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Urolithiasis Revisited: An Updated Comprehensive Review with Case Illustrations

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Abstract

Urolithiasis is the most prevalent global urological disorder characterized by the formation of urinary calculi resulting from super saturation and crystallization of minerals such as calcium oxalate, calcium phosphate, uric acid, struvite and cystine. Its incidence varies significantly across regions due to dietary habits, climate, genetic predisposition and metabolic abnormalities. This review aims to provide an updated understanding of the epidemiology, chemical types and etiological contributors of urolithiasis, while also outlining the underlying mechanisms involved in stone formation. The objectives include describing the key physicochemical events super saturation, nucleation, crystal growth, aggregation and retention and outlining the major concepts that explain how these early crystalline changes progress within the urinary tract. These processes reflect the dynamic interaction between metabolic abnormalities, urinary pH variations, epithelial responses and the accumulation of crystalline particles, ultimately contributing to the formation and persistence of urinary stones. Clinically, urolithiasis commonly presents with acute renal colic, hematuria, nausea and vomiting, with symptom severity depending on stone size and location.

Although diagnostic and therapeutic advances have improved outcomes, recurrence remains high. Future perspectives in this field involve shifting toward precision-based prevention, using metabolic profiling, stone-specific interventions and novel research areas including microbiome dynamics, genetic markers and crystallization-modulating agents to achieve more sustained control of stone recurrence. To strengthen the clinical relevance of this review, two case studies are included to demonstrate typical presentations of renal and ureteric calculi and to emphasize the practical aspects of diagnosis, imaging interpretation and early therapeutic decision making.

Keywords: Urolithiasis; Nephrolithiasis; Urinary Stones; Epidemiology; Pathophysiology; Super Saturation; Randall's Plaque; Recurrence; Future Perspectives

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Abbreviations

Hypertension (HTN), American Urological Association (AUA), European Association of Urology (EAU), Calcium oxalate (CaOx), Calcium phosphate (CaP), ureterovesical junction (UVJ), lower urinary tract symptoms (LUTS), Estimated Glomerular Filtration Rate (eGFR), complete blood count (CBC), Computed Tomography of Kidneys, Ureters and Bladder (CT KUB), Magnetic Resonance Imaging (MRI), Computed Tomography (CT), Medical Expulsive Therapy (MET), Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), acetohydroxamic acid (AHA), Extracorporeal shock wave lithotripsy (ESWL), Ureteroscopy (URS), Holmium:Yttrium Aluminum Garnet (Ho:YAG), Percutaneous Nephrolithotomy (PCNL), Unique Health Identification (UHID), Sri Venkateswara Institute of Medical Sciences (SVIMS).

Introduction

Urolithiasis is the most prevalent urological condition which is commonly known as urinary calculi [1]. Primarily stones are formed with in the kidneys which are known as Nephrolithiasis, while the passage of stones from renal pelvis to ureter (ureterolithiasis) bladder (cystolithiasis) and urethra (urethrolithiasis) is known as urolithiasis [2]. Urinary stones are caused by various factors

but primary one is building up of calcium phosphates and oxalates in the urinary tract system. These are the most prevalent types, accounting for nearly 80% of all kidney stones. Other types of urinary stones include uric acid and struvite stones form about 9% and 10% while urate, ammonium acid and cystine stones make up roughly 1%. Uric acid stones are primarily composed of uric acid ($C_5H_4N_4O_3$), while struvite stones consist of magnesium ammonium phosphate ($Mg(NH_4)PO_4{\cdot}6H_2O$). Urate stones are formed from sodium or potassium urate salts, ammonium acid urate stones are made up of ammonium acid urate ($NH_4C_5H_3N_4O_3$), and cystine stones originate from cystine ($C_6H_{12}N_2O_4S_2$) [1, 3].

