Journal Homepage: www.ijarpr.com ISSN: 3049-0103 (Online)

International Journal of Advance Research Publication and Reviews

Vol 03, Issue 10, pp 285-290, October, 2026

Urolithiasis: A Short Review

Sayoojya R [1], Jayachandran T P [2], Dr. Jyoti Harindran [3]

[1]Final year M. Pharm student, Department of Pharmacology, Department of Pharmaceutical Sciences, Department of Pharmaceutical Sciences Rd, Cheruvandoor, Ettumanoor, Kerala -686631, India.
[2] Associate Professor, Department of Pharmacology, Department of Pharmaceutical Sciences, Department of Pharmaceutical Sciences Rd, Cheruvandoor, Ettumanoor, Kerala -686631, India.
[3]Principal, Department of Pharmacology, Department of Pharmaceutical Sciences, Department of Pharmaceutical Sciences Rd, Cheruvandoor, Ettumanoor, Kerala -686631, India.

ABSTRACT :

Urolithiasis, commonly known as kidney stone disease, is a significant global health concern characterised by the formation of polycrystalline concretions within the urinary tract. Its prevalence is increasing worldwide, driven by lifestyle factors such as diet, obesity, and environmental changes. The disease is multifactorial, involving a complex interplay of genetic predisposition, metabolic abnormalities, and dietary habits. Diagnosis primarily relies on advanced imaging techniques like non-contrast computed tomography, while management ranges from medical expulsive therapy to minimally invasive surgical procedures. Prevention remains a cornerstone of care, emphasising hydration, dietary modification, and metabolic evaluation to reduce the high recurrence rate associated with the condition.

Keywords: Urolithiasis; kidney stones; nephrolithiasis; renal colic; urinary obstruction; lithotripsy; ureteroscopy; stone prevention.

INTRODUCTION

Urolithiasis is one of the most common disorders affecting the urinary tract and represents an important cause of acute pain, emergency consultation, hospital admission, and urological intervention. The term includes stone formation anywhere along the urinary tract, but the majority of clinically relevant stones are located in the kidneys or ureters [1, 2]. Although a stone may remain silent for a prolonged period, migration into the ureter can produce acute obstruction and severe renal colic. The clinical burden therefore ranges from an incidental, asymptomatic renal calculus to complicated disease involving infection, hydronephrosis, obstruction, or deterioration of renal function [2, 3].

The development of a urinary stone is not explained by a single abnormality. It is the result of a multifactorial process involving urinary supersaturation with calcium, oxalate, phosphate, uric acid, or cystine; crystal nucleation; crystal growth and aggregation; and eventual retention within the renal tubules or urinary tract [4-8]. These physicochemical processes are influenced by urine volume, urinary pH, dietary intake, metabolic abnormalities, genetic predisposition, urinary infection, anatomical conditions, and environmental exposure. Protective substances such as citrate and other urinary inhibitors may reduce crystallisation, whereas dehydration, hypercalciuria, hyperoxaluria, hypocitraturia, hyperuricosuria, and low urinary volume increase the likelihood of stone formation [5, 9].

The epidemiology of urolithiasis differs between populations and is affected by climate, ethnicity, heredity, occupation, socioeconomic conditions, dietary patterns, and access to medical care [1, 10]. The disease has become increasingly relevant in both developed and developing regions. Warmer temperatures and recurrent dehydration may increase urinary concentration, while obesity, metabolic syndrome, high sodium intake, and increased consumption of animal protein may promote specific metabolic abnormalities associated with stone formation [10, 11]. Although men have traditionally been affected more often, the sex gap has narrowed in several populations, partly because of changing lifestyle patterns and increasing obesity among women [12, 3].

Urolithiasis is also a recurrent disease. Removal of an existing stone does not necessarily correct the metabolic or environmental conditions that caused it. Consequently, long-term care should include not only treatment of the acute episode but also identification of the stone type, evaluation of recurrence risk, correction of dietary and metabolic abnormalities, and individualised preventive therapy [5, 13, 14]. This short review summarises the definition, epidemiology, pathogenesis, clinical presentation, diagnostic approach, treatment options, and preventive principles of urolithiasis.

