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Review Article

Medicinal Plants in the Management of Urolithiasis: A Review

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ABSTRACT

Urolithiasis, or urinary stone disease, is a common and recurrent urological disorder affecting the kidneys, ureters, bladder and urethra, with a prevalence of 1% to 20% in developed countries and ten-year recurrence rates of about 60%. Calcium oxalate is the most frequent stone type, and formation follows a multifactorial pathway of supersaturation, nucleation, crystal growth, aggregation and retention, influenced by dietary, metabolic, genetic and anatomical factors. Conventional management includes medical expulsive therapy, chemolysis, shock wave lithotripsy, ureteroscopy and percutaneous nephrolithotomy; however, these approaches are costly, may cause complications, and do not fully prevent recurrence. Consequently, interest in plant-based therapy has grown. This review discusses the epidemiology, types, risk factors, pathophysiology, clinical features, diagnosis and conventional treatment of urolithiasis, followed by the principal mechanisms of anti-urolithiatic activity: diuresis, inhibition of crystal nucleation, growth and aggregation, antioxidant action and anti-inflammatory effects. Eight medicinal plants are reviewed in terms of botany, phytochemistry, experimental evidence and safety: *Phyllanthus niruri*, *Nigella sativa*, *Tribulus terrestris*, *Bryophyllum pinnatum*, corn silk, *Aerva lanata*, *Asparagus racemosus* and *Ammi visnaga*. Polyherbal formulations such as Subap Plus and Mofatet showed encouraging trial results, whereas Cystone® did not show efficacy in a small crossover trial. *Phyllanthus niruri* has the most human data, while evidence for most other plants is largely preclinical. Standardization, rigorous clinical trials and long-term safety evaluation are needed before herbal agents can be recommended for clinical use.

INTRODUCTION:

Urolithiasis, or urinary stone disease, is the presence of stones in the upper (kidneys, ureter) or lower (bladder, urethra) urinary tract. In economically developed countries its prevalence ranges from 1% to 20%, and relapse within ten years is about 60% [1,2]. Occurrence varies with geography, age, sex, nutrition and climate

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[2,3], and men are affected more often than women [4]. Stones may cause acute pain, haematuria and infection, and in chronic cases irreversible renal damage [5]. Treatment planning depends on the size, location and number of stones, urinary anatomy, comorbidities, age and performance status [2].

Calcium oxalate stone formation begins with nucleation, crystal growth and aggregation. Urinary supersaturation is shaped by promoters (low urine volume; high urinary calcium, oxalate and urate) and inhibitors (citrate, magnesium, potassium, nephrocalcin, urinary prothrombin fragment-1 and osteopontin) [6]. Because diet strongly influences formation and recurrence, current advice is to increase fluid intake, keep calcium intake balanced, limit sodium and animal protein, and eat more fruit and fibre. Owing to the side effects associated with drugs and surgery, many patients prefer plant-based therapy, which is widely regarded as safe, effective and free of undesirable effects [7].

The management of urolithiasis is also a socioeconomic concern [2]. Medicinal plants act through multiple mechanisms that target several pathways of stone formation at once, are generally linked to fewer side effects and better tolerance, are affordable and accessible (particularly in developing nations where traditional medicine remains the main source of healthcare), and may be used for long periods to prevent recurrence. Their wider use, however, requires that lack of standardization, variable phytochemical composition and scarcity of clinical data be addressed [8].

2. EPIDEMIOLOGY:

Kidney stone disease is more prevalent in hot areas and typically occurs between 20 and 60 years of age. About 10% of people are affected at some stage of life; 50% experience a recurrence within 5–10 years and 75% within 20 years. Over the past 30 years incidence has risen in developed countries, especially among women, whose incidence is now almost identical to that of men [9]. Reported prevalence is 7–13% in North America, 5–9% in Europe and 1–5% in Asia [10]. A recent meta-analysis of 22 observational studies (1,276,826 adults) gave a pooled global prevalence of 10.85% (95% CI 8.76–13.14%), higher in males (12.93%) than in females (8.91%) and ranging from 22.3% in Africa to 8.3% in North America, with very high heterogeneity; after adjusting for diagnostic method and sampling strategy, the apparent rise over time was not significant, suggesting it reflected changes in study methods [5].

3. TYPES OF STONES:

The chemical composition of kidney stones reflects anomalies in urine chemistry, and stones differ in size, shape and mineralogy [11]. The main types are summarised in Table 1.

