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Review Article

A review on Current Perspectives on Nephrolithiasis: A Multidisciplinary Approach to Management

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Abstract:

In Germany, nephrolithiasis prevalence is 4.7%, with recurrence rates of 50–80% without secondary prevention, reduced to 10–15% with tailored strategies. The study utilized various academic databases to compile relevant literature, guided by the European Association of Urology's nephrolithiasis guidelines. Diagnosis of acute renal colic is typically straightforward, and treatment options for stones vary, including surgery and lithotripsy. Most ureteric stones under 5 mm pass spontaneously, with 75% of patients experiencing no complications. Basic assessments for secondary prevention can be performed by general practitioners, while complex metabolic evaluations should occur in specialized settings for symptomatic patients. Effective treatment involves understanding individual patient needs, though further high-quality research on secondary prevention is required, focusing on nephrolithiasis's etiology and pathophysiology.

Keyword: Nephrolithiasis**,** Secondary Prevention, Renal Colic, , Metabolic Evaluation, Chronic kidney disease**.**

Introduction

Kidney stones are mineral deposits that form in the renal calyces and pelvis, containing crystalline and organic components. They develop when urine becomes supersaturated with respect to a mineral, with calcium oxalate being the main constituent of most stones (Khan et al, 2016). The formation of kidney stones is a complex, multi-step process influenced by physical, chemical, and biological factors (Dong et al., 2024). Nephrolithiasis is a common health concern affecting millions worldwide, with prevalence rates up to 14.8% and increasing. The recurrence rate is high, reaching up to 50% within the first 5 years of the initial stone episode (Khan et al., 2016). Interestingly, kidney stones are more prevalent in individuals with a family history, suggesting a genetic component to stone formation (Curhan et al., 1997; Howles & Thakker, 2020). In conclusion, kidney stone formation is a multifactorial process involving both metabolic and environmental risk factors. It is considered a systemic disorder associated with various health complications, including chronic kidney disease, bone loss, increased risk of coronary artery disease, hypertension, and type 2 diabetes mellitus (Sakhaee et al., 2012). Understanding the pathophysiology of nephrolithiasis is crucial for developing new therapeutic options and preventive strategies to address this growing global health concern.

Kidney stone formation involves complex pathogenetic mechanisms, including both metabolic and environmental risk factors (Sakhaee et al., 2012). The process, known as urolithiasis, can occur anywhere in the renal tract and has been a longstanding health problem since ancient times (Kasote et al., 2017). Causes of kidney stones include hypercalciuria, hyperoxaluria, and decreased urinary citrate and magnesium levels (Böhles et al., 1988). Genetic factors also play a significant role, with studies estimating heritability of >45% for nephrolithiasis and >50% for hypercalciuria (Howles & Thakker, 2020). Additionally, certain medical conditions like inflammatory bowel disease and HIV treatment with indinavir can increase the risk of kidney stone formation (Böhles et al., 1988,Saltel et al., 2000).

Treatment approaches include both conventional and herbal remedies. Dietary modifications are often recommended as a preventive measure, although drugs may be necessary in some cases (Morgan & Pearle, 2016). Herbal treatments have gained attention due to their perceived safety and efficacy. Plants such as Phyllanthus niruri L. and Elymus repens (L.) Gould, as well as herbal products like 'Wu-Ling-San' formula, 'Cystone', and 'Herbmed' have shown promise in clinical trials (Kasote et al., 2017). Some isolated phytochemicals, including berberine, lupeol, and khelin, have also demonstrated potent antiurolithiatic activity (Kasote et al., 2017). In conclusion, while conventional treatments remain important, herbal remedies offer potential alternative or complementary approaches for managing kidney stones. However, more research is needed to establish the efficacy and safety of many herbal treatments (Kasote et al., 2017).

Epidemology:

Nephrolithiasis is common, affecting approximately 1 in 11 people in the United States. By age 70, 19.1% of men and 9.4% of women report ever having a kidney stone. The burden of this disease appears to be growing, with the National Health and Nutrition Examination Survey noting an increase in the self-reported prevalence of kidney stones, from 3.2% in 1976-1980 to 8.8% in 2014. The male-to-female ratio has decreased from 3:1 to about 2:1 in the past 2 decades, attributed to an increasing prevalence of obesity. Obesity and diabetes are strongly associated with a history of kidney stones in multivariate models, particularly for women. Racial and ethnic differences are also evident, with the prevalence of nephrolithiasis being higher in White male patients, intermediate in Hispanic and Asian patients, and less common in Black patients. The highest risk of stone formation is reported in men in the United Arab Emirates and Saudi Arabia. In the United States, there is an