DEFINITION

Urolithiasis is the formation of solid crystalline deposits or polycrystalline concretions, commonly called urinary stones or uroliths, within the urinary tract [1, 2]. Stones may arise in the renal calyces, renal pelvis, ureters, urinary bladder, or urethra, although the kidneys and ureters are the most frequent

clinically significant sites [2]. Most stones contain a mixture of crystalline and organic material, and their final composition depends on urinary chemistry, infection, diet, inherited disorders, and local urinary conditions [1, 5].

Stone disease is commonly classified according to composition. Calcium oxalate and calcium phosphate stones are the most frequent, while uric acid, struvite, and cystine stones occur in characteristic metabolic, infectious, or inherited settings [1, 4]. This classification is clinically important because stone composition helps explain the cause of disease and guides dietary advice, medical treatment, and prevention of recurrence. Uroliths can be regarded as urinary biominerals; however, unlike normal mineralized tissues, their formation reflects abnormal supersaturation, crystallization, aggregation, and retention rather than organized physiological development [1].

EPIDEMIOLOGY

The epidemiology of urolithiasis varies considerably between countries and regions. Climate, temperature, humidity, ethnicity, heredity, occupation, diet, body weight, and access to diagnosis all influence reported incidence and prevalence [1, 10]. Prevalence has increased in many populations over recent decades, partly because of changing dietary habits, increasing obesity and metabolic syndrome, improved imaging, and greater recognition of asymptomatic stones [1, 10].

A higher burden has traditionally been reported in warmer regions, sometimes described as a “stone belt,” because heat exposure and inadequate fluid replacement reduce urine volume and increase urinary concentration [1]. However, climate alone does not explain regional differences; dietary sodium, animal-protein intake, calcium and oxalate consumption, genetic background, and socioeconomic conditions also contribute. The disease commonly affects adults between approximately 25 and 55 years of age, although children and older adults may also develop stones [11, 15].

Men have historically shown a higher prevalence than women, but the difference has narrowed in several populations. Increasing obesity, metabolic syndrome, dietary change, and other shared risk factors may contribute to the growing burden among women [12, 3]. Occupational dehydration may further increase risk in people who work for long periods in hot environments or cannot drink water regularly. These epidemiological patterns emphasise that urolithiasis is influenced by both biological susceptibility and modifiable lifestyle conditions.

PATHOGENESIS AND RISK FACTORS

The pathogenesis of urolithiasis is a stepwise physicochemical process that begins when urine becomes supersaturated with stone-forming substances such as calcium, oxalate, phosphate, uric acid, or cystine [4, 5]. Supersaturation permits crystal nucleation, after which crystals may enlarge, aggregate, attach to epithelial surfaces, and become retained within renal tubules or the urinary tract [6-8]. Adequate urine volume and crystallization inhibitors, particularly citrate, help maintain solubility and reduce crystal growth. In contrast, dehydration, low urine volume, altered urinary pH, and increased excretion of lithogenic solutes favor stone formation [5, 9].

Stone formation also depends on the interaction between crystals and the urinary epithelium. Crystals that are rapidly washed out may never become clinically important, whereas crystals that adhere to tissue or become trapped in anatomical sites can enlarge and form a symptomatic calculus. Urinary infection may alter pH and promote the formation of infection-related stones, while obstruction and urinary stasis can further support stone growth and recurrent infection [1, 16]. Genetic factors may influence renal tubular transport, urinary calcium and oxalate handling, cystine excretion, and the activity of crystallization inhibitors [5].

Important modifiable risks include inadequate fluid intake, high sodium intake, excessive animal-protein consumption, obesity, and dietary patterns that increase urinary calcium, oxalate, uric acid, or reduce citrate [11, 9]. Non-modifiable or less-modifiable risks include family history, inherited disorders such as cystinuria, recurrent urinary infection, congenital or acquired urinary tract abnormalities, and previous stone formation. A previous stone episode is itself a major indicator of future recurrence, which is why prevention should begin after the first clinically significant episode rather than only after repeated disease.