Table 1: Types of Renal Stones, Their Composition and Incidence [11]

No.	Stone	Composition	Approx. incidence
1	Calcium oxalate	Calcium; oxalate	~70%
2	Calcium phosphate	Calcium; phosphate	~10%
3	Struvite	Ammonia; calcium; phosphate	~10%
4	Uric acid	Uric acid	~5–10%
5	Cystine	Cystine	<1%
6	Silicate / iatrogenic (medication-induced)	Depends on the agent (e.g. acetazolamide, ciprofloxacin, ephedrine, guaifenesin, indinavir, silica, triamterene)	<1%

4. RISK FACTORS:

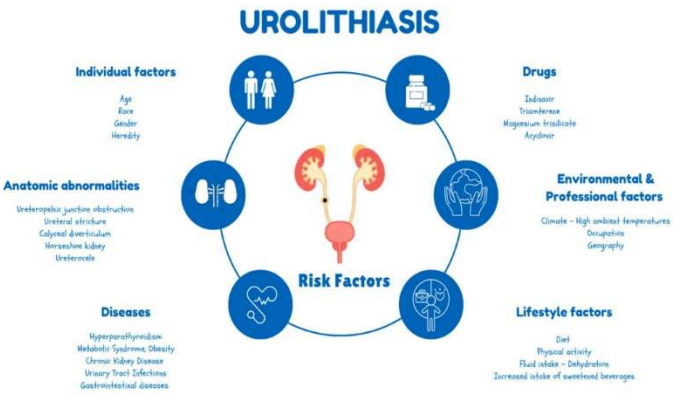

Figure None: Schematic Representation of Risk Factors Associated with Urolithiasis.[2]

Risk factors are either modifiable or non-modifiable (Table 2). Low fluid intake (24-h urine volume <2.0 L) concentrates urine and promotes supersaturation. Dietary oxalate (spinach, nuts), sodium, animal protein and fructose increase lithogenic potential, whereas inadequate dietary calcium paradoxically raises risk by allowing greater intestinal oxalate absorption. Hyperparathyroidism, inflammatory bowel disease, gout and metabolic acidosis predispose to specific stone types, and familial and Mendelian disorders (cystinuria, primary hyperoxaluria, distal renal tubular acidosis) are linked to early-onset, recurrent stones.

Table 2: Non-modifiable and Modifiable Risk Factors for Kidney Stone Formation

Type	Risk factor	Pathophysiological impact
Non-modifiable	Genetic mutations (SLC3A1, SLC7A9, CLDN14); family history	Alter tubular solute handling and urine composition; raise risk of cystine and calcium stones; strong familial clustering
	Age and sex	Male predominance; post-menopausal hormonal shifts raise risk in women
	Anatomical abnormalities	Medullary sponge kidney and horseshoe kidney promote urinary stasis
Modifiable	Low fluid intake	Concentrated urine and increased supersaturation
	High sodium diet	Promotes calciuria by impairing calcium reabsorption
	Excess animal protein	Raises acid load and calcium excretion; lowers citrate
	High dietary oxalate	Promotes calcium oxalate crystallization
	Obesity, metabolic syndrome, diabetes	Acidic urine (low pH), hyperuricosuria; promote uric acid and calcium stones
	Medications (e.g. topiramate, diuretics)	Alter urinary pH and solute excretion

5. PATHOPHYSIOLOGY:

Stone formation is a multifactorial process involving urinary chemistry, renal physiology, genetics and environment, and proceeds through supersaturation, nucleation, crystal growth, aggregation and crystal retention [12]. Crystals form in supersaturated urine and adhere to the urothelium, creating a nidus for further growth. Many, but not all, calcium oxalate stones develop on Randall's plaques, composed of calcium phosphate (hydroxyapatite), which erode the urothelium and form a nucleus for calcium oxalate deposition. More recent theories emphasise cell-surface molecules that favour or inhibit crystal adhesion; urothelial injury and repair after a stone episode may increase their expression, so that "stones beget stones". Prevention therefore focuses on identifying and correcting the risk factors for crystal formation [13].

6. CLINICAL MANIFESTATION AND DIAGNOSIS:

Stones typically present with sudden severe flank pain (renal colic) that radiates towards the groin and changes

location as the stone migrates, together with gross or microscopic haematuria, dysuria, urinary urgency, nausea and vomiting, fever, chills and difficulty passing urine. Complicated obstruction may lead to hydronephrosis, urinary infection and renal impairment [14].

