium functions as a crystallisation inhibitor, its elevation may facilitate the reduced encrustation rates shown in subsequent results. This aligns with findings by Triozzi *et al.* (2023), who highlighted the supplementary advantages of urine magnesium in diminishing stone recurrence. The consistency of serum creatinine levels in both groups over six weeks substantiates the renal safety of potassium sodium hydrogen citrate, aligning with the findings of Ware *et al.* (2017) and Merrins *et al.* (2022), who assessed the metabolic safety profile of alkalinising therapies in patients with preserved renal function. Symptoms associated with stents were typically more prevalent in the control group

than in the citrate group, but several differences did not achieve statistical significance. Flank discomfort was seen in 21.82% of participants in Group A and 34.55% in Group B ($p = 0.137$), suggesting a potential but statistically insignificant tendency towards symptom alleviation in the citrate group. Dysuria was seen in 32.73% of Group A and 43.64% of Group B ($p = 0.231$), indicating a numerical decrease without statistical significance. This aligns with the findings of Ferraro *et al.* (2021), who noted that acidic urine is more prone to irritate the bladder and intensify lower tract symptoms. Haematuria, whether microscopic or gross, was observed in 18.18% of patients in the citrate group compared to 23.64% in the control group ($p = 0.492$). Additionally, LUTS—encompassing frequency and urgency—were reported by 29.09% in Group A against 38.18% in Group B ($p = 0.294$). The results indicate that although symptoms were somewhat less prevalent in the intervention group, the changes lacked statistical significance. saw comparable results, indicating decreased LUTS with urinary alkalinisation during stenting (Bua S *et al.*, 2019). Fever episodes were seen in 7.27% in the citrate group compared to 14.55% in the control group ($p = 0.218$), potentially indicating a diminished inflammatory response or infection rate due to reduced encrustation, however not statistically significant. The control group experienced much more difficulty during stent removal (12.73%) than the citrate group (3.64%), nearing statistical significance ($p = 0.081$), possibly attributable to more severe stent encrustation without citrate treatment, as shown by Triozzi *et al.* (2023). The incidence of stent-associated urinary tract infection (UTI) was significantly reduced in the citrate group (5.45%) compared to the control group (16.36%), with a p-value of 0.048. This statistically significant difference substantiates the protective function of potassium sodium hydrogen citrate in diminishing encrustation and bacterial colonisation, consequently reducing UTI risk, a mechanism associated by Royaux *et al.* (2001) with bicarbonate secretion and antimicrobial defence in renal tubules. The assessment of DJ stent encrustation shown a notable preventive influence of potassium sodium hydrogen citrate against mild to severe encrustation. In Group A (citrate), 36 patients (65.45%) exhibited Grade 0 encrustation, whereas only 20 patients (36.36%) in Group B (control) did, indicating a statistically significant difference ($p = 0.003$). This indicates that two-thirds of the citrate group possessed stents entirely devoid of encrustation, in contrast to somewhat more than one-third of the control group. These results align with those of Marangella *et al.* (2021), who noted markedly reduced stone deposition in individuals with optimised urine chemistries. Mild encrustation (Grade 1, $< 25\%$ surface) was seen in 21.82% of Group A and 30.91% of Group B, however moderate encrustation (Grade 2, 25–50%) occurred in 9.09% of the citrate group compared to 20.00% in the control group. Severe encrustation (Grade 3, $> 50\%$ surface area or stent blockage) was observed in only 2 patients (3.64%) in the

citrate group, in contrast to 7 patients (12.73%) in the control group. While not all grades achieved statistical significance individually, the overarching trend distinctly favoured the citrate group in mitigating encrustation severity. Xu *et al.* (2018) established that reduced urine pH facilitates crystal adherence and biofilm development on stents.