STONE TYPES AND MECHANISMS

- Calcium oxalate stones: These are the most common stones and are frequently associated with hypercalciuria, hyperoxaluria, low urinary citrate, or reduced urine volume [1, 5, 17]. Adequate dietary calcium is generally important because it can bind intestinal oxalate; therefore, unnecessary calcium restriction may increase oxalate absorption in some patients.
- Uric acid stones: These are favored by persistently acidic urine, particularly a urine pH below approximately 5.5, and may be associated with hyperuricosuria, obesity, insulin resistance, or a purine-rich diet [4, 9, 18]. Urinary alkalinization is an important principle in their prevention and treatment.
- Struvite stones: These infection-related stones develop in association with urease-producing organisms. They may grow rapidly, occupy a large portion of the renal collecting system, and recur if infection and residual stone fragments are not adequately managed [1, 16].

- Cystine stones: These result from cystinuria, an inherited defect in renal tubular transport. They often occur at a younger age and have a high tendency to recur, requiring lifelong preventive care [14].

Stone composition should be determined whenever a stone is retrieved because composition provides information about the underlying mechanism and helps personalize preventive treatment. Infrared spectroscopy and X-ray diffraction are useful methods for laboratory characterization of stone material [5, 19].

MODIFIABLE AND NON-MODIFIABLE RISK FACTORS

Modifiable risk factors include low fluid intake, high sodium intake, excessive animal-protein consumption, obesity, sedentary lifestyle, and dietary patterns that increase urinary oxalate or uric acid [1. 9, 11]. Environmental heat exposure and dehydration increase urinary concentration and may trigger stone formation in susceptible individuals [1]. Non-modifiable factors include family history, inherited transport disorders, previous stone disease, and anatomical abnormalities. Genetic variants in pathways involving calcium, oxalate, and cystine transport may also alter individual susceptibility [5].

CLINICAL PRESENTATION

Urolithiasis may remain asymptomatic for a long period, particularly when a stone is small and non-obstructing [1]. Symptoms commonly develop when a stone moves into the ureter. The typical manifestation is renal colic: sudden, severe, cramping flank pain that may radiate toward the lower abdomen, groin, or genital region [3]. The patient may be restless because the pain is difficult to relieve by changing position. Hematuria is common, although its absence does not exclude a stone.

Associated symptoms include nausea, vomiting, dysuria, urinary frequency, and urgency, particularly when the stone is located near the vesicoureteric junction [17]. Fever, chills, rigours, marked malaise, or systemic toxicity raise concern for urinary infection behind an obstruction. An obstructed infected system is a urological emergency and requires urgent clinical assessment and drainage rather than observation alone [2, 20]. Some stones are detected incidentally during imaging performed for unrelated conditions, while long-standing obstruction may present with hydronephrosis or impaired renal function [1, 2].

DIAGNOSIS

The diagnostic approach combines history-taking, physical examination, laboratory testing, imaging, and, when appropriate, metabolic evaluation. Important clinical questions include the onset and radiation of pain, previous stones, family history, fluid and dietary habits, medication use, urinary infection, pregnancy status, and symptoms suggesting obstruction or sepsis.

- Imaging: Non-contrast helical computed tomography is highly sensitive for detecting urinary calculi and defining their size, location, density, and degree of obstruction [17, 20, 21]. It is particularly useful when the diagnosis is uncertain or when complications are suspected. Ultrasonography is commonly used as an initial modality and is preferred in children and pregnant patients because it avoids ionizing radiation [15, 17, 20]. Plain radiography may detect some radiopaque stones but is less sensitive and is not sufficient as a stand-alone test.
- Laboratory analysis: Urinalysis can identify hematuria, pyuria, crystalluria, urine pH, and possible infection. Urine culture is important when infection is suspected. A complete blood count and serum biochemical tests, including creatinine, electrolytes, calcium, and uric acid when indicated, help assess systemic illness, renal function, and metabolic risk [18, 22].
- Metabolic workup: Patients with recurrent stones, multiple stones, bilateral disease, early-onset disease, a family history, or high-risk stone composition may benefit from a detailed metabolic evaluation. A 24-hour urine collection can assess urine volume, calcium, oxalate, citrate, uric acid, sodium, and pH, thereby identifying abnormalities such as hypercalciuria, hyperoxaluria, hypocitraturia, or hyperuricosuria [5, 23, 24]. Stone analysis should be combined with this evaluation whenever possible [5, 19].