Ultrasound is the primary imaging tool but should not delay emergency care; KUB radiography distinguishes radiopaque from radiolucent stones and aids follow-up; non-contrast CT is the standard modality for acute flank pain after ultrasound, assessing stone location, burden, density and anatomy. Laboratory work-up comprises urinalysis and 24-hour urine collection, blood tests (creatinine, uric acid, ionised calcium, sodium, potassium, blood count, C-reactive protein) and stone analysis [15].

7. CONVENTIONAL MANAGEMENT AND TREATMENT:

Conventional treatment aims to relieve symptoms, clear calculi and limit recurrence [16]. Depending on stone size, composition, location and the patient's profile, it comprises medical care, minimally invasive procedures and surgery [17].

Medical management. This is the first line for small, simple stones [18], and patient education is an important component [19]. *Medical expulsive therapy* with α-blockers (an off-label class effect) is effective for ureteral stones, with greatest benefit for stones >5 mm in the distal ureter [15]. *Analgesia* relies on NSAIDs such as ibuprofen and diclofenac, with opioids under close monitoring for extreme pain and acetaminophen when NSAIDs are contraindicated [20]. *Oral chemolysis* is strongly recommended for uric acid stones by alkalinisation with sodium bicarbonate or citrate (urine pH 7.0–7.2), with a reported success rate of 80.5% and 15.7% of patients needing additional care [15]. *Dietary and lifestyle measures* include fluid intake giving at least 2.5 L/day of urine, reduced sodium, moderate protein, adequate calcium and restricted oxalate (spinach, almonds) [21,22], together with weight control and exercise [12].

Minimally invasive techniques. For stones too large or resistant for medical therapy, these procedures work well for stones smaller than 20 mm with little morbidity [23,24]. Extracorporeal shock wave lithotripsy (ESWL) fragments stones with acoustic shock waves; being non-invasive, low-cost and associated with fewer complications and shorter hospital stays, it is first-line for renal, proximal and mid-ureteral stones [12,25]. Ureteroscopy removes stones with baskets or breaks them by laser lithotripsy and has a high stone-free rate for lower ureteral stones [26].

Surgical interventions. Percutaneous nephrolithotomy (PCNL) is the gold standard for staghorn calculi and stones >20 mm; clearance rates are high, but bleeding, infection and injury to adjacent structures are risks, which smaller devices and robotic assistance have helped reduce [27,28]. Open or laparoscopic surgery is rarely needed, for very large or anatomically difficult stones [29].

8. MEDICINAL PLANTS IN UROLITHIASIS AND MECHANISMS OF ANTI-UROLITHIATIC ACTIVITY:

An estimated 80% of the world's population relies on traditional medicine, and at least one third of all medicinal products have plant origin. Medicinal plants are regarded as an acceptable, cheap, easily available source of active compounds [30], and phytotherapeutic agents could be used as an alternative or adjunct in urolithiasis [8]. Their activity involves several mechanisms:

- **Diuresis:** increased urine flow lowers supersaturation by diluting calcium, oxalate and uric acid, and removes small crystals before they are retained in the tubules [31].
- **Inhibition of crystal nucleation:** reduced supersaturation and interference with the initial aggregation of calcium and oxalate ions; magnesium and citrate form soluble complexes with calcium, lowering free ion availability [8].
- **Inhibition of crystal growth and aggregation:** inhibitory molecules adsorb to crystal surfaces, block deposition of new ions, alter crystal shape and reduce adhesiveness [32].
- **Antioxidant activity:** reactive oxygen species damage tubular epithelium and expose crystal-binding sites; polyphenols, flavonoids and tannins

scavenge free radicals and boost endogenous defences, reducing crystal adherence [33].

- **Anti-inflammatory activity:** suppression of prostaglandins, interleukins and TNF-α reduces renal inflammation and the creation of crystal-adhesion sites; many herbs combine antioxidant and anti-inflammatory properties [8,31].