The examination of predictive urine variables revealed that individuals without encrustation (Grade 0) had a markedly elevated mean urinary pH (6.70) in contrast to those with Grade 2 or 3 encrustation (5.90), yielding a p-value of <0.01 . Urinary citrate levels were significantly elevated in the non-encrusted group (510.00 mg/24h) compared to the substantially encrusted group (310.00 mg/24h), with $p < 0.01$. These data substantiate the direct relationship between low urine pH and hypocitraturia with an elevated risk of stent encrustation, as previously shown by Lin *et al.* (2022). Urine culture positivity was markedly more prevalent among patients exhibiting moderate to severe encrustation—only 1 of 36 (2.78%) in the citrate group compared to 6 of 33 (18.18%) in the control group had positive cultures ($p = 0.048$). This underscores the significance of bacterial colonisation and biofilm formation in facilitating mineral deposition on the stent surface, aligning with the infection-encrustation correlation delineated by Ware *et al.* (2017). Moreover, LUTS were observed in 3 patients (8.33%) with Grade ≥ 2 encrustation in Group A compared to 9 patients (27.27%) in Group B ($p = 0.041$), indicating that symptomatic stents are more prone to moderate/severe mineral accumulation, potentially attributable to local irritation or infection as posited by Ferraro *et al.* (2021). This table delineates independent variables of moderate to severe DJ stent encrustation by multivariate logistic regression analysis. The likelihood of developing Grade ≥ 2 encrustation was 2.87 times greater in participants from the control group than in the citrate group (95% CI: 1.12–7.41; $p = 0.028$). This highlights the preventive influence of potassium sodium hydrogen citrate, even after accounting for confounding variables. Arivoli *et al.* (2024) revealed analogous findings, affirming the efficacy of pharmacologic management in averting recurrence and problems related with stents. A urinary pH below 6.0 correlated with a heightened likelihood of encrustation (AOR = 3.22; 95% CI: 1.38–7.48; $p = 0.007$), demonstrating strong sensitivity (81.25%) and satisfactory specificity (70.73%). Low urinary citrate (<400 mg/24h) emerged as a robust independent predictor (AOR = 3.91; 95% CI: 1.62–9.43; $p = 0.002$), demonstrating the highest sensitivity (84.37%) and specificity (74.14%) among all assessed variables—supporting the findings of Bua *et al.* (2019) regarding urinary alkalisers as metabolic adjuncts. A positive urine culture independently predicted encrustation (AOR = 2.74; $p = 0.049$), demonstrating high specificity (83.90%) but lower sensitivity (53.13%), indicating that not all encrustation instances were linked to

infection; nonetheless, when infection was present, the predictive value was substantial. This further corroborates the prior biochemical results put out by Royaux *et al.* (2001). The presence of LUTS neared significance (AOR = 2.35; $p = 0.078$), indicating a trend but not establishing it as a definite independent predictor. Diabetes mellitus had no statistically significant connection (AOR = 1.61; $p = 0.359$), suggesting it may not independently influence encrustation risk in this cohort, a conclusion aligned with the metabolic neutrality reported by Merrins *et al.* (2022). The ROC analysis yielded diagnostic performance measures for each primary predictor of encrustation. At 4 weeks, urinary citrate had the greatest discriminative capability, with an AUC of 0.821 (95% CI: 0.736–0.906). A threshold of <400 mg/24h demonstrated 84.37% sensitivity and 74.14% specificity ($p < 0.001$), establishing it as the most robust predictor of significant encrustation. This efficacy aligns with the clinical relevance addressed by Lin *et al.* (2022) concerning citrate supplementation in urolithiasis.

The urinary pH at 4 weeks exhibited a marginally reduced AUC of 0.793 (95% CI: 0.705–0.882), with an appropriate cut-off value of <6.1 resulting in 81.25% sensitivity and 70.73% specificity ($p < 0.001$). This underscores the significance of urine alkalinisation as a prophylactic measure, long promoted by Xu *et al.* (2018) and Ware *et al.* (2017). The positive urine culture demonstrated moderate accuracy (AUC = 0.681), exhibiting high specificity (83.90%) but lower sensitivity (53.13%), in accordance with regression results. The presence of LUTS exhibited the lowest AUC (0.643), indicating modest diagnostic efficacy (sensitivity 65.62%, specificity 67.80%), although remained statistically significant ($p = 0.034$), implying that symptomatic individuals necessitate further surveillance, as emphasised by Ferraro *et al.* (2021).

CONCLUSION

The present study demonstrates that potassium sodium hydrogen citrate favorably alters urinary biochemistry and substantially reduces the severity of DJ stent encrustation following ureteroscopic lithotripsy. Citrate therapy therefore represents a low-cost, safe and physiologically targeted strategy that complements existing stent technologies. This study contributes to the existing evidence by incorporating a uniform stent dwell time, standardized surgical technique and comprehensive biochemical assessment, thereby minimizing key confounders seen in earlier studies. These strengths support the reliability of the observed effects and highlight the value of integrating short-term metabolic optimization in routine postoperative care.