increasing prevalence of nephrolithiasis from North to South and from West to East. Heat-related increases in urinary concentration from nonrenal water losses, geographic differences in diabetes and obesity rates, and other environmental and genetic factors likely explain these variances. The link between increased environmental temperatures and increased rates of stone disease is well documented, thus predicting a further increase in its incidence with climate change. The risk of stone recurrence is high, with a relapse rate of 50% in 5-10 years and 75% in 20 years. Risk factors for recurrent stones include multiple prior stone episodes, younger age of onset, male gender, family history of kidney stones, and higher body mass index (BMI). Stone characteristics which predict recurrence include the presence of 2 or more stones across both kidneys, the presence of stones in the renal pelvis or lower kidney pole, and a stone composition consisting of uric acid, struvite, or brushite (a particularly hard form of calcium phosphate). The online Recurrence of Kidney Stone (ROKS) nomogram Nephrolithiasis has been associated with significant morbidity beyond the urologic system. Among a cohort in Olmsted County, Minnesota, stone-formers were at increased risk of developing chronic kidney disease (CKD) compared to non-stone-formers. In a population cohort study from Canada with a median follow-up period of 11 years, the risks of end-stage kidney disease, late-stage CKD, and doubling of serum creatinine were significantly higher among participants with 1 or more episodes of nephrolithiasis, especially in women and those younger than 50 years. Struvite stone-formers with staghorn calculi and patients with cystinuria are at especially high risk for CKD. (Romero et al.2010)Nephrolithiasis is also associated with an increased risk of cardiovascular disease. Studies reveal a greater prevalence of hypertension and possibly increased carotid wall thickness in stone patients, even when controlling for major atherosclerotic risk factors. One analysis revealed a 31% increase in risk for myocardial infarction in those with a history of nephrolithiasis despite adjusting for known risk factors for cardiovascular disease. Finally, the link between kidney stones and reduced bone mineral density and fractures is particularly robuststates. By age 70, 19.1% of men and 9.4% of women report ever having a kidney stone. The burden of this disease appears to be growing, with the National Health and Nutrition Examination Survey noting an increase in the self-reported prevalence of kidney stones, from 3.2% in 1976-1980 to 8.8% in 2014. The male-to-female ratio has decreased from 3:1 to about 2:1 in the past 2 decades, attributed to an increasing prevalence of obesity. (Scales CD et al .2012)Obesity and diabetes are strongly associated with a history of kidney stones in multivariate models, particularly for women. Racial and ethnic differences are also evident, with the prevalence of nephrolithiasis being higher in White male patients, intermediate in Hispanic and Asian patients, and less common in Black patients. The highest risk of stone formation is reported in men in the United Arab Emirates and Saudi Arabia. In the United States, there is an increasing prevalence of nephrolithiasis from North to South and from West to East. Heat-related increases in urinary concentration from non renal water losses, geographic differences in diabetes and obesity rates, and other environmental and genetic factors likely explain these variances. The link between increased environmental temperatures and increased rates of stone disease is well documented, thus predicting a further increase in its incidence with climate change

Table 1: Classification of urinary stone patients as high risk

Finding	Action
Age: child or adolescent	Consider assessing siblings for risk of lithogenesis
Brushite, Infectious stones, Uric acid/urate,	Bear other accompanying minerals in mind during diagnosis and treatment
Chronic Psychovegetative stress	Establish severity, possibly with validated stress-assessment systems
Single kidney	Higher risk – requires closer monitoring
Malformation of the urinary tract	Requires special management
Disorders of gastrointestinal function (e.g., Crohn's disease, ulcerative colitis, sprue, chronic pancreatitis, liver cirrhosis, small bowel resection)	Careful evaluation of metabolic risk factors
High recurrence rate (e.g., >3 stones in 3 years; changes in stone type or composition)	Indicates altered metabolic conditions – requires thorough reassessment
Hyperparathyroidism (HPT – five forms: primary to quinary)	Identify and treat underlying cause
Nephrocalcinosis (various causes: RTA, primary hyperoxaluria, sarcoidosis, HPT, chronic glomerulitis, etc.)	Consider metabolic and systemic causes
Positive family history	Assess patient's children for risk of lithogenesis
Primary hyperoxaluria (two types, autosomal-recessive)	Lifelong management; consider genetic screening
Renal tubular acidosis	Test using urinary pH curve, blood gas analysis, and ammonium chloride load test
Residual stone fragments	Consider endoscopic removal, especially if stone type resists ESWL (e.g., brushite, cystine, whewellite)
Cystine, 2,8-dihydroxyadenine, xanthine stones	Genetically determined; lifelong metaphylaxis is mandatory

Pathogenisis.