The geographical distribution of urolithiasis shows variation with western countries generally exhibiting higher incidence rates than those in eastern countries due to dietary factors (rich animal protein diet), lifestyle factors, climate, genetic and metabolic predisposition [4]. The global incidence and recurrence of urolithiasis are steadily increasing, despite the scarcity of effective therapeutic options. Incidence rates are influenced by factors such as location, climate, ethnicity, dietary patterns and genetic factors. Approximately 12% of world's population is affected during their life time [5, 49, 50]. Kidney stone formation is most prevalent in men aged 20–49 years. Inadequate preventive interventions can result in recurrence rates approaching 75% within two decades [5]. Despite a higher lifetime risk among men, incidence among women has significantly increased, reducing the male-to-female ratio from 3:1 to 2:1, a trend linked to the growing prevalence of obesity and diabetes in women. In India, around 12% of the population is prone to urolithiasis, with higher prevalence in the northwestern "stone belt" states like Gujarat, Rajasthan, Punjab, Haryana, Maharashtra, and Delhi. Regional studies, such as in Manipur showing 22.4% prevalence, highlight significant geographical variation influenced by climate, diet, and lifestyle factors [6, 51, 52].

Urolithiasis is linked to a higher risk of cardiovascular disease, including Hypertension (HTN) and increased carotid wall thickness, even after adjusting for other risk factors. Studies shows a 3%higher risk of myocardial infarction in patients. It is strongly associated with low bone mineral density and fractures due to excess calcium loss from bones through urine [7, 53, 54]. The American Urological Association (AUA) and the European Association of Urology (EAU) are key organizations that establish evidence-based clinical guidelines for the management of urolithiasis. These associations aim to advance research, provide education and enhance public awareness, influence healthcare policy development, and uphold high standards of clinical practice and patient care.

Types of Urolithiasis

Urinary tract stone disease encompasses a spectrum of calculi differentiated primarily by their chemical composition and underlying lithogenic mechanisms [8, 55, 56]. The predominant category, calcium-based stones, comprises roughly 70-80% of cases in many series, with Calcium oxalate (CaOx) being the most frequent, followed by Calcium phosphate (CaP) and mixed forms [9, 10]. CaOx stones arise in the context of hypercalciuria, hyperoxaluria, hypocitraturia or low urine volume, and may form monohydrate or dihydrate crystalline phases, each with distinct morphological features and recurrence risk. CaP stones tend to form in more alkaline urine and are associated with disorders such as renal tubular acidosis and hyperparathyroidism [11].

Beyond calcium-based calculi lie non-calcium types, each with unique pathophysiology and clinical implications. **Uric acid stones** represent approximately 5-10% of cases and form in persistently acidic urine, often in association with obesity, metabolic syndrome, gout or high purine intake [11]. These stones are radiolucent and amenable to dissolution via urinary alkalinization. Struvite stones (magnesium ammonium phosphate) often develop in the setting of urease-producing urinary tract infections (e.g., Proteus mirabilis), tend to grow rapidly, and may present as large staghorn calculi requiring infection control plus surgical management [10, 11]. Cystine stones, although rare (≈1% of stones), arise in the hereditary disorder cystinuria, wherein impaired proximal tubular reuptake of cystine leads to high urinary cystine concentration, crystallization and recurrent stone formation [10, 12].

Aetiology

Urinary tract infections, insufficient urine excretion, and nutritional imbalances caused by high calcium and oxalates ingestion are all factors that might lead to stone formation. Vitamin deficiencies or excess vitamin D, as well as metabolic illnesses such as hyperthyroidism, cystinuria, gout, and intestinal dysfunction, can all cause kidney stones and urolithiasis. This complex system results from an imbalance between the kidney's promoters and inhibitors. The various risk factors will cause urolithiasis explained below,

Metabolic factors: Variations in urine composition will leads to crystal formation that accumulates into stones. Factors contributing to stone formation include hypercalciuria (increased levels of calcium in urine), hypercitraturia (increased levels of citrate), hyperoxaluria (increased levels of oxalate) and hypocitraturia (decreased levels of citrate).

Genetic Predisposition: Kidney stones are more likely to form in people with certain hereditary abnormalities such cystinuria and hyperoxaluria. These disorders have an impact on the metabolism of chemicals like oxalate and cystine, increasing the likelihood that their excretion may result in stones.

Environmental Factors: Environmental factors such as climate (hot and dry climates can cause dehydration and concentrated urine), occupation (including exposure to particular chemicals), and geographical location (areas with high occurrence rates due to water composition) all contribute to stone formation.

Dietary Factors: A high diet of oxalate-rich foods (such as spinach and almonds), purine-rich meals (which raise uric acid levels), and sodium can all contribute to stone formation. Inadequate fluid intake reduces urine volume, which increases the concentration of stone-forming chemicals.

