International Journal of Community Medicine and Public Health
Alghamdi AM et al. Int J Community Med Public Health. 2025 Jul;12(7):3389-3394
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20251770

Review Article

Management of recurrent urolithiasis: advances in prevention strategies

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Received: 25 May 2025
Accepted: 09 June 2025

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ABSTRACT

Recurrent urolithiasis is a chronic and extremely common condition that is characterized by the development of calculi in the urinary tract. It poses a serious health burden, affecting roughly 10-15% of the world's population and having recurrence rates of over 50% within five years. Therefore, minimizing recurrence, enhancing patient outcomes, and lowering healthcare costs all depend on effective prevention strategies. Conventional preventative strategies emphasize lifestyle modifications like drinking more water, consuming less protein and sodium, and consuming oxalate-rich foods in moderation. Pharmacological strategies such as citrate supplementation, thiazide diuretics, and allopurinol have demonstrated promise; however, new data raises doubts about the effectiveness and adverse effect profiles of some regimens. Additionally, new studies emphasize the significance of tailored interventions based on genetic susceptibility, metabolic assessment, and urine biochemistry. Prospective avenues for the future include probiotics that decrease oxalate absorption, genomic profiling techniques, and engagement of machine learning and algorithms to identify high-risk individuals. This review thoroughly explores both conventional and cutting-edge approaches to the changing field of recurrent urolithiasis prevention. It highlights how important multidisciplinary care, customized treatment planning, and routine follow-up are to the success of stone prevention.

Keywords: Recurrent urolithiasis, Kidney stones, Urolithiasis prevention, Hyperoxaluria, Genetic predisposition, Machine learning

INTRODUCTION

Urolithiasis is a common disorder marked by the development of stones in the urinary tract, most frequently the kidney, due to the urine becoming oversaturated with minerals like calcium oxalate, phosphate, or uric acid.[1] About 1 in 11 people worldwide are afflicted by urolithiasis in their lifetime, which presents with variable degrees of severity and frequency.[2] A longitudinal study conducted in 204 countries stated that the rate of nephrolithiasis increased in most countries.[3,4] Up to 50% of people who get stones end up getting them again within 5-10 years.[5] In addition to causing severe pain and discomfort, recurrent episodes raise the risk of permanent renal damage and raise medical expenses for hospital stays, surgeries, and diagnostic tests.[6,7]

Genetic predispositions, metabolic syndromes, environmental exposures, and dietary factors are all part of the complex pathophysiology of urolithiasis.[8,9] The main causes of stone formation are crystal nucleation, growth, aggregation, retention in the kidneys or lower urinary tract, and urine supersaturation.[10,11] Stones are classified as calcium oxalate, calcium phosphate, uric acid, struvite (infection stones), and cystine stones based on their biochemical makeup.[12,13] Each of these types of stones requires a different approach to treatment.[14] Calcium oxalate stones are the most common kind, with more than 70% of all cases.

Although acute stone removal procedures like ureteroscopy, percutaneous nephrolithotomy, and extracorporeal shock wave lithotripsy (ESWL) are effective, recurrence is still a substantial risk.[15,16] Therefore, after the first stone events, preventive care needs to be given priority. To pinpoint specific risk factors and customize interventions appropriately, urological societies' guidelines stress the significance of comprehensive metabolic workups, which include 24-hour urine testing and stone analysis.[16]

Conventional preventive strategies include limiting dietary sodium and oxalate, consuming animal protein in moderation, and increasing fluid intake to dilute urine.[17,18] Commonly used pharmaceuticals include potassium citrate (for hypocitraturia and uric acid stones), thiazide diuretics (for hypercalciuria), and allopurinol (for hyperuricosuria).[19] But new research has raised questions about the long-term safety and effectiveness of some drugs, indicating the need for a more complex and customized strategy.

New developments in genetics and microbiome research present promising avenues for enhancing prevention.[20] Oxalate has been demonstrated to be broken down by gut bacteria such as *Oxalobacter formigenes*, and genetic profiling may be used to identify patients who are more likely to experience a recurrence.[21,22] Precision medicine in the prevention of stones is made possible by these developments.[23] By highlighting evidence-based recommendations, identifying gaps in current practices, and suggesting future directions, this review aims to assess both established and new approaches for the prevention of recurrent urolithiasis. The review seeks to assist physicians in managing this difficult condition thoroughly and in a patient-centered manner.