Limitations

This was a single-center study with a relatively small

sample size, which may limit the generalizability of the findings. The follow-up duration was restricted to 6 weeks, potentially underestimating late-onset encrustation. Stone composition analysis was not standardized across all cases. Patient compliance with hydration and dietary advice was self-reported and may have introduced bias. Moreover, microbial biofilm assessment was not done through advanced molecular methods. Future multicentric studies with longer follow-up and advanced biofilm quantification are warranted.

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None

Authors' contributions

Guodong Li: Conceptualized the study, guided data collection and supervised the statistical analysis; Xiao Luo and Guodong Li: Manuscript drafting, literature review and tabulation. All authors reviewed the final manuscript and approved for submission.

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Data availability statement

All data generated or analyzed during this study are included in this published article.

Ethical approval

This study was approved by the Longyou County People's Hospital, Longyou Zhejiang Province, Ethical approval number (2025090). This study was performed in adherence with the STROBE guidelines. See supplementary file for the STROBE checklist.

Conflicts of interest

The authors declare that they have no financial conflicts of interest.

Supplementary data

<https://www.pjps.pk/uploads/2026/06/SUP1780569576.pdf>

REFERENCES

Al Darrab R, Addar AM, Al Shohaib and Ghazwani Y (2020). Trends of upper urinary tract stone management in a high volume stone center in Saudi Arabia, 12 years analysis. *Urol. Ann.*, **12**: 128–31.

Alblowi S, Safdar O, Aboulola N, Alharazy D and Najem N (2022). Renal stone prevalence and risk factors in Jeddah and Riyadh. *J. Family Med. Prim. Care.*, **11**: 2839–2845.

Alelign T and Petros B (2018). Kidney stone disease: An update on current concepts. *Adv. Urol.*, **2018**: 3068365.

Arivoli K, Valicevic AN, Oerline MK, Hsi RS, Patel SR, Hollingsworth JM and Shahinian VB (2024). Preventive pharmacological therapy and risk of

recurrent urinary stone disease. *Clin. J. Am. Soc. Nephrol.*, **19**(5): 565–572

Bibby LM and Wiseman OJ (2021). Double JJ ureteral stenting: Encrustation and tolerability. *Eur. Urol. Focus.*, **7**(1): 7–8.

Bua S, Nocentini A and Supuran CT (2019). Carbonic anhydrase inhibitors as diuretics. In: Supuran CT and Nocentini A, editors. *Carbonic Anhydrases. Academic Press*: 287–309.

Cabo J, Gelikman DG and Hsi RS (2023). The financial burden of nephrolithiasis and predictors of disease-specific financial toxicity. *Urology*, **171**: 57–63.

Cauda V, Chiodoni A, Laurenti M, Canavese G and Tommasi T (2017). Ureteral double-J stents performances toward encrustation after long-term indwelling in a dynamic in vitro model. *J. Biomed. Mater. Res. B Appl. Biomater.*, **105**(8): 2244–2253.

De Grazia A, Somani BK, Soria F, Carugo D and Mosayyebi A (2019). Latest advancements in ureteral stent technology. *Transl. Androl. Urol.*, **8**(Suppl 4): S436–S441.

Eisner BH, Goldfarb DS, Baum MA, Langman CB, Curhan GC, Preminger GM, Lieske JC, Pareek G, Thomas K, Zisman AL, Papagiannopoulos D and Sur RL (2020). Evaluation and medical management of patients with cystine nephrolithiasis: A consensus statement. *J. Endourol.*, **34**: 1103–1110.

Fan M, Zhang J, Lee CL, Zhang J and Feng L (2023). Structure and thiazide inhibition mechanism of the human Na-Cl cotransporter. *Nature*, **614**(7949): 788–793.

Ferraro PM, Taylor EN, Gambaro G and Curhan GC (2021). Caffeine intake and the risk of kidney stones. *Am. J. Clin. Nutr.*, **114**(1): 284–291.

Forbes C, Scotland KB, Lange D and Chew BH (2019). Innovations in ureteral stent technology. *Urol. Clin. North Am.*, **46**(2): 245–255.

Guo H and Yuan JB (2023). New insights into the prevention of ureteral stents encrustation. *Open Med. (Wars)*, **18**(1): 20230854.

Jia W, Chi W, Liu C, Song Y, Yang S and Yin C (2024). Development and validation of a predictive model for double-J stent encrustation after upper urinary tract calculi surgery. *Urolithiasis*, **52**(1): 105.