9. ANTI-UROLITHIATIC MEDICINAL PLANTS:

9.1 *Phyllanthus niruri* Linn. (Phyllanthaceae)

Botany and traditional use. Stone breaker, chanca piedra, quebra-pedra, and bhumyamalaki are names for this tiny, annual, erect herb (30–50 cm) found in the tropics and subtropics. Its aerial parts are employed in South American traditional medicine for kidney stones and gravel, and in Ayurveda for jaundice, dyspepsia, and urinary ailments [34, 35]. When evaluating the literature, it is important to keep in mind taxonomic confusion with allied species (P. amarus, P. fraternus, P. urinaria). phytochemistry. Lignans (phyllanthin, hypophyllanthin, niranthin), flavonoids (quercetin, rutin), ellagitannins (geraniin, corilagin), securinine-type alkaloids, phenolic acids, and triterpenes are the main types [34, 35]. In experimental models, a number of lignans reduced uric acid [36].

Evidence. In vitro, aqueous extracts suppressed the formation of calcium oxalate crystals, changed their morphology and aggregation, and decreased renal tubular cells' endocytosis of crystals [37, 38]. In rats, the extract decreased the quantity and size of renal crystal deposits [38]. P. niruri has the most human data of all the plants reviewed, albeit it is limited. Treatment changed urinary calcium, citrate, and magnesium in stone formers [39], normalized urinary calcium in hypercalciuric patients [40], and increased the stone-free rate following ESWL in a randomized long-term study, particularly in patients with residual fragments [41]. Inhibition of crystal formation and aggregation, decreased crystal adhesion and endocytosis, hypocalciuric and inhibitor-modifying effects, and relaxing of ureteric smooth muscle with antioxidant, anti-inflammatory, and uric acid-lowering effects are the suggested mechanisms [35, 36, 42].

Safety. Acute rodent toxicity is low, but no long-term safety data exist; botanical identity must be controlled, and additive effects with antihypertensive and hypoglycaemic agents and effects on hepatic drug metabolism are possible [35].

9.2 *Nigella sativa* Linn. (Ranunculaceae)

Botany and phytochemistry. Native to southwest Asia and the Mediterranean, black seed (kalonji) is an annual herb used for respiratory, digestive, and urinary issues in Unani, Ayurvedic, and Islamic medicine [43,44]. Alkaloids (nigellicine, nigellidine), saponins, flavonoids, and tocopherols are also found in the seeds, along with fixed oil (28–36%) rich in linoleic and oleic acids and volatile oil (0.4–2.5%) with thymoquinone as its primary bioactive component [43–45].

Evidence. Relevant effects include diuretic and antispasmodic actions, anti-inflammatory activity through suppression of eicosanoid production and cytokine signaling, and antioxidant protection of the kidney against toxin-induced oxidative harm [43–45]. An ethanolic seed extract decreased the quantity and amount of calcium oxalate deposits in rats with calculi caused by ethylene glycol [46]. Oral seeds and tamsulosin were evaluated in a randomized trial by Shakeri et al. to evaluate pain alleviation and stone passage in patients with kidney and ureteral stones measuring 4–10 mm; the results pertain to existing stones rather than preventing recurrence [47].

Safety. While thymoquinone has a smaller therapeutic margin, seeds and oil are well tolerated at dietary dosages (allergic contact dermatitis has been recorded; hypotensive and hypoglycemic effects may potentiate prescription medicines; pregnancy concerns occur at very high doses) [43–45]. Standardized protocols and larger studies are required [47].

9.3 *Tribulus terrestris* Linn. (Zygophyllaceae)

Botany and phytochemistry. The prostrate annual gokshura, also known as the pierce vine or caltrop, grows in warm, arid climates. Chinese medicine uses it for urogenital problems and hypertension, whereas Ayurveda uses it as a dashamoola and mutral (diuretic) medication for calculi and dysuria [48]. Steroidal saponins (diosgenin, tigogenin, hecogenin; protodioscin as a marker chemical), flavonoids, alkaloids,

and tannins are characteristic elements; the saponin profile varies significantly with regional origin [48,49].

Evidence. Similar to Bergenia ligulata, extracts reduced the development of calcium oxalate monohydrate crystals in vitro [50], and an aqueous extract shielded renal tubular cells from oxalate damage [51]. The plant altered oxalate metabolism in rats [53], and certain fruit fractions were effective against induced urolithiasis in rats, indicating particular components rather than nonspecific diuresis [52]. A comparison with maize silk revealed diuretic and smooth-muscle effects [54].

Safety. Saponin-rich sports items may differ from the conventional medication, but toxicity has been recorded in grazing animals at high consumption [48]. Traditional doses are often well tolerated. There is insufficient evidence for an antilithiatic effect in humans.

