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Urolithiasis: Comprehensive Review of Etiology, Diagnosis and Treatment

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ABSTRACT

Urolithiasis, commonly referred to as kidney stone disease, is a prevalent and recurrent urological condition with significant global health and economic implications. The formation of urinary calculi results from a multifactorial interplay between genetic predisposition, metabolic abnormalities, dietary habits, and environmental factors. The incidence of urolithiasis has been rising worldwide, particularly in developed countries, contributing to an increasing burden on healthcare systems.

This review provides a comprehensive analysis of the etiology and risk factors associated with kidney stone formation, including dietary and non-dietary contributors such as high animal protein intake, calcium and oxalate metabolism, obesity, diabetes, and climate influences. Advances in diagnostic modalities, particularly the role of non-contrast computed tomography (CT), ultrasonography, and metabolic evaluation, have improved early detection and classification of urinary stones.

Treatment options range from conservative medical management to surgical interventions, including extracorporeal shock wave lithotripsy (ESWL), ureteroscopy (URS), and percutaneous nephrolithotomy (PCNL), with evolving techniques enhancing stone clearance and minimizing recurrence. Furthermore, lifestyle modifications, adequate hydration, and pharmacological interventions play crucial roles in the prevention of recurrent stone formation.

The growing prevalence of urolithiasis underscores the need for continuous advancements in both preventive and therapeutic strategies. Further research into stone pathophysiology, metabolic risk assessment, and novel treatment modalities is essential to reduce morbidity and healthcare costs associated with this condition.

KEYWORDS: Urolithiasis, Extracorporeal Shock Wave Litthotripsy (ESWL), Ureterorenoscopy (URS), Percutaneous Nephrolithotomy (PCNL), Renal Colic, Urinary Tract Infections (UTI)

INTRODUCTION

Urolithiasis, commonly known as urinary stone disease, is a widespread urological condition that affects populations globally, with a lifetime prevalence of approximately 10% in men and 6% in women. The incidence of kidney stone formation has been increasing in many developed countries, contributing to a growing public health concern (1-4). This condition is characterized by the formation of urinary calculi due to an imbalance between stone-forming promoters and inhibitors in a process known as lithogenesis (5). Urolithiasis not only causes severe acute back pain but can also lead to serious complications such as pyelonephritis or acute renal failure (6). Moreover, the recurrence rate remains high, with nearly 60% of patients experiencing a second episode of renal colic ten years after initial treatment (7). More than 10% of patients may experience additional relapses (8). Furthermore, the recurrence rate of urinary calculi in patients with specific stone mineral compositions and morphologies can be as high as 82.4% (9).

The etiology of urolithiasis is multifactorial, involving complex interactions between genetic and environmental factors. Lifestyle, obesity, dietary habits, and dehydration are among the key environmental contributors (10,11), while hormonal, genetic, and anatomical abnormalities also play a significant role in stone formation (12). Additionally, variations in prevalence are observed across different regions, with rates ranging from 7–13% in North America, 5–9% in Europe, and 1–5% in Asia (13-15). The increasing prevalence of kidney stone disease, particularly in individuals over the age of 30, has led to a substantial economic burden on healthcare systems worldwide (16). In the United States alone, urolithiasis accounts for approximately $2 billion in healthcare expenditures annually (17) , with projections estimating an additional $1.24 billion per year by 2030 (18). Globally, the cost of managing kidney stone disease reached an estimated $5.3 billion in 2014, making it the second most expensive urological disorder. (19). Considering the significant morbidity and economic impact associated with urolithiasis, continuous advancement in diagnostic techniques and treatment modalities remains crucial.

ETIOLOGY

The etiology of urolithiasis is complex, including both dietary and non-dietary Factors.

1.Dietary factors

Diet and fluid consumption significantly influence urine composition and the risk of urolithiasis. Daily liquid intake, particularly water, is crucial for maintaining proper urine

volume and concentration (20). The most important dietary factors include animal protein, minerals (calcium, sodium, potassium), and certain compounds like oxalate and drugs (21-24).

1.1. Minerals

Calcium: Dietary calcium actually helps prevent stones by binding to oxalate in the intestines, reducing its absorption. A key study demonstrated that normal calcium intake (1200 mg/day) combined with low animal protein reduced stone recurrence by 50% compared to a low-calcium diet (400 mg/day) (19). However, calcium supplements may paradoxically increase stone risk, particularly in older women (6).

Potassium: Higher dietary potassium consumption appears protective against kidney stones in both men and older women (25). It works through two mechanisms: reducing urinary calcium excretion and increasing urinary citrate levels, both of which help prevent stone formation (6).