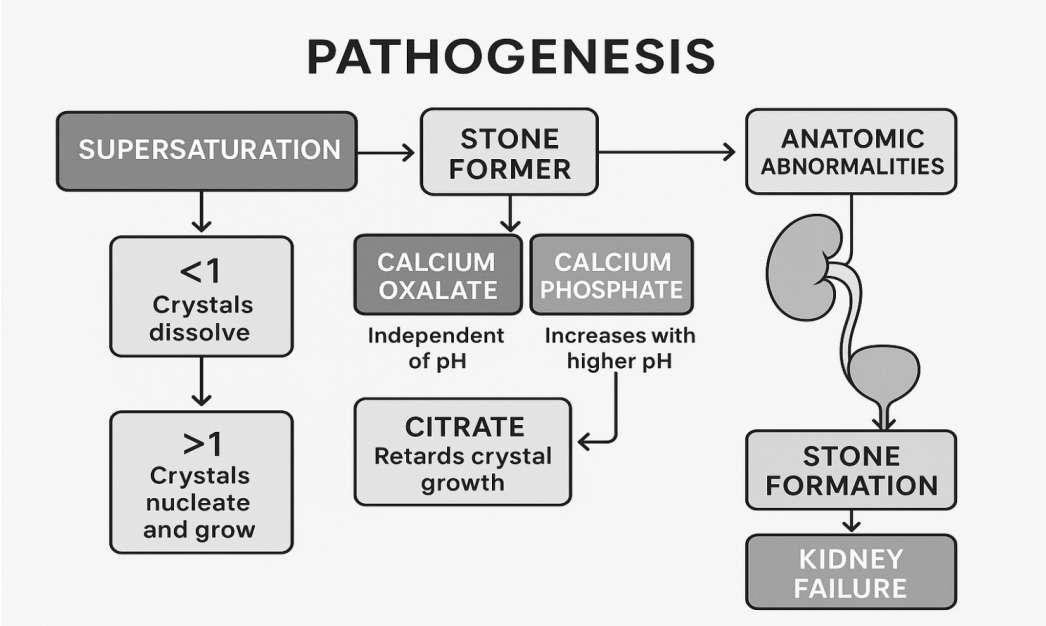

Figure 1: Pathogenesis of kidney stone

Supersaturation, often expressed as the ratio of urinary calcium oxalate or calcium phosphate concentration to their solubility, is the driving force for stone formation. At levels of supersaturation below 1, crystals dissolve, whereas at supersaturation levels above 1 crystals can nucleate and grow, promoting stone formation. Supersaturation is generally higher in stone formers than non-stone formers, and the type of stone formed correlates with urinary supersaturation. Calcium oxalate supersaturation is independent of urine pH, but calcium phosphate supersaturation increases rapidly as urine pH rises from 6 to 7. Since calcium oxalate stones form over an initial calcium phosphate layer (Evan AP et al.2007), treatment optimally should lower the supersaturation of both species. Most 24-hour kidney stone risk panels from specialized labs include calculated supersaturation values. Urine also contains substances that can accelerate or retard urinary crystallization (De Yoreo JJ et al. 2006). The only such substance which can be modified in practice at this time is citrate, which can slow calcium crystal growth (Tiselius HG et al .1993). Anatomic abnormalities, in particular those that result in urinary stasis (such as ureteropelvic junction obstruction, horseshoe kidney, or polycystic kidney) may precipitate or worsen stone formation (Gambaro G et al 2006). Patients with a single functioning kidney are at particular risk, as stone passage with ureteral obstruction can result in acute kidney failure.