Anatomical Factors: Structural abnormalities in the urinary tract, such as narrow ureters or kidney malformations, can obstruct urine flow and promote stone formation.

Medical Conditions: Certain medical problems, such as recurrent urinary tract infections, inflammatory bowel disease, and metabolic diseases like hyperparathyroidism, enhance the likelihood of kidney stone formation [57, 58].

Specific Etiological Conditions Related to Types of Urolithiasis

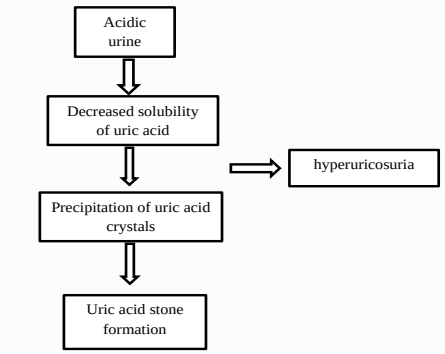
Struvite stones typically form in alkaline urine secondary to infections with urease producing bacteria particularly *proteus* and

klebsiella. Uric acid stones arise under conditions of persistently acidic urine. Cystine stones arise from inherited cystinuria linked to SLC3A1 and SLC7A9 mutations [13].

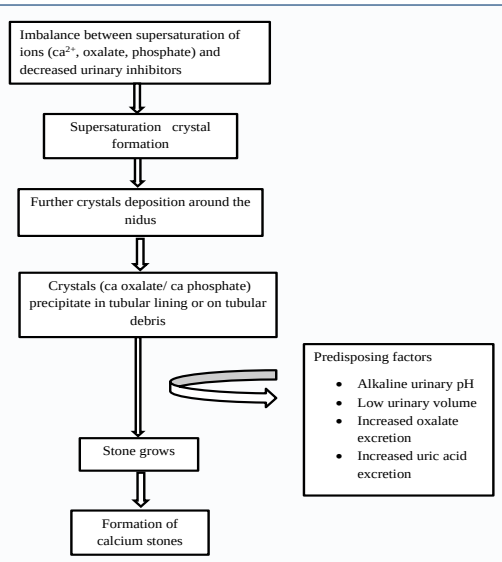
Pathophysiology

Urinary calculi form through a complex, multistep physicochemical process that begins with supersaturation of urine with stone-forming solutes such as calcium oxalate, calcium phosphate, uric acid, or cystine. Supersaturation creates favourable conditions for precipitation, leading to nucleation, where dissolved solutes transform into solid crystals. Nucleation may be homogeneous or occur around biological substrates such as proteins, cellular debris, or epithelial cells. These initial crystals then undergo growth and aggregation, forming larger, more stable particles. Crystal interaction with renal tubular epithelial cells through adhesion, endocytosis, or epithelial injury activates inflammatory pathways and promotes the release of macromolecules that further enhance stone formation.

Several theories explain these early events, including the **free particle theory**, which proposes intratubular crystal growth in supersaturated urine, and the **fixed particle theory,** which emphasizes crystal adherence to damaged epithelium. The **Randall's plaque theory** suggests calcium phosphate deposition in the loop of Henle basement membrane, later exposed to urine and serving as a nidus for stone growth. Overall, stone formation reflects an imbalance between crystallization promoters and inhibitors such as citrate and magnesium, allowing progressive stone development and eventual migration into the urinary tract [14, 15, 59, 60] (Flow chart 1-4).



Flow chart 1: Pathophysiology of uric acid stones.



Flow chart 2: Pathophysiology of calcium stones.

Clinical Manifestations

Urolithiasis most often presents with acute renal colic, which is simply described as sudden onset, severe flank pain radiating towards the lower abdomen or groin. The pain usually comes in waves because of ureteric peristalsis attempting to expel the obstructing stone [16, 17, 61]. During an acute episode, patients commonly develop nausea, vomiting, diaphoresis, and intense restlessness. These autonomic symptoms occur because severe visceral pain triggers sympathetic overactivity and gastrointestinal dysmotility [16]. Haematuria, either gross or microscopic, is another frequent finding. It results from abrasion of the urothelium as the stone migrates through the collecting system. However, the absence of hematuria does not exclude the diagnosis, as bleeding may be intermittent or masked by hydration status and urine output variations [18].