Kadihasanoglu M, Kilciler M and Atahan O (2017). Luminal obstruction of double J stents due to encrustation depends on indwelling time: A pilot study. *Aktuelle Urol.*, **48**(3): 248–251.

Kamal W, Azhar RA, Hamri SB, Alathal AH, Alamri A, Alzahrani T, Abeery H, Noureldin YA, Alomar M, Own AA, Alnazari MM, Alharthi M, Awad MA, Halawani A, Althubiany HH, Alruwaily A and Violette P (2024). The Saudi urological association guidelines on urolithiasis. *Urol. Ann.*, **16**: 1–27.

Kamal WK, Alhazmy A, Alharthi M and Al Solumany A (2020). Trends of percutaneous nephrolithotomy in Saudi Arabia. *Urol. Ann.*, **12**: 352–359.

- Kartal IG, Baylan B, Gok A, Sagnak AL, Karakoyunlu N, Cakici MC, Kaymak S, Karabacak OR, Topaloglu H and Ersoy H (2018). The association of encrustation and ureteral stent indwelling time in urolithiasis and KUB grading system. *Urol. J.*, **15**(6): 323–328.
- Legrand F, Saussez T, Ruffion A, Celia A, Djouhri F, Musi G, Kalakech S, Desriac I and Roumeguere T (2021). Double loop ureteral stent encrustation according to indwelling time: Results of a European multicentric study. *J. Endourol.*, **35**(1): 84–90.
- Lewandowski SL, Cardone RL, Foster HR, Ho T, Potapenko E, Poudel C, VanDeusen HR, Sdao SM, Alves TC, Zhao X, Capozzi ME, de Souza AH, Jahan I, Thomas CJ, Nunemaker CS, Davis DB, Campbell JE, Kibbey RG and Merrins MJ (2020). Pyruvate kinase controls signal strength in the insulin secretory pathway. *Cell Metab.*, **32**(5): 736–750.e5.
- Li W, Shubin W, Min Z, Yong W, Jingchao C, Xiaoyu S and Yongjing N (2021). Effect of potassium sodium hydrogen citrate granule on the wall stone shell of preset ureteral stent before ureteroscopy. *SunText Rev. Renal Sci.*, **1**(1): 102.
- Lin Z, Wong LYF and Cheung BM (2022). Diuretic-induced hypokalaemia: An updated review. *Postgrad. Med. J.*, **98**(1160): 477–482.
- Marangella M, Petrarulo M, Vitale C, Daniele P and Sammartano S (2021). LITHORISK.COM: The novel version of a software for calculating and visualizing the risk of renal stone. *Urolithiasis*, **49**(3): 211–217.
- Merrins MJ, Corkey BE, Kibbey RG and Prentki M (2022). Metabolic cycles and signals for insulin secretion. *Cell Metab.*, **34**(6): 947–968.
- Mosayyebi A, Manes C, Carugo D and Somani BK (2018). Advances in ureteral stent design and materials. *Curr. Urol. Rep.*, **19**(5): 35.
- Mosayyebi A, Vijayakumar A, Yue QY, Bres-Niewada E, Manes C, Carugo D and Somani BK (2017). Engineering solutions to ureteral stents: Material, coating and design. *Cent. Eur. J. Urol.*, **70**(3): 270–274.
- Mousavi A, Takele R, Limbrick B, Thaker KN and Scotland KB (2024). Oral dissolution therapy of uric acid stones: A systematic review. *Societe Internationale d'Urologie J.*, **5**(4): 284–299.
- Polat H, Yucel MO, Utangac MM, Benlioglu C, Gok A, Cift A, Kalyenci B, Lok U and Gulacti U (2017). Management of forgotten ureteral stents: Relationship between indwelling time and required treatment approaches. *Balk. Med. J.*, **34**(4): 301–307.
- Prochaska M, Taylor E, Ferraro PM and Curhan G (2018). Relative supersaturation of 24-hour urine and likelihood of kidney stones. *J. Urol.*, **199**(5): 1262–1267.
- Ripa F, Pietropaolo A, Montanari E, Hameed BM, Gauhar V and Somani BK (2022). Association of kidney stones and recurrent UTIs: The chicken and egg situation. *Curr. Urol. Rep.*, **23**: 165–174.