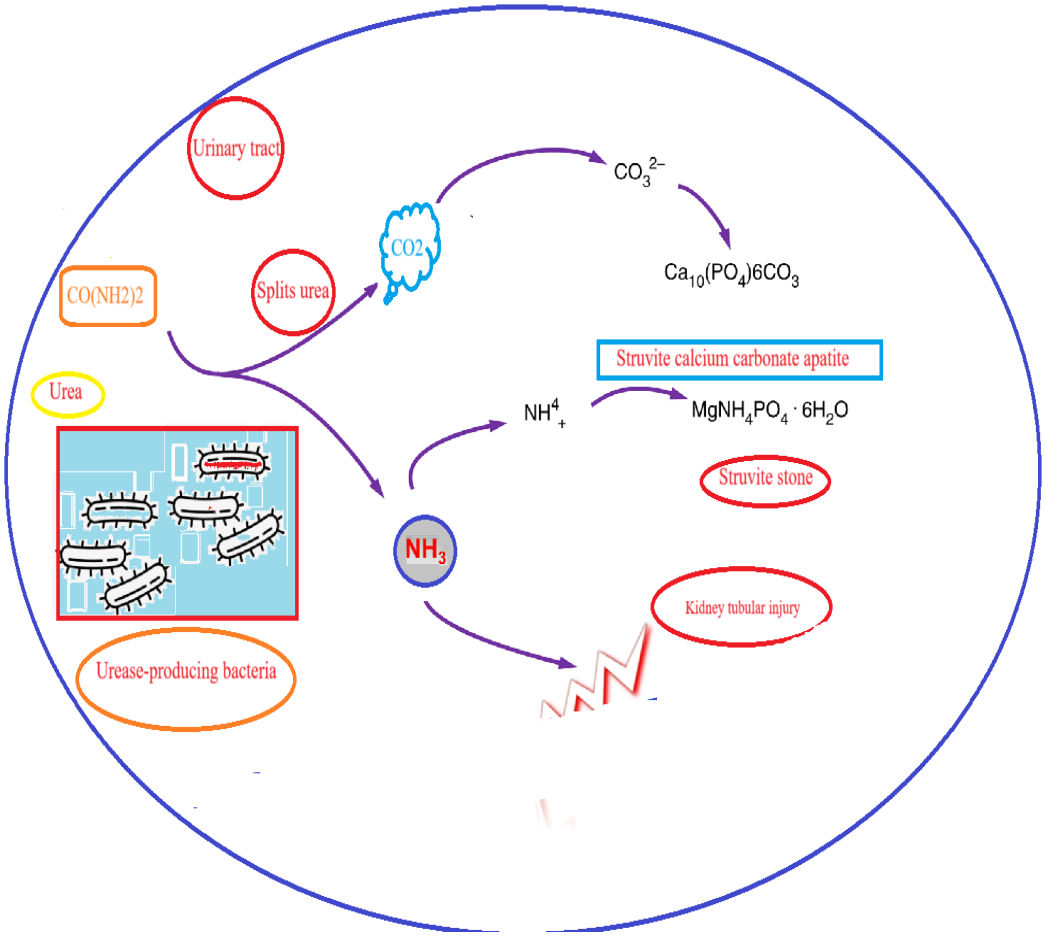

Figure 2: Role of urease-producing bacteria in stone formation. The urease-producing bacteria split urea and promote the formation of ammonia and carbondioxide, leading to kidney tubular injury and urine alkalinization and subsequent formation of phosphate salts

Metabolic Factors

Supersaturation is caused by imbalances in the excretion of calcium, oxalate, and water. The most prevalent metabolic disorder among calcium stone formers is hypercalciuria, which is primarily idiopathic and familial (Worcester EM et al 2007) and heavily impacted by diet. Because ingested calcium is quickly eliminated, those with idiopathic hypercalciuria have higher gut calcium absorption but unchanged serum calcium levels (Coe FL, Favus MJ 2008). These people frequently excrete more calcium than they consume when on a low-calcium diet, and their urine calcium excretion increases significantly after consuming calcium-free foods such simple oral glucose; in these situations, bone is the sole available source. While hypercalciuria can occasionally be separated into subtypes (renal leak, absorptive, and resorptive), although this categorization is not useful for directing treatment. Serum calcium levels should be measured, nevertheless, in order to diagnose patients with primary hyperparathyroidism. (Favus MJ, Coe FL, 2008).

Perhaps as a result of greater intestinal oxalate absorption, calcium stone formers have somewhat higher oxalate excretions than non-stone formers (Voss S, Hesse A 2006). High protein consumption and ascorbic acid may boost the synthesis of oxalate (Coe FL, Favus MJ, 2008). When dietary calcium is low, oxalate is more easily absorbed because calcium binds to it in the gut and prevents its absorption (Borghi L, Schianchi T, Meschi T, et al 2002). This could be the reason why recurrences of stones are not effectively prevented by a low-calcium diet (Hamm LL, Hering-Smith KS, 2002). Hypocitraturia is a risk factor for stones because citrate chelates calcium in the urine, reducing supersaturation and crystal formation . Although the origin of hypocitraturia in the majority of stone formers is unknown, it is caused by distal renal tubular acidosis, hypokalemia, and carbonic anhydrase inhibitors such as topiramate (Hamm LL, Hering-Smith KS, 2002). It is believed that hyperuricosuria, which is frequently caused by a high purine diet, increases the risk of calcium stones by decreasing the solubility of calcium oxalate (Coe FL, Kavalach AG 1974).