The clinical manifestations vary substantially depending on where the stone is lodged within the urinary tract:

Renal calculi may remain asymptomatic for long periods, especially when non-obstructing. They may cause only mild, intermittent flank discomfort, often mistaken for musculoskeletal pain.

Mid or upper ureteric stones most often cause classic flank pain because the obstruction lies proximal to the pelvic brim.

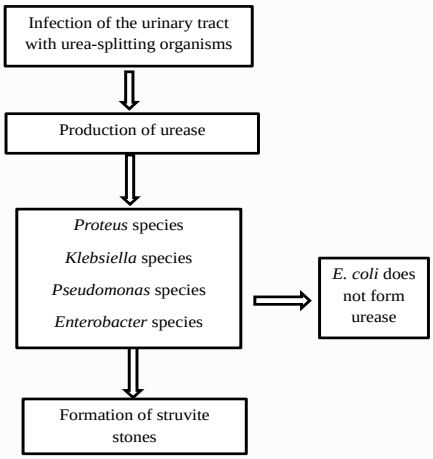
Distal ureteric stones, lodged near the ureterovesical junction (UVJ), may radiate pain to the scrotum or labia and are more likely to produce lower urinary tract symptoms (LUTS) such as urgency, frequency, dysuria, hesitancy, or suprapubic discomfort. These symptoms arise due to irritation of the bladder trigone and pelvic nerves [19].

Systemic features such as fever, chills, rigors, and malaise should raise concern for an infected obstructing stone, also known as urosepsis secondary to obstructive pyelonephritis. This condition represents a true urological emergency because ongoing obstruction in the presence of infection can lead to bacteremia and septic shock. Prompt recognition and urgent decompression (with a ureteral stent or percutaneous nephrostomy) are essential to prevent morbidity and mortality [20, 21].

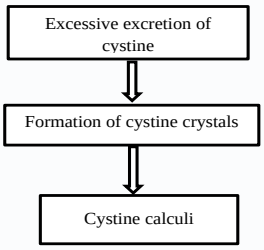
Certain populations may present with atypical or subtle symptoms. Elderly patients and individuals with diabetes may not experience the classic severe colicky pain due to altered pain perception or neuropathy. Instead, they may present directly with fever, confusion, lethargy, or even sepsis, making early diagnosis more challenging. Pregnant women may report nonspecific flank discomfort, lower abdominal pain, or urinary symptoms, which can be easily mistaken for obstetric causes. Physiological hydronephrosis of pregnancy further complicates clinical assessment and delays diagnosis [22].

Patients with bladder stones, which are more common in individuals with bladder outlet obstruction or chronic infections, often present with recurrent UTIs, suprapubic pain, dysuria, terminal haematuria, or an intermittent urinary stream caused by mechanical irritation and intermittent blockage of the bladder neck [26].

Finally, severe obstruction, particularly bilateral ureteric



Flow chart 3: Formation of struvite stones.



Flow chart 4: Formation of cystine calculi.

obstruction or obstruction in a solitary kidney, may lead to oliguria or anuria, which should be considered a red-flag symptom. Early identification and intervention are critical to prevent irreversible renal damage [21].

Diagnosis

Diagnosis of urolithiasis begins with a thorough clinical assessment, which guides appropriate investigations and management. A detailed history helps distinguish renal colic from other causes of abdominal or flank pain by evaluating the onset, character, severity, and radiation of pain, along with associated symptoms such as haematuria, dysuria, and urinary frequency. Assessment of hydration status, dietary habits (high salt or oxalate intake), prior stone episodes, and metabolic or systemic risk factors is essential. Red-flag features including persistent vomiting, high-grade fever, anuria, or a solitary functioning kidney suggest severe obstruction or infection and mandate urgent imaging and intervention [16, 20].

Urinalysis is a key initial investigation. **Microscopic haematuria** is common, though its absence does not exclude stones. **Pyuria or bacteriuria** suggests concomitant infection, which significantly increases risk in obstructing stones. **Urine pH** provides diagnostic clues, with acidic urine favouring uric acid stones and alkaline urine associated with struvite or calcium phosphate stones, particularly in urease-producing infections [18, 23].