Sodium: High sodium intake increases urinary calcium excretion independent of calcium consumption (26). Reducing dietary salt effectively lowers urinary calcium levels, with particularly notable benefits in people who have high urinary calcium excretion (hypercalciuria). (6)

1.2.Animal protein

A diet high in animal protein increases urinary oxalate excretion while lowering urinary pH and citrate levels (19). The association between high animal protein intake and nephrolithiasis is widely reported, leading to the common recommendation of restricting dietary animal protein (25, 27-32). Consequently, protein intake is generally advised to be limited to 1g/kg/day (6).

1.3.Oxalates

Urinary oxalate comes from both dietary absorption (10-50%) and endogenous metabolism of substances like glycine and vitamin C. While dietary oxalate appears more crucial for stone formation, absorption varies based on diet, gut bacteria, and health status. Notably, up to one-third of calcium oxalate stone formers show increased oxalate absorption, sometimes due to reduced gut colonization by Oxalobacter formigenes (20,21,33,34).

1.4.Drugs

Drug-induced calculi account for 1–2% of renal stones, often resulting from the crystallization of poorly soluble molecules with high urinary excretion, especially at high doses. Common culprits include protease inhibitors (indinavir, atazanavir) and antiseptics (sulfonamides, ceftriaxone). Some drugs also induce metabolic changes that promote stone formation, such as calcium plus vitamin D supplements and carbonic anhydrase inhibitors, leading to calcium oxalate or calcium phosphate stones (35).

1.5.Fluids

Low fluid intake is a major risk factor for kidney stone formation, while increased intake reduces the risk. A urine output of >2 L/day is recommended which requires a daily fluid intake of 2.0–2.5 L (27,31,36-38).

2.Non dietary risk factors

2.1.Inheritance and genetics

Idiopathic calcium oxalate urolithiasis, the most prevalent form, results from genetic and environmental interactions. While genetic factors are part of the diagnostic assessment, confirmed genetic causes remain rare. However, familial studies have established a strong link between calcium oxalate urolithiasis and primary hypercalciuria, the most common metabolic risk factor (11,39,40).

2.2.Obesity

Higher weight and BMI are linked to an increased risk of kidney stone disease in both men and women, as shown in large prospective studies (11,41). The risk associated with elevated BMI is greater in women than men (21,41). Individuals with a BMI ≥30 have a 30% higher risk in men and nearly double the risk in women compared to those with a BMI of 21–23. Additionally, a weight gain of about 16 kg from early adulthood raises the risk by 40% in men and 80% in women (42). Obesity and insulin resistance contribute to increased acid excretion, leading to more acidic urine (21,43).

2.3.Gout

A history of gout significantly increases the risk of kidney stones, particularly uric acid and calcium oxalate types (44). A nationally representative study found that individuals with gout had a 49% higher likelihood of developing kidney stones, even after adjusting for key factors like age, body mass index, gender, race, and hypertension (45). This association was further validated in a prospective study of men, which revealed that those with gout had more than double the risk of kidney stones compared to those without gout (age-adjusted relative risk: 2.06; multivariate-adjusted relative risk: 2.12) (46).

2.4.Diabetes

Recent studies identify diabetes mellitus (DM) as a risk factor for kidney stones (47,48), primarily due to insulin resistance lowering urine pH (49). Patients with DM are significantly more likely to develop uric acid stones . A study of 2,464 calculi found uric acid stones in 35.7% of type 2 diabetics compared to 11.3% in non-diabetics ($p < .0001$) (43). Research also confirms that insulin resistance disrupts ammoniagenesis, reducing urine pH and increasing

stone risk (46). Diabetes may also alter urine composition, further promoting both uric acid and calcium-containing stones (50).

2.5.Hypertension

The association between hypertension and kidney stones remains unclear. Two 8-year longitudinal studies found a higher incidence of kidney stones in hypertensive patients (51,52), while larger prospective studies showed no independent link (53,54). The Third NHANES survey reported a 69% higher likelihood of self-reported hypertension in female stone formers, but not in men (55). Meanwhile, three cohort studies found that following a DASH-style diet significantly reduced kidney stone risk (56), likely by increasing urinary citrate levels and volume (57).

2.6.Environmental factors

Warmer and more humid regions contribute to a higher incidence of kidney stones, primarily due to chronic dehydration, which leads to low urine volume and hypocitraturia, especially increasing uric acid stone formation (32,58). Seasonal variations show peak cases in summer (59,60). Climate change may further expand high-risk areas, with projections indicating that the U.S. population in these zones could rise from 40% in 2000 to 56% by 2050 and 70% by 2095 (61).