9.4 *Bryophyllum pinnatum* (Lam.) Oken (Crassulaceae)

Botany and phytochemistry. The succulent perennial air plant, also known as miracle leaf (Hindi patharchur, "stone-breaker"), is used to treat wounds, inflammation, infections, and renal and urinary diseases, especially kidney stones. Kalanchoe pinnata is sometimes used as a synonym. Because citrate can complex calcium, the flavonoids (quercetin, kaempferol), bufadienolides, phenolic acids, triterpenoids, tannins, mucilage, and organic acids (malic, isocitric, and citric) found in the leaves are of interest [55].

Evidence. Leaf extracts have been shown to have anti-inflammatory, diuretic, and antioxidant properties [55,56], and they shielded rats against gentamicin-induced nephrotoxicity [56]. The evidence is encouraging but preliminary because there are limited reports of antiurolithiatic effects in rats treated with ethylene glycol that lack methodological detail and replication.

Safety. Because the bufadienolides are cytotoxic and cardioactive steroids, they should be used alongside digoxin and at large doses in cases of heart disease [55].

9.5 Corn silk (*Zea mays* L., Poaceae)

Botany and phytochemistry. The stigmas and styles of the maize flower make up corn silk (Stigma maydis), which is used in traditional medicine to treat urinary stones, cystitis, prostatitis, edema, and as a moderate diuretic [57]. It has high radical-scavenging activity [58] and includes flavonoids (maysin, luteolin derivatives, rutin), phenolic acids, tannins, saponins, polysaccharides, vitamins C and K, allantoin, sterols, and a lot of potassium [57,58].

Evidence. Zea mays changed urinary risk factors for stones in rats [60], and extracts were diuretic in rats, altering glomerular filtration and raising urine potassium excretion [59]. While the antioxidant flavonoids may shield tubular cells from oxalate damage, increased urine flow dilutes lithogenic ions. **Limitations and safety.** Corn silk has been lightly researched in stone models and no controlled human study exists; its best-supported activity, mild diuresis, may be obtained with water, therefore it is a supportive adjunct recommended largely for safety. It is safe as a tea, but caution is advised with potassium-sparing diuretics, in kidney failure and with antihypertensive or antidiabetic medicines [57].

9.6 *Aerva lanata* L. (Amaranthaceae)

Botany and phytochemistry. One of Kerala's dashapushpam herbs, it is a prostrate or erect perennial found in tropical Asia, Africa, and Australia (gorakhbuti, polpala). In Sri Lankan and South Indian medicine, a decoction is a common treatment for ashmari, while roots and aerial portions are used for kidney stones, urinary infections, edema, and diabetes [61]. It includes alkaloids (derivatives of canthin-6-one), flavonoids (kaempferol, quercetin, isorhamnetin), saponins, tannins, and triterpenes [61,62]. **Evidence.** The extract improved renal biochemistry, decreased renal crystal deposition, and decreased urine excretion of lithogenic ions in rats with induced calcium oxalate urolithiasis [61]. This is the main primary report, and the plant has not been evaluated in people. A supplementary nephroprotective mechanism is suggested by the preservation of renal function in cisplatin and gentamicin animals [63]. **Safety.** Rodent studies show a wide safety margin but no systematic human toxicology exists; concomitant antidiabetic drugs or diuretics should be monitored [62,63].

9.7 Shatavari (*Asparagus racemosus* Willd., Asparagaceae)

Botany and phytochemistry. A tuberous-rooted, spiky shrub found in India, Sri Lanka, and the Himalayas, it is primarily used as a galactagogue and female reproductive tonic. Ayurveda also categorizes it as a diuretic and rasayana, and it has been used to treat urinary calculi and dysuria [64]. Steroidal saponins (shatavarins, sarsasapogenin glycosides), isoflavones, alkaloids (racemosol), flavonoids, inulin-type polysaccharides, mucilage, and the antioxidant racemofuran are all present in the roots [64,65].

Evidence. A root extract increased renal antioxidant status and decreased calcium, oxalate, and crystal formation in the urine and kidneys of male Wistar rats with ethylene glycol-induced lithiasis [66]. Although there is little concrete evidence, saponins and mucilage may impede the formation of crystals. **Safety.** Although generally thought to be safe, allergic reactions and gastrointestinal issues can arise; long-term usage in hormone-sensitive conditions requires caution due to the saponins' oestrogen-like activity; and their diuretic impact may intensify other diuretics [64].