Blood tests complement urine findings by assessing renal function and metabolic contributors. **Serum creatinine** and Estimated Glomerular Filtration Rate (**eGFR**) evaluate obstruction-related impairment, while **electrolytes** and **complete blood count (CBC)** identify dehydration, infection, or inflammation. Elevated serum calcium or uric acid may indicate underlying metabolic disorders predisposing to stone formation [24].

Imaging confirms diagnosis**. Non-contrast Computed Tomography of Kidneys, Ureters and Bladder (CT KUB)** is the gold standard due to high sensitivity and specificity, accurately defining stone size, location, density, and obstruction. Low-dose CT reduces radiation without loss of accuracy, while contrast CT is avoided as stones may be obscured [17, 25, 62, 63]. **Ultrasound** is preferred in pregnancy and paediatrics, detecting hydronephrosis and stones using Doppler twinkling artifact [19, 26]. **KUB X-ray** is limited to follow-up of radiopaque stones [20], and Magnetic Resonance Imaging (MRI) is reserved when Computed Tomography (CT) is contraindicated [22].

For recurrent or high-risk patients, 24-hour urine metabolic evaluation identifies abnormalities and enables targeted preventive therapy, reducing recurrence and improving outcomes [27, 28, 64, 65].

Management

Management of urolithiasis depends on multiple factors including stone size, location, composition, severity of symptoms, presence of infection, and renal function. The objectives are to relieve pain, facilitate stone expulsion or removal, prevent complications, and reduce recurrence. Management strategies can broadly be divided into non-pharmacological, pharmacological, and surgical/interventional approaches.

Non-Pharmacological Management

Non-pharmacological measures form the cornerstone of both acute management and long-term prevention of urolithiasis. Adequate hydration is the most effective preventive strategy across all stone types. Patients are advised to increase fluid intake to achieve a urine output of at least 2–2.5 L/day, which dilutes urinary solutes, reduces supersaturation, and limits crystal formation. Multiple studies confirm that high fluid intake significantly lowers stone recurrence, particularly in recurrent calcium stone formers [29, 66, 67].

Dietary modification plays a critical role in reducing stone risk. Sodium restriction to <2 g/day is recommended, as high sodium intake increases urinary calcium excretion. Normal dietary calcium intake (1000–1200 mg/day) is encouraged, since low calcium diets paradoxically increase oxalate absorption and stone risk [30]. Limiting oxalate-rich foods such as spinach, nuts, chocolate, tea, and soy is advised in calcium oxalate stone formers. Excess animal protein intake should be moderated because it raises urinary calcium and lowers citrate, while high sugar and fructose consumption is associated with increased stone risk [31].

Lifestyle modifications include weight management, regular physical activity, and increased hydration in hot climates. Obesity, sedentary behavior, and climate-related dehydration are recognized contributors to the rising global prevalence of urolithiasis [32].

Pharmacological Management

Pharmacological management of urolithiasis serves three key purposes: symptomatic relief during acute stone episodes, facilitation of stone passage through Medical Expulsive Therapy (MET), and long-term prevention of recurrence based on stone composition and metabolic risk factors. When combined with lifestyle modification, a targeted drug-based approach significantly reduces recurrence and improves patient quality of life.

1. Analgesic and Supportive Therapy in Acute Renal Stones

Acute renal colic requires prompt pharmacological treatment due to severe visceral pain. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) such as diclofenac, ketorolac, and ibuprofen are first-line agents. By inhibiting prostaglandin synthesis, NSAIDs reduce renal pelvic pressure and ureteric spasm, providing effective analgesia. Studies show NSAIDs are superior to opioids, with fewer adverse effects and reduced need for rescue analgesia [18]. Opioids (e.g., tramadol, morphine) are reserved for patients in whom NSAIDs are contraindicated or ineffective, due to risks of nausea, sedation, respiratory depression, and dependency [33].

2. Medical Expulsive Therapy (MET)

MET is recommended for stones 5–10 mm, particularly distal ureteric calculi. Alpha-1 Adrenergic Blockers, especially tamsulosin, relax ureteric smooth muscle, reduce spasm, and facilitate stone passage. Meta-analyses demonstrate improved expulsion rates, shorter passage time, reduced analgesic requirements, and fewer hospital visits, especially for stones >5 mm [34]. Calcium channel blockers are less favoured due to limited efficacy.