CLINICAL PRESENTATION

Acute renal colic presents as crampy, intermittent flank and abdominal pain as kidney stones move through the ureter (62). The pain often radiates to the groin or lower abdomen and worsens as the stone nears the bladder, sometimes causing dysuria and urinary urgency (63,64). Patients may experience nausea, vomiting, and hematuria, though hematuria is absent in some cases (64). Fever and chills suggest an infected stone or concurrent urinary tract infection (UTI), while tachycardia and hypertension may result from pain (64).

Pain varies from excruciating to a dull pressure, with constant pain at onset raising concern for severe obstruction (64). Patients often appear restless and unable to find a comfortable position (62). Physical exam findings may include costovertebral angle or lower quadrant tenderness (62).

Elderly patients are more likely to have atypical presentations, such as gastrointestinal symptoms, pyuria, or UTI (64,65). They also tend to have larger stones and a higher need for surgical intervention. The physical exam should focus on ruling out other conditions, such as urinary tract infection, musculoskeletal inflammation or spasm, ectopic pregnancy, testicular

torsion, or malignancy (61,66,67,68). In primary care, initial workup should include urinalysis to detect hematuria and rule out other conditions (61,67,69,70).

DIAGNOSIS

A few routine lab tests are useful in evaluating acute flank pain, but imaging is key for diagnosing nephrolithiasis.

1.Laboratory tests

Initial laboratory testing involves urinalysis and serum chemistry to evaluate hematuria and creatinine clearance. A complete blood count with differential is reasonable if the patient presents with systemic signs of infection or an alternate abdominal etiology is strongly suspected. A woman of reproductive age should receive a pregnancy test. Microscopic hematuria is present in more than 90% of cases. Pyuria is also often present, but not necessarily indicative of concurrent infection. The stone itself can cause ureteral inflammation, resulting in white blood cells (WBCs) on urinalysis. The greater the degree of pyuria, the greater the likelihood of infection. More than 50 WBCs per highpower field is associated with a 60% culture-positive rate. The presence of leukocyte esterase or nitrites also increases the likelihood of a concurrent urinary tract infection (64).

2.Diagnostic imaging

2.1.Ultrasound

Ultrasonography is increasingly used worldwide for kidney stone diagnosis, including in the U.S (63,64,71). Its advantages include no radiation exposure, lower cost, and ease of use. It is more accessible than CT in many regions and is the first-line imaging choice for children and pregnant women due to radiation concerns (72).

Ultrasound detects stones indirectly by identifying hydronephrosis (64,71) and directly as hyperechoic lines with distal shadowing. However, its accuracy depends on the operator's experience and patient body habitus (72).

A 2014 study comparing ultrasound and CT for suspected nephrolithiasis found (73) that although ultrasound had lower sensitivity and specificity, there were no significant differences in clinical outcomes, including complications, pain, emergency visits, or hospitalizations (73). This suggests that stones missed by ultrasound are likely small and pass spontaneously.

2.2.Abdominal X-Ray

Offers quick accessibility in emergency settings but demonstrates limited diagnostic value for kidney stones, with both low sensitivity and specificity. While not reliable as a standalone diagnostic tool, its utility increases when used in conjunction with CT scanning (74).

2.3.Computed Tomography (CT-scan)

A non-contrast CT scan of the abdomen and pelvis is the gold standard for diagnosing nephrolithiasis in the U.S (63,64,71,72). It detects all stone types, accurately locates them within the urinary system, and measures their size to help determine prognosis (64,71,72). CT can also identify hydronephrosis and other potential causes of pain if no stone is found (64,71). The main drawback of CT is radiation exposure. However, low-dose CT protocols offer comparable sensitivity and specificity, though they may miss small stones (<2–3 mm) and be less effective in obese patients (64,72).

2.4.Magnetic Resonance Imaging (MRI)

MRI does not directly visualize stones but detects them through calcifications and signal voids. Due to its high cost, longer scan time, and limited availability, it has minimal utility in diagnosing nephrolithiasis (72). However, it is primarily used in pregnant women when ultrasound findings are inconclusive (64,72).

TREATMENT OPTIONS

1.Medical Treatment

Several drugs are available for medical expulsive therapy (MET) in urolithiasis (75-77), including β-blockers, calcium channel blockers, and phosphodiesterase type 5 (PDE5) inhibitors. Although β-blockers are an off-label option, meta-analyses have demonstrated their effectiveness for MET (77). Guidelines recommend β-blockers for distal ureteral stones >5 mm (EAU: strong recommendation) and for distal ureteral stones ≤10 mm (AUA: strong recommendation) (78).