9.8 *Ammi visnaga* (L.) Lam. (Apiaceae)

Botany and phytochemistry. Since ancient times, the Mediterranean and Egyptian herb Khella (Al-Khillah), a member of the carrot family, has been used to treat renal colic and ureteric stone passage. Its furochromone, khellin, was the primary ingredient in cromolyn and amiodarone. The fruits include coumarins, flavonoids, essential oil, khellin and visnagin (0.3–1.2%), and the pyranocoumarins visnadin and samidin [67].

Evidence. Both the kidney and the ureter are affected by its antilithiatic action: khellin is a well-known smooth-muscle relaxant that is used to treat renal colic [67,68], whereas visnagin relaxes smooth muscle by blocking calcium influx [68]. The extract and its constituents, khellin and visnagin, prevented oxalate-induced damage to renal epithelial cells in culture [70], and treatment inhibited oxalate nephrolithiasis in hyperoxaluric rats [69]. This constituent-level attribution is uncommon but results from cell culture and requires in vivo confirmation.

Safety. Khellin caused nausea, vomiting, headache, dizziness, sleeplessness, and reversible liver-enzyme increase at the high mid-twentieth-century doses used for coronary vasodilation, which limited its usage. Photosensitization and interactions with anticoagulants and antihypertensives have also been proposed. It is better to think of it as a pharmacologically active medication rather than a harmless tea [67].

10. POLYHERBAL FORMULATIONS:

According to conventional wisdom, combining many medications results in synergy, boosts efficacy, balances the effects of different herbs, and lowers toxicity. A common stone composition, available as tablets, capsules, syrups, decoctions, bhasma or guggulu preparations, combines a diuretic, a litholytic or crystal inhibitor, an antispasmodic, an antioxidant, and an anti-infective drug [71,72]. The composition, safety, and supporting data of seven commercially available or researched formulations are compiled in Table 3.

Table 3: Polyherbal Formulations Used in Urolithiasis

Formulation (system) and main ingredients	Model, dose, duration	Key findings	Safety
Subap Plus (Ayurveda): *Crateva nurvala* bark, *Musa paradisiaca*, *Hordeum vulgare* and *Achyranthes aspera* preparations; 500 mg capsule [73]	Double-blind placebo-controlled RCT; renal calculi 4–9 mm; 65 completers; 1 capsule twice daily, 6 months	Stone surface area 27.6→21.7 mm² (p = 0.024), unchanged in placebo; expulsion 20.6% vs 3.2% (p = 0.03); VAS pain 6.9→1.8 vs 7.2→6.8	No major adverse events; mild belching in 3 patients; rodent LD50 >2000 mg/kg