3. Pharmacological Prevention of Recurrence (Based on Stone Type)

A. Calcium-Based Stones

Thiazide diuretics (hydrochlorothiazide, indapamide) reduce urinary calcium excretion and significantly lower recurrence, particularly when combined with sodium restriction [35, 68, 69]. Potassium citrate increases urinary citrate, alkalinizes urine, and inhibits calcium crystal aggregation, effectively preventing calcium oxalate stone recurrence [35].

B. Uric Acid Stones

Urinary alkalinization with potassium citrate or sodium bicarbonate (target pH 6.5–7.0) improves uric acid solubility. Xanthine oxidase inhibitors (allopurinol, febuxostat) reduce uric acid production and recurrence in hyperuricemia or hyperuricosuria, especially in metabolic syndrome and gout [36].

C. Struvite Stones

Management requires complete stone clearance and infection control. Culture-directed antibiotics are essential, while acetohydroxamic acid (AHA), a urease inhibitor, may be used in selected cases when surgical clearance is incomplete [22].

D. Cystine Stones

Thiol-binding drugs (tiopronin, D-penicillamine) reduce cystine crystallization. Urinary alkalinization (pH >7) improves solubility. Combined therapy with high fluid intake, alkalinization, and thiol agents significantly reduces recurrence [37].

Surgical Management of Urolithiasis

Surgical intervention is indicated when conservative or pharmacological management fails, or when stones cause complications such as persistent obstruction, infection, severe pain, or renal function deterioration. Modern urology favours minimally invasive techniques, significantly reducing morbidity and hospital stay. Procedure selection depends on stone size, location, composition, anatomy, and patient factors.

1. Extracorporeal Shock Wave Lithotripsy (ESWL)

Extracorporeal shock wave lithotripsy (ESWL) is a non-invasive method that uses focused acoustic shock waves to fragment urinary stones into smaller pieces that pass spontaneously in urine.

ESWL is most effective for:

- Renal stones <2 cm
- Upper ureteric stones
- Radiopaque, moderately dense stones

It is typically performed as an outpatient procedure under analgesia or light sedation. Success depends on stone density, location, skin-to-stone distance, and patient anatomy. Recent studies confirm that ESWL remains effective with appropriate patient selection.

Limitations include:

- Lower pole renal stones
- Hard stones (e.g., cystine, calcium oxalate monohydrate)
- Obese patients

Complications are usually mild, including hematuria, flank bruising, and renal colic due to fragment passage [38].

2. Ureteroscopy (URS)

Ureteroscopy is widely used for ureteric and small-to-moderate renal stones. A rigid or flexible ureteroscope is passed through the urinary tract, and stones are fragmented using Holmium: Yttrium Aluminium Garnet (Ho:YAG) laser or removed with baskets.

Indications include:

- Mid and distal ureteric stones
- Renal stones <20 mm
- ESWL failure

Anatomical conditions limiting ESWL efficacy: Advances in endoscopy and laser technology have improved stone-free rates, with flexible URS showing high success for stones <2 cm. Temporary ureteral stenting is commonly used post-procedure.

Complications such as hematuria, ureteric injury, infection, and rare strictures may occur, but URS remains a safe, low-morbidity option [39, 70, 71].

3. Percutaneous Nephrolithotomy (PCNL)

PCNL is the gold standard for large (>2 cm) and complex renal stones. A percutaneous tract is created into the kidney, allowing direct stone fragmentation and removal.

Indications include:

- Large renal stones (>2 cm)
- Partial or complete staghorn calculi
- Multiple or ESWL-resistant stones

Mini- Percutaneous Nephrolithotomy (PCNL) and ultra-mini PCNL techniques reduce bleeding, pain, and hospital stay while maintaining high clearance rates. Studies show mini-PCNL offers less blood loss and shorter hospitalization in selected patients [40].

Although more invasive, PCNL is the most effective option for

large stone burdens. Complications include bleeding, fever, organ injury, and rarely sepsis, with significantly reduced rates using modern techniques [40, 41].

4. Open Surgery

Open surgery is now rarely required due to advances in ESWL, URS, and PCNL. Indications include failed minimally invasive procedures, complex anatomy, morbid obesity, skeletal deformities, non-functioning kidneys, ectopic kidneys, giant bladder stones, and select pediatric cases. Laparoscopic stone surgery has largely replaced open approaches in such situations [38].