MANAGEMENT

Management should be individualized according to stone size, location, composition, symptoms, obstruction, infection, renal function, patient preference, and the likelihood of spontaneous passage. The immediate priorities are relief of pain, assessment for infection or renal impairment, and identification of patients who require urgent decompression. A small, uncomplicated stone may be managed conservatively, whereas persistent obstruction, uncontrolled pain, infection, renal impairment, or a low probability of spontaneous passage may require active intervention [20, 17].

MEDICAL MANAGEMENT

- Pain control: Non-steroidal anti-inflammatory drugs are commonly used as first-line treatment for acute renal colic when renal function and other clinical factors permit. Additional analgesia and antiemetic therapy may be required for severe pain or vomiting [20, 17].

- Observation and spontaneous passage: Selected patients with small ureteral stones can be observed with adequate analgesia, hydration according to thirst and clinical status, and follow-up imaging. Observation is inappropriate when there is infection, progressive renal impairment, persistent obstruction, or uncontrolled symptoms.
- Medical expulsive therapy: Alpha-blockers such as tamsulosin may facilitate passage of selected distal ureteral stones by reducing ureteral smooth-muscle tone [4, 15, 18]. The expected benefit depends on stone size, location, and patient factors, so treatment should be individualized rather than used automatically for every stone.
- Treatment of infection: Antibiotics are required when infection is present, but antibiotics alone are not sufficient for an infected obstructed system. Such patients require urgent urological assessment and drainage [2, 20].
- Pharmacotherapy: After metabolic evaluation, agents such as thiazide diuretics, potassium citrate, or allopurinol may be used for selected recurrent stone formers. Emerging therapies, including SGLT2 inhibitors and other compounds, remain areas of investigation [13, 25].

SURGICAL AND MINIMALLY INVASIVE INTERVENTIONS

Active stone removal is considered when a stone is unlikely to pass, causes repeated symptoms, produces obstruction, is associated with infection, enlarges during surveillance, or threatens renal function. The choice of procedure depends on stone burden, location, composition, urinary anatomy, comorbidities, available expertise, and patient preference.

- Extracorporeal shock-wave lithotripsy: Shock waves fragment a stone into smaller pieces that can pass through the urinary tract. It is less invasive but may require more than one treatment and may be less effective for some large, hard, or unfavourably located stones [7, 18, 20].
- Ureteroscopy: A small endoscope is passed through the urinary tract to visualise, fragment, and remove ureteral or renal stones. It provides direct treatment and is widely used for stones in several locations [18, 20, 25].
- Percutaneous nephrolithotomy: This procedure removes larger or complex renal stones through a percutaneous tract created in the back. It is particularly useful for a high stone burden, including some staghorn calculi [4, 18, 20].
- Open surgery: Open procedures are now uncommon because of advances in endourology and shock-wave treatment, but they may remain relevant in complex cases or settings where modern equipment is unavailable [3].

PREVENTION

Prevention is essential because urolithiasis frequently recurs and stone removal does not eliminate the underlying tendency to crystallize. Preventive care should be based on stone composition, serum studies, urine chemistry, dietary assessment, medication review, and the patient's recurrence risk [5, 14].