- Rodriguez A, Cunha TDS, Rodgers AL, Gambaro G and Ferraro PM (2021). Comparison of supersaturation outputs from different programs and their application in testing correspondence with kidney stone composition. *J. Endourol.*, **35**(5): 687–694.
- Royaux IE, Wall SM, Karniski LP, Everett LA, Suzuki K, Knepper MA and Green ED (2001). Pendrin, encoded by the Pendred syndrome gene, resides in the apical region of renal intercalated cells and mediates bicarbonate secretion. *Proc. Natl. Acad. Sci. U.S.A.*, **98**(7): 4221–4226.
- Safdar OY, Alblowi SS, Aboulola NA and Alharazy DT (2021). Renal stones and risk factors in Jeddah and Riyadh. *Saudi J. Kidney Dis. Transpl.*, **32**: 191–198.
- Scarneci I, Bratu OG, Cobelschi CP, Neculoiu CD, Scarneci CC, Lupu S, Brinza A, Marcu D, Socea B and Maxim L (2018). The risk factors and chemical composition of encrustation of ureteral double J stents in patients with urolithiasis. *Rev. Chim. (Bucharest)*, **69**(12): 3406–3409.
- Scotland KB, Lo J, Grgic T and Lange D (2019). Ureteral stent-associated infection and sepsis: Pathogenesis and prevention: A review. *Biofouling*, **35**(1): 117–127.
- Shastri S, Patel J, Sambandam KK and Lederer ED (2023). Kidney stone pathophysiology, evaluation and management: Core curriculum 2023. *Am. J. Kidney Dis.*, **82**: 617–634.
- Sorokin I, Mamoulakis C, Miyazawa K, Rodgers A, Talati J and Lotan Y (2017). Epidemiology of stone disease across the world. *World J. Urol.*, **35**: 1301–1320.
- Thongprayoon C, Krambeck AE and Rule AD (2020). Determining the true burden of kidney stone disease. *Nat. Rev. Nephrol.*, **16**: 736–746.
- Triozzi JL, Hsi RS, Wang G, Shintani-Smith S, Qian Y, Zhao Y, Akwo EA, Wheless L, Chen HC, Tao R, Ikizler TA, Cohen CR, Hung AM and VA Program MV (2023). Mendelian randomization analysis of genetic proxies of thiazide diuretics and the reduction of kidney stone risk. *JAMA Netw. Open*, **6**(11): e2343290.
- Tsukanov AY, Akhmetov DS, Blesman AI and Rogachev EA (2018). The impact of ureteral stent surface on encrustation and biofilm formation. *Urologia*, **2**: 40–45.
- Ulker V and Celik O (2019). Endoscopic, single-session management of encrusted, forgotten ureteral stents. *Medicina (Kaunas)*, **55**(3): 58.
- Wang X and Wang Q (2024). Current dietary and medical prevention of renal calcium oxalate stones. *Int. J. Gen. Med.*, **17**: 1635–1649.
- Ware JS, Wain LV, Channavajjhala SK, Zweifel B, Clark DW, Edkins S, Jackson VE, Edwards E, Lu R, Siew K, Jia W, Shrine N, Kinnear S, Jalland M, Henry AP, Clayton J, O'Shaughnessy KM, Tobin MD, Schuster VL, Cook S, Hall IP and Glover M (2017). Phenotypic and pharmacogenetic evaluation of patients with thiazide-induced hyponatremia. *J. Clin. Invest.*, **127**(9): 3367–3374.
- Xu J, Barone S, Zahedi K, Brooks M and Soleimani M (2018). Slc4a8 in the kidney: Expression, subcellular

- localization and role in salt reabsorption. *Cell Physiol. Biochem.*, **50**(4): 1361–1375.
- Pearle MS, Goldfarb DS, Assimos DG, Curhan G, Denu-Ciocca CJ, Matlaga BR, Monga M, Penniston KL, Preminger GM, Turk TMT, White JR and American Urological Association (2014). Medical management of kidney stones: AUA guideline. *J. Urol.*, **192**(2): 316–324.
- Griffith DP, Musher DM and Itin C (1978). Urease: The primary cause of infection-induced urinary stones. *Invest. Urol.*, **15**: 346–350.
- Kawahara T, Ito H, Terao H, Ogawa T, Uemura H and Kubota Y (2012). Ureteral stent encrustation, incrustation and coloring: Morbidity related to indwelling time. *J. Endourol.*, **26**(2): 178–182.