Post-Surgical Follow-Up and Prevention

Postoperative care includes regular imaging, metabolic evaluation, and long-term preventive therapy. Without prevention, up to 50% of patients experience recurrence within 5–10 years, highlighting the importance of combining surgery with metabolic and lifestyle management [29, 35, 72, 73].

Ayurvedic Management of Urolithiasis

Urolithiasis (Mutrashmari) management integrates allopathic and traditional approaches. Ayurveda emphasizes correcting doshic imbalance using Ashmarighna and Mutravirechaniya herbs such as Bergenia ligulata and polyherbal formulations like Gokshuradi Yog, which exhibit diuretic, antioxidant, and crystal-inhibitory effects, supported by experimental and clinical evidence [74, 75, 76]. Allopathy focuses on rapid stone clearance and symptom relief through NSAIDs, alpha-blockers, potassium citrate, and interventions such as ESWL and ureteroscopy [77]. While allopathic care is effective for acute and obstructive stones, Ayurveda is valuable for prevention and recurrence reduction, highlighting the potential of integrative, evidence-based urolithiasis management [74, 78].

Case Study-1

The patient is a 42-year-old male with Unique Health Identification (UHID) No: XXXX, admitted at Sri Venkateswara Institute of Medical Sciences (SVIMS), Tirupati, under Department of Nephrology, who is a known case of type-2 diabetes mellitus, hypertension and presented with complaints of right-sided flank pain for the past 3 days associated with burning micturition and reddish discoloration of urine. The pain was colicky in nature, radiating towards the groin and aggravated on movement, with no history of fever, trauma, or urinary retention. There was no past history of renal stones or renal surgery. The patient had a mixed diet, occasional alcohol intake, and reduced oral fluid intake due to work strain.

On examination, the patient was conscious, oriented, moderately built and nourished. Vitals were stable. Blood pressure was 140/90 mmHg, pulse rate 88 beats per minute, respiratory rate 18 breaths per minute, body temperature 98.6°F. Mild right costovertebral angle tenderness was present. No pedal edema or pallor noted and abdominal examination was normal.

Laboratory investigations revealed microscopic haematuria on urine routine examination. Serum creatinine and blood urea were within normal limits. Random blood sugar was elevated. Ultrasound abdomen showed right renal calculus measuring around 6 mm with mild hydronephrosis. CT KUB confirmed a right ureteric calculus causing partial obstruction. Other laboratory parameters were within normal limits.

Based on clinical features and investigations, the patient was diagnosed with right ureteric calculus with mild hydronephrosis.

The patient was started on intravenous fluids to improve hydration. For pain management, diclofenac injection was given. He was initiated on medical expulsive therapy with tamsulosin. Proton pump inhibitor and antiemetic were added for supportive care. Adequate glycemic and blood pressure control was continued with regular medications. He was advised to increase oral fluid intake to at least 2.5–3 litres per day and follow dietary modifications including reduced salt and oxalate-rich foods. He was monitored for spontaneous stone passage and symptom relief. The plan included follow-up imaging after 2 weeks to assess stone position. In case of non-passage, ureteroscopy with holmium laser lithotripsy was planned.

Pharmacist Interventions

Here, we counselled the patient on increasing oral fluid intake to 2.5–3 litres/day and advised dietary modifications such as reducing salt, oxalate-rich foods, and limiting alcohol and also monitored the patient for pain relief, haematuria, and ensured follow-up imaging after 2 weeks to track stone passage and educated the patient on correct use of prescribed medications, including taking tamsulosin at bedtime for better stone passage and avoiding overuse of diclofenac due to renal risks and reinforced adherence to diabetes and hypertension medications and advised regular blood glucose monitoring to prevent renal complications.

Case Study-2

A 28-year-old female patient was admitted to the Department of Nephrology at Sri Venkateswara Institute of Medical Sciences (SVIMS), Tirupati with complaints of fever with chills and rigors, left loin pain and nausea for one day. The pain was sudden in onset, moderate in intensity and localized to the left lumbar region. There was no history of burning micturition or gross haematuria. The patient also complained of reduced appetite due to nausea and discomfort. On general examination, she was conscious, cooperative and oriented to time, place and person. Her vital signs were blood pressure 110/70 mmHg, pulse rate 72 beats per minute, respiratory rate 28 breaths per minute, temperature 98.7°F and oxygen saturation 97%. Pallor was noted. On systemic examination, tenderness was present in the left renal angle while other systems were within normal limits.