LITERATURE SEARCH

A narrative approach was used in this article to investigate both new and current approaches to prevent recurrent urolithiasis. Electronic databases such as PubMed, Scopus and Google Scholar were used to perform a thorough literature search. Only studies written in English and released between 2010 and 2025 were included in the search. “Recurrent kidney stones”, “urolithiasis prevention”, “fluid intake and kidney stones” “dietary oxalate” “citrate therapy “, “thiazide diuretics”, “probiotics and oxalate” “and genetic susceptibility to kidney stones” were among the keywords that were used. RCTs, cohort studies, clinical practice guidelines and systematic reviews and meta-analyses pertaining to adult populations with a documented history of recurrent kidney stones were all involved in the inclusion criteria. Particular focus was placed on research examining risk-stratified or customized approaches as well as studies contrasting traditional and innovative preventative strategies. Using themes such as lifestyle interventions, pharmacological management, gut microbiota modification, and genomic risk stratification data were extracted and synthesized. To highlight current guidelines gaps in the evidence and potential avenues for future research in recurrent stone prevention a qualitative synthesis was conducted instead.

DISCUSSION

Recurrent urolithiasis remains a significant clinical challenge due to its high prevalence, tendency for recurrence, and associated healthcare burden. The risk of recurrence is influenced by a variety of factors, including genetic predisposition, metabolic abnormalities, dietary habits, dehydration, and comorbid conditions such as obesity, hypertension, and diabetes. This recurrent nature not only impacts patients’ quality of life but also contributes to increased healthcare costs due to repeated imaging, surgical interventions, and hospital admissions. Effective management relies heavily on thorough risk assessment and patient stratification to guide individualized preventive strategies.

Dietary and lifestyle modifications

The easiest and most economical way to stop recurrent urolithiasis is to make dietary and lifestyle adjustments. The amount of lithogenic substances in urine is greatly decreased by adequate fluid intake, with a daily urine output of at least 2 to 5 liters as the goal.[24] A Mendelian randomization study showed that patients who continued to consume large amounts of fluid had recurrence rates that were almost 50% lower.[25] Studies indicate that beverages like citrus juices, especially lemonade and orange juice, may also aid in prevention because of their citrate content, which binds calcium and prevents crystal formation.[26] Another important variable that can be changed is sodium intake. By inhibiting proximal sodium and calcium reabsorption, too much sodium in the diet raises urinary calcium excretion.[27,28] This mechanism raises the risk of calcium-based stones and causes hypercalciuria. According to clinical recommendations, sodium consumption should not exceed 2-3 grams per day.[29] Consuming animal protein also raises the acid load, decreases the pH of the urine, elevates the excretion of calcium and uric acid, and lowers protective citrate levels.[30,31] Reducing animal protein intake to less than 0.8-1.0 g/kg/day is therefore frequently recommended.[32] It is especially crucial to manage oxalate in calcium

oxalate stone formers. Oxalate is found in foods like spinach, tea, chocolate, rhubarb, and nuts. When taken in excess, particularly when calcium intake is low, oxalate absorption rises, which encourages the development of stones.[33] Oxalate bioavailability can be decreased, though, if calcium-containing meals are consumed with oxalate-rich foods. Another layer of complexity is added by emerging evidence that suggests the composition of the gut microbiota as a whole may have an impact on gut oxalate metabolism.[34] Lastly, stone risk is independently correlated with metabolic syndrome, obesity, and a sedentary lifestyle. Higher lithogenic substance excretion is correlated with higher body mass index (BMI).[35] Thus, it may be protective to promote physical activity, weight loss, and dietary patterns like the DASH or Mediterranean diets.[36]