- Hydration: A high fluid intake distributed throughout the day is the central preventive measure. The practical goal is to maintain a urine output of approximately 2.0 to 2.5 liters per day, adjusted for climate, activity, body size, and medical conditions [5, 14, 17]. Water is generally preferred, and additional attention is needed during hot weather, exercise, fever, vomiting, or occupations involving heat exposure.
- Dietary modification: Reducing dietary sodium can help lower urinary calcium excretion. Excessive animal protein should be limited, particularly in patients with hyperuricosuria or recurrent calcium stones. Adequate dietary calcium should usually be maintained rather than severely restricted, while oxalate-rich foods and purine-rich foods should be modified according to stone composition and urine findings [5, 9, 17].
- Weight and lifestyle: Gradual weight control, regular physical activity, and reduction of highly processed foods may improve the metabolic environment associated with stone formation. Individual advice is preferable to unnecessarily restrictive diets.
- Pharmacological prevention: Thiazide diuretics may be considered for selected patients with recurrent calcium stones and hypercalciuria. Potassium citrate may be useful in hypocitraturia and some uric-acid or calcium-stone phenotypes, while allopurinol may be considered for selected patients with hyperuricosuria [5, 14, 18]. Treatment should be monitored with clinical review and, when appropriate, repeat urine testing.
- Follow-up: Follow-up imaging can assess residual or recurrent stones, and repeat metabolic assessment can determine whether the preventive plan is effective. Patients should be encouraged to seek early medical attention for fever, persistent flank pain, vomiting, reduced urine output, or urinary symptoms suggestive of obstruction or infection.

CONCLUSION

Urolithiasis is a common and recurrent multifactorial disease produced by the interaction of urinary chemistry, crystallisation processes, genetic susceptibility, infection, anatomy, diet, hydration, and environmental exposure. Its manifestations range from an incidental asymptomatic stone to severe renal colic, obstructive uropathy, infection, and renal impairment. Accurate diagnosis requires clinical assessment, appropriate imaging, laboratory testing,

and, in selected patients, stone analysis and metabolic evaluation.

Modern management combines analgesia and observation for suitable uncomplicated stones with medical expulsive therapy and minimally invasive procedures when indicated. However, successful acute treatment should be followed by recurrence prevention. Adequate fluid intake, balanced dietary modification, correction of metabolic abnormalities, targeted pharmacotherapy, and regular follow-up provide the foundation of long-term care. A personalised approach is therefore more appropriate than a uniform diet or treatment plan for every patient.