2.Surgical treatment

2.1.Extracorporeal Shock Wave Lithotripsy (ESWL)

ESWL is a non-invasive procedure that uses shock waves from an extracorporeal generator to break stones into smaller fragments. Ultrasound is used for stone localization. Success rates vary based on stone size, type, and location, averaging 60–80% for kidney stones and up to 80% for ureteral Stones (79). The EAU lists several contraindications to shock wave lithotripsy (SWL), including uncontrolled UTIs, severe skeletal deformities, obesity, pregnancy, bleeding disorders, anatomical obstructions distal to the stone, and arterial aneurysms near the stone (78).

2.2.Ureteroscopy (URS)

URS is an invasive procedure used to remove stones from the ureters or bladder, while also allowing examination of the upper urinary tract. A ureteroscope, a thin wire with a camera at the end, is inserted through the urethra into the bladder to locate and extract stones. URS offers

a higher stone-free rate (SFR) and better clinical outcomes compared to ESWL (80), with improved safety and lower complication rates in modern endourology (81). Pre-procedural stenting is generally not recommended (76,77), as it is not proven to significantly enhance outcomes. Post-procedure, stenting should be avoided unless necessary, as it increases morbidity and is not cost-effective. If a stent is placed, β-blockers may help reduce discomfort. Stone removal should be performed under direct visualization, with a safety guide wire when possible. A ureteral access sheath (UAS) is useful for prolonged procedures or when dealing with large or multiple renal Stones (82). The holmium: yttrium-aluminum-garnet (Ho:YAG) laser is the preferred method for stone fragmentation, though the thulium fiber laser (TFL) shows promising results. Perioperative prophylactic antibiotics are recommended before any endoscopic procedure. Percutaneous antegrade URS may be considered if SWL has failed and retrograde URS is not an option (78). Flexible URS is a viable alternative for stones >2 cm if PCNL and SWL are not suitable. URS is also the preferred treatment for patients requiring stone removal without stopping antithrombotic therapy. Aside from general anesthesia-related risks and untreated urinary tract infections, URS is considered safe with minimal contraindications (77).

2.3.Percutaneous Nephrolithotomy (PCNL)

Percutaneous nephrolithotomy (PCNL) is an invasive procedure in which a nephroscope is inserted through a percutaneous opening to fragment and remove stones larger than 2 cm or irregularly shaped stones, such as coralliform calculi. PCNL is the first-line treatment for large renal calculi, offering a high stone-free rate (SFR) regardless of stone size or composition (83,84). Pre-procedural imaging, such as ultrasound or CT, is essential to map the collecting system and surrounding structures for a safe percutaneous approach.

Both prone and supine positioning are considered safe, depending on surgical expertise and available equipment. Mini-PCNL (12–22Fr) is associated with shorter hospital stays, less blood loss, and similar SFRs compared to standard PCNL (>22Fr) (77). Flexible nephroscopy should be standard to access stone fragments in difficult locations. Normal saline is recommended as the irrigation solution to prevent electrolyte imbalances (78).

For uncomplicated cases, tubeless or totally tubeless PCNL (without nephrostomy or ureteral stent) can reduce hospital stays and improve postoperative pain control. Intraoperative urine or stone cultures from the renal pelvis are advised to better predict and manage potential post-procedural sepsis (85,86).

Contraindications for PCNL include tumors in the access tract, malignant renal tumors, pregnancy, untreated UTIs, and anticoagulant therapy, which must be carefully managed preoperatively (77).

2.4.Open surgery

Advancements in endourology have made open and laparoscopic approaches for urolithiasis treatment rare. These methods are considered only when SWL, URS, and PCNL are unlikely to be effective. Open or laparoscopic surgery may be beneficial in cases where stone removal is needed alongside anatomical reconstruction (87).

CONCLUSIONS

Urolithiasis remains a significant public health issue due to its increasing incidence, high recurrence rates, and substantial economic burden on healthcare systems worldwide. The multifactorial nature of stone formation highlights the importance of individualized prevention and treatment strategies based on dietary habits, metabolic risk factors, and patient comorbidities.

Advances in diagnostic imaging, particularly low-dose CT and ultrasonography, have improved the accuracy of stone detection while minimizing radiation exposure. Treatment options have evolved, with minimally invasive procedures such as ESWL, URS, and PCNL demonstrating high success rates and reduced morbidity. Pharmacological interventions and lifestyle modifications, including increased fluid intake and dietary adjustments, remain crucial in reducing recurrence risk.

Despite these advancements, further research is needed to better understand the molecular mechanisms of lithogenesis, improve risk stratification, and develop more effective preventive therapies. A multidisciplinary approach involving urologists, nephrologists, and nutritionists is essential for optimizing patient outcomes and minimizing the long-term impact of urolithiasis.

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