Pharmacologic methods

When a 24-hour urine analysis reveals metabolic abnormalities in a patient, pharmacologic therapy is usually indicated. These drugs work particularly well when combined with lifestyle modifications. Thiazide diuretics such as chlorthalidone or hydrochlorothiazide lower calcium levels in the urine by encouraging distal tubular reabsorption. For idiopathic hypercalciuria, they are the first line of treatment.[37] However, prolonged use may result in dyslipidemia, hypokalemia, and glucose intolerance.[38] Recent experiments (e. g., the NOSTONE study), its effectiveness might rely on assessing the effectiveness of different hydrochlorothiazide doses in preventing kidney stones.[39] Another popular treatment, especially for hypocitraturia or acidic urine, is potassium citrate.[40] It is especially useful in preventing calcium and uric acid stones because it not only raises urine citrate but also alkalinizes the urine.[41] Potassium citrate improves bone mineral density, which is frequently weakened in recurrent stone formers, and lowers recurrence rates.[42] Particularly for calcium oxalate stone formers with elevated uric acid, allopurinol is helpful in cases of hyperuricosuria. By blocking xanthine oxidase, it reduces the production of uric acid. Although it may pose cardiovascular risks to some populations, febuxostat is an alternative for patients who are unable to tolerate allopurinol. Acetohydroxamic acid, a urease inhibitor that lowers ammonia production, may be necessary for patients with infection-related struvite stones. But only refractory cases can use it due to its side effects, which include neurotoxicity and gastrointestinal intolerance. Patient adherence, electrolyte levels, and kidney function must all be closely monitored during long-term medication treatment. For high-risk stone formers, the best course of action continues to be a combination of pharmacological and nutritional approaches.

Emerging personalized therapies

Customized treatment regimens based on microbial genetic and biochemical profiling have gained attention as a result of recent developments in our understanding of the pathophysiology of stone disease. One area of significant research interest is the function of gut microbiota in oxalate metabolism. In the colon, oxalate is broken down by the gram-negative anaerobe *Oxalobacter formigenes*, hyperoxaluria is linked to its absence, which is frequently brought on by the use of antibiotics or dietary habits.[43,44] Probiotic supplementation of formigenes targeting oxalate degradation is under investigation, with early-phase trials indicating promise for more recent bacterial restoration therapies.[45,46] In the future, engineered probiotics might provide a sustainable and safe way to lower urine oxalate levels.[47,48]

More is being learned about the genetic predisposition to stone formation. Variants in genes that affect renal calcium phosphate and oxalate handling including CLDN14, SLC26A1 and SLC34A1, have been found by genome-wide association studies (GWAS).[49,50] Individualized treatments and early genetic diagnosis are beneficial for patients with monogenic disorders like cystinuria or primary hyperoxaluria.[51] Additionally, polygenic risk scores may eventually direct to the intensity of the preventive strategies in populations that are at risk. Predicting the recurrence of stones is being investigated using artificial intelligence and machine learning.[52-54] It has been demonstrated that algorithms trained on sizable datasets that include biochemical clinical and demographic factors can accurately predict which patients will benefit from particular treatments.[55,56] By offering customized risk profiles these models may support clinical decision-making and enhance patient involvement.[57,58] Furthermore, new treatments that use enzymes that break down oxalate (e. g. oxalate decarboxylase) and RNA-based treatments for monogenic disorders, and targeted transport inhibitors are being developed.[59,60] The prevention landscape may change over the next ten years as a result of these tactics.

CONCLUSION

Since recurrent urolithiasis is a complex condition, prevention must be both comprehensive and tailored to each patient. Lifestyle changes remain fundamental, especially proper hydration, normalizing calcium intake, reducing sodium and oxalate, and taking supplements of citrate. Although pharmacological treatments like citrate therapy, allopurinol, and thiazides are useful, they must be customized according to risk profiles and metabolic abnormalities. These new strategies could improve treatment regimens and more successfully lower recurrence. Clinical integration, cost-effectiveness analyses, and additional research are necessary for broad adoption, though. Precision medicine, better diagnostic instruments, and digital health initiatives that improve patient education and adherence should be the main focuses of future research. In nephrology and urology practice, maximizing prevention will continue to be a top priority due to the rising incidence of kidney stone disease worldwide.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Alghamdi AM, Alqurashi EA, Alsubaie AT, Alamri AM, Alamri AM, Abdulmajeed AH, et al. Management of recurrent urolithiasis: advances in prevention strategies. Int J Community Med Public Health 2025;12:3389-94.