Table 2: Principal Substances Used in Medicinal Prophylaxis of Urinary Stones

Substance	Goal	Dosage	Remarks	Stone Types Amenable to Treatment
Alkaline citrates	– Alkalization of urine – Compensation of hypocitraturia – Lowers proportion of ionized Ca in urine – Regulation of acid–base balance in RTA and metabolic acidosis	5–12 g/day (14–36 mmol/day); children: 0.1–0.15 g/kg BW/day *Target urinary pH:* – Ca oxalate metaphylaxis: 6.5–6.8 – Uric acid metaphylaxis: 6.5–6.8 – Uric acid litholysis: 7.0–7.2 – Cystine metaphylaxis: 8.0–8.5	Dose depends on urinary pH or acidosis Cave: phosphate precipitation possible in cystine metaphylaxis (→ high urinary pH)	Calcium oxalate, Uric acid, Cystine, Non-infection-associated calcium phosphates
Allopurinol	– Lowering of hyperuricosuria – Lowering of hyperuricemia	100–300 mg/day; children: 1–3 mg/kg BW/day (100–200 mg/day	Dose adjustment in renal insufficiency Cave: high dose	Uric acid, Calcium oxalate (only if uric acid elevated), Ammonium urate,

		in isolated hyperuricosuria)	may lead to xanthinuria	2,8-Dihydroxyadenine
Calcium (Ca)	– Lowers enteral hyperoxaluria	160 mg (≈100 mg Mg) with each meal; max 500 mg/day	Intake 30 min before each main meal Cave: hypercalciuria (→ testing)	Calcium oxalate
L-Methionine	– Urinary acidification – Regulate acid–base balance in RTA and metabolic acidosis	600–1500 mg/day *Target urinary pH*: 5.8–6.2	Cave: contraindicated in RTA Pointless in Ca phosphates unless infection present (→ supporting antibiosis)	Infectious stones, Ammonium urate, Calcium phosphate
Magnesium (Mg)	– Compensation of isolated hypomagnesiuria – Lowers enteral hyperoxaluria – Non-lithogenic vs. Ca	200–400 mg/day; children: 6 mg/kg BW/day	Dose reduction in renal insufficiency; intake with meals	Calcium oxalate
Sodium carbonate	– Urinary alkalinization – Compensation of hypocitraturia – Regulation of acid–base balance in RTA and metabolic acidosis	4.5 g/day *Target urinary pH*: see alkaline citrates	Dose depends on urinary pH or acidosis	Calcium oxalate, Uric acid, Cystine
Pyridoxine (Vitamin B6)	– Lowering of endogenous hyperoxaluria	Initially 5 mg/kg BW/day; max 20 mg/kg BW/day	If no effect, discontinue after 1 year Cave: polyneuropathy	Calcium oxalate
Thiazide (Hydrochlorothiazide)	– Increase tubular Ca reabsorption in hypercalciuria (>8 mmol/day) → lowers renal Ca excretion	12.5–50 mg/day (gradually increase); children: 0.5–1 mg/kg BW/day	Cave: ↓ glucose tolerance, ↑ uric acid Cave: hypotension, K^+ loss, hypocitraturia	Calcium oxalate, Calcium phosphate
Tiopronin	– Converts poorly soluble cystine → soluble cysteine + cysteine–drug complex	Initially 250 mg/day; max 2000 mg/day	Cave: tachyphylaxis, proteinuria	Cystine

Conclusion and future perception

The current review article summarized new insights into kidney stone disease-related metabolic risk factors, receptors, promoters, and inhibitors. It also examined the roles of immune response, microbiome, and sex hormones in the development and formation of stones. Crystallization procedures are insufficient to fully explain the pathophysiology of kidney stone disease.

However, several kidney stone production study areas are still poorly known and were not covered here due to current research limitations. In order to create new preventative and therapeutic strategies, more thorough research is required to clarify the mechanisms of the microbiome and immune response in kidney stone formation.

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