Laboratory investigations revealed haemoglobin of 13.2 g/dL, total leukocyte count of 14,600 cells/mm^3, platelet count of 1.92 lakhs/mm^3, blood urea 28 mg/dL, serum creatinine 0.7 mg/dL and random blood sugar of 98 mg/dL. Serum electrolytes showed sodium 139mmol/L, potassium 4.3mmol/L and chloride 108mmol/L. Urine routine examination showed the presence of pus cells along with a few red blood cells, indicating urinary tract irritation or infection.

Ultrasonography of the abdomen revealed a left renal calculus measuring around 8 mm with mild back pressure changes and no significant hydronephrosis. Based on the clinical presentation, laboratory findings and imaging results, a diagnosis of left renal calculus with associated urinary tract infection was made.

The patient was managed conservatively normal saline 0.9% sodium chloride along with cefoperazone -sulbactum according to culture and sensitivity reports and Inj. Tramadol for pain management and Inj. Ondansetron for nausea and vomiting. She was also monitored for pain relief, urine output and resolution of fever. Over the period of hospitalization, the patient showed good clinical

improvement with subsidence of fever and significant reduction in loin pain.

On discharge, she was advised to increase her oral fluid intake to at least 2.5 to 3 litres per day, follow dietary modifications to prevent recurrence of renal stones and avoid foods rich in oxalates and salt. She was also advised regular follow-up with repeat ultrasound and urine examination to monitor the status of the calculus. This case highlights the importance of early diagnosis and appropriate management of renal calculi to prevent complications such as urinary obstruction and renal damage.

Pharmacist interventions

Here, we advised the patient to drink plenty of fluids (around 2.5–3 litres per day) to help flush the infection and support the passing of the stone and counselled her on diet, suggesting that to limit oxalate-rich foods and salty foods to prevent future stone formation and also instructed her to complete the full antibiotic course and to come for follow-up with repeat urine tests and ultrasound to monitor the stone and infection status.

And also gave awareness regarding the usage of NSAID's

NSAIDs + Dehydration or reduced fluid intake may worsen kidney function, so hydration is important.

Future Perspectives

Although diagnostic and therapeutic advances have significantly improved short-term outcomes in urolithiasis, recurrence continues to pose a major clinical challenge. Emerging evidence suggests that future management must shift from a generalized approach to a precision-based prevention model, where individual metabolic, genetic and lifestyle determinants guide clinical decision-making [42].

A key component of this precision approach is metabolic profiling, which involves detailed biochemical assessment of urine and serum to identify abnormalities such as hypercalciuria, hypocitraturia, hyperoxaluria or persistently acidic urine. Advances in analytical technologies now allow more accurate detection of subtle metabolic defects, supporting personalized dietary and pharmacological interventions tailored to each patient's lithogenic risk [43, 44].

Novel research has also highlighted the role of urinary microbiome dynamics in stone formation. Shifts in microbial composition may influence urinary pH, citrate metabolism, and inflammatory responses, suggesting that microbiome-modifying therapies could emerge as adjunctive preventive tools [45]. Similarly, investigations into genetic markers and polygenic risk scores are beginning to identify individuals with heightened predisposition to stones, making early prevention possible before stone formation even begins [46, 47, 48].

Conclusion

Urolithiasis is a frequent and recurring urological condition with multiple causes, including genetic, metabolic, nutritional, and environmental variables. Advances in understanding the processes of stone formation have led to better diagnostic accuracy and therapeutic techniques. Non-contrast computed tomography remains the diagnostic gold standard, with ultrasonography serving as a viable option in certain groups. The care of urolithiasis has changed toward minimally invasive procedures such as extracorporeal shock wave lithotripsy, ureteroscopy, and percutaneous nephrolithotomy, resulting in lower morbidity and better patient outcomes. However, preventing a recurrence remains a significant issue. Long-term management involves metabolic examination, lifestyle and nutritional changes, appropriate hydration, and tailored medication. To reduce recurrence rates and disease burden, a complete and tailored approach that includes early diagnosis, effective treatment, and preventative efforts is required. Further study is needed to improve urolithiasis prevention and management.

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