REFERENCES

1. Fisang C, Anding R, Müller S, Latz S, Laube N. Urolithiasis--an interdisciplinary diagnostic, therapeutic and secondary preventive challenge. *Deutsches Arzteblatt International*. 2015-02-06;112(6):83-91. doi:10.3238/ARZTEBL.2015.0083.
2. Eads ED, Herbst KA, Lee J. Diagnosis and treatment of urolithiasis. *Advanced Emergency Nursing Journal*. 2007-04-01;29(2):98-110. doi:10.1097/01.TME.0000270331.50767.EB.
3. Hounnasso PP, Avakoudjo JDG, Paré AK, Brahima K, Ouattara A, Agounkpe MM, et al.. Symptomatic Urinary Lithiasis: Epidemiology and Management at Urology Department of University Hospital of Cotonou. *Open Journal of Urology*. 2015-02-06;05(2):7-12. doi:10.4236/OJU.2015.52002.
4. Назаров ТХ, Ахмедов МА, Рычков ИВ, Трубникова КЕ, Николаев ВА, Турсунов АИ. Мочекаменная болезнь: этиопатогенез, диагностика и лечение. 2019-09-30;20(3):43-51. doi:10.17650/2070-9781-2019-20-3-43-51.
5. Krasiuk IV, Hrydniev A, Pylypenko Y, Krasiuk IV, Hrydniev A, Pylypenko Y. A modern view on the prevention and metaphylaxis of urolithiasis. *Počki*. 2025-05-27;14(2):161-167. doi:10.22141/2307-1257.14.2.2025.515.
6. Recinos PJM. Possible Complications Associated to Renal Litiasis, A Brief Review. 2025-06-25. doi:10.5281/zenodo.15735535.
7. Urolithiasis: Advances in Pathophysiology, Microbiome Role, Immune Pathways, and Evolving Diagnostic and Therapeutic Strategies for Kidney Stone Formation and Management. 2025-05-20. doi:10.5281/zenodo.15473205.
8. Goswami PK. Urolithiasis: An Overview. *International Journal of Pharmaceutical & Biological Archive*. 2014-04-18;4(6).
9. Sowtali SN, Faizal NFFM, Baharudin AS. Knowledge, Awareness and Practice on Dietary Management Among Patients with Urolithiasis: A Scoping Review. *International Journal of Care Scholars*. 2022-07-31;5(2):126-132. doi:10.31436/ijcs.v5i2.252.
10. Yasui T, Ando R, Okada A, Tozawa K, Iguchi M, Kohri K. Epidemiology of urolithiasis for improving clinical practice. *Hinyokika kiyo. Acta urologica Japonica*. 2012-12-01;58(12):697-701.
11. Mishra V, Singh AP. A Study on Risk Factors among Patients Suffering Urolithiasis. *International Journal Of Medical Science And Clinical Invention*. 2018-03-01;5(3):3580-3582. doi:10.18535/IJMSCI/V5I3.03.
12. Gomase PV, Pawar SP. Urolithiasis (kidney stones) current pharmacological diagnosis and management. *Journal of Drug Delivery and Therapeutics*. 2019-07-18;9(4):726-737. doi:10.22270/jddt.v9i4.3219.
13. Kapil S, Sharma S, Bala K, Gupta A. Recent Advancements in the Management of Nephrolithiasis: A Comprehensive Review. 2025-09-07. doi:10.5281/zenodo.17072475.
14. Zisman AL. Effectiveness of Treatment Modalities on Kidney Stone Recurrence. *Clinical Journal of The American Society of Nephrology*. 2017-10-06;12(10):1699-1708. doi:10.2215/CJN.11201016.
15. Kyarimov IA. Urolithiasis in children: modern possibilities of diagnosis and treatment. *Rossijskij pediatričeskij žurnal*. 2023-06-27;26(3):218-221. doi:10.46563/1560-9561-2023-26-3-218-221.
16. Wallace BK, Chmiel JA, Al KF, Bjazevic J, Burton JP, Goldberg H, et al.. The Role of Urinary Modulators in the Development of Infectious Kidney Stones. *Journal of Endourology*. 2022-12-23. doi:10.1089/end.2022.0458.
17. Current Approaches to the Etiology, Prevention, Diagnosis and Treatment of Nephrolithiasis: A Review of Recent Literature. *Formosa Journal of Multidisciplinary Research*. 2025-10-27;4(10):4973-4990. doi:10.55927/fjmr.v4i10.554.
18. Sharanya NKYPS. Evaluation And Management Of Urolithiasis. 2023-09-13. doi:10.5281/zenodo.8339887.
19. Šerić V, Babić-Ivančić V, Kuveždić H, Tucak A. Analiza sastava bubrežnih kamenaca FTIR spektroskopijom: dvadesetogodišnje iskustvo. 2010-12-01;42:285-288.
20. Wróblewski K, Wróblewska P, Szukalska S, Karczewska M, Lichwala K, Samborska A, et al.. Current Perspectives on Urolithiasis: Pathogenesis, Clinical Management, and Treatment. *Cureus*. 2026-01-09. doi:10.7759/cureus.101141.
21. Oliveira RGd, Souza MCd. Urolitíase: revisão da literatura. *Revista Ibero-Americana de Humanidades, Ciências e Educação*. 2022-07-30;8(7):1157-1165. doi:10.51891/rease.v8i7.6400.
22. Girase CJ, Deore HV. Modern Pathogenetic Aspects and Updates on Diagnostic Modalities of Urolithiasis Development: A Review. *Asian Journal of Research in Pharmaceutical Science*. 2025-10-04:418-422. doi:10.52711/2231-5659.2025.00062.

23. Naeem M, Ahmad T, Khan MK, Ullah H. Gender and age distribution, and urinary metabolic abnormalities in patients with recurrent urinary tract stones. *Gomal Journal of Medical Sciences*. 2015-06-30;13(2).

24. Bandi G, Nakada SY, Penniston KL. Practical approach to metabolic evaluation and treatment of the recurrent stone patient. *WMJ : official publication of the State Medical Society of Wisconsin*. 2008-04-01;107(2):91-100.

25. Ramadhan R, Wijaya MP, Hidayat DR. Advances in Urology: A Comprehensive Literature Review on Urolithiasis Management and Innovations. *Brawijaya Journal of Urology*. 2025-07-01;6(01):11-19. doi:10.11594/bjurology.2025.006.01.3.