Formulation (system) and main ingredients	Model, dose, duration	Key findings	Safety
Mofatet (Traditional Persian Medicine): equal parts seeds of *Cucumis sativus*, *Cucurbita pepo*, *Apium graveolens*, *Foeniculum vulgare*, *Pimpinella anisum* and *Tribulus terrestris* fruit [74]	Double-blind placebo-controlled RCT; calcium stones ≥5 mm; 49 completers; 500 mg twice daily, 5 weeks	Stone size 8.15→3.2 mm (−60.73%, $p < 0.001$), two stones disappeared; urinary calcium fell ($p = 0.02$), magnesium rose ($p < 0.001$); serum calcium 9.13→9.82 mg/dL	No significant change in BUN, creatinine, AST, ALT; no clinically significant reactions
Safoof-e-Pathar Phori (Unani): *Didymocarpus pedicellatus* (50 g/100 g), *Hordeum vulgare*, *Rheum webbianum*, *Macrotyloma biflorum*, *Raphanus*, potassium nitrate [75]	Rats, ethylene glycol + ammonium chloride; 700 and 1000 mg/kg, 21 days; standard Neeri	Urinary calcium 8.90→5.95 and 5.28 mg/dL; BUN 134.7→43.3 and 23.7 mg/dL; creatinine 0.69→0.27 and 0.20 mg/dL; no crystals in treated groups	90-day study at 700 mg/kg: no mortality or abnormal histology
Neeri (Ayurveda): syrup of 19 ingredients incl. *Boerhavia diffusa*, *Tribulus terrestris*, *Crataeva nurvala*, *Bergenia ligulata*, shilajeet [76]	In vitro: crystallization, radical scavenging, NRK-52E cells; 250–1500 µg/mL	Crystallization inhibition 50.7→88.1%; cell viability under oxalate injury 46.0→82.4% (500 µg/mL); LDH leakage 221.1→43.3 U/L	Heavy metals, aflatoxins, pesticides not detected
Gokshuradi (Ayurveda): 1:1:1:1:1 *Tribulus terrestris* fruit, *Hygrophila spinosa* seed, roots of *Ricinus communis*, *Solanum anguivi*, *S. surattense* [77]	Rats; 50 and 100 mg/kg, 21 days; standard Cystone	At 100 mg/kg urinary oxalate 2.12→0.68 mg, urinary calcium 1.84→3.16 mg; creatinine 1.43→0.89 mg/dL; BUN 57.0→20.5 mg/dL; crystals in 1/6 high-dose rats	No dedicated toxicity study
Calcury (Ayurveda): *Bergenia ligulata*, *Boerhavia diffusa*, *Crateva nurvala*, *Tribulus terrestris* [78]	Open-label single-arm trial; n = 50; 2 tablets twice daily, 4 weeks	Stone size 6.5→4.0 mm ($p = 0.001$); number 3.5→2.1; passage 60→90% ($p = 0.03$); VAS 6.5→3.2	Haematological, renal, hepatic parameters stable
Cystone® (Ayurveda): *Didymocarpus pedicellata* 130 mg, *Bergenia ligulata* 98 mg, plus seven other ingredients (26–32 mg each) [79]	Double-blind placebo-controlled crossover, 10 recurrent calcium stone formers; 6 weeks each, open extension to 52 weeks	No significant change in urinary supersaturation; stone volume 93→114 mm^3 ($p = 0.07$); 8 kidneys increased, 8 unchanged, 2 decreased; authors conclude no support for efficacy	No adverse effects attributed

The most clinically supported of these formulations are Subap Plus and Mofatet, whose suggested mechanisms are mostly derived from the herbs that make them up. It is believed that Subap Plus, an Ayurvedic preparation of extracts from Crateva nurvala, Musa paradisiaca, Achyranthes aspera, and Hordeum vulgare, works in concert at various stages of stone formation (nucleation, crystal growth, aggregation, and retention) by reducing the rate of stone-forming salts and increasing urinary citrate excretion. H. vulgare also has an anti-inflammatory effect. Animal research indicates a reno-protective impact on renal cells damaged by crystal deposition, and analgesic activity has also been shown. Litholytic and expulsive activity may account for the trial's decreased stone size and increased stone passage.

Kidney and bladder stones are usually treated with mofatet powder from traditional Persian medicine. Its

components are thought to be beneficial: Apium graveolens is a diuretic, Tribulus terrestris dissolves stones through an oxalate-converting protein, Cucurbita pepo seed extract may prevent all stages of calcium oxalate stone formation, and several components (C. pepo, Cucumis sativus, Foeniculum vulgare, Pimpinella anisum) are reported to protect the kidney. Reduced supersaturation and crystal inhibition are consistent with the rise in urine magnesium and reduction in urinary calcium in the clinical trial, but renal and hepatic function did not change . Further in vitro and in vivo research is necessary to confirm the precise processes of both formulations.[12]

11. BIOACTIVE PHYTOCHEMICALS IN THE PREVENTION OF UROLITHIASIS:

The pharmacologically active phytochemicals found in medicinal and nutritional plants are largely responsible for their therapeutic and preventative actions, while the molecular mechanisms behind the majority of these activities are still unknown. Because oxalate and calcium oxalate crystals produce free radicals and lipid peroxidation, lower cellular antioxidant defenses, and damage renal epithelial cells, the anti-lithiatic effect is believed to primarily result from strong antioxidant activity. Antioxidant therapy may therefore restrict crystal nucleation, binding, and growth. The most prevalent antioxidants in the human diet, dietary polyphenols, also have anti-inflammatory properties against renal impairment linked to oxidative stress.[7] Below is a summary of five of the most powerful polyphenols: catechin, epigallocatechin, diosmin, quercetin, and rutin.

11.1 Catechin

The main catechins in green tea are epigallocatechin, epicatechin, and epigallocatechin-3-gallate (EGCG), which makes up 50–80% of the total [7]. Catechins are a flavonoid subclass of tea polyphenols having antioxidant and cytoprotective qualities. By scavenging superoxide produced by NADPH oxidase, they aid in the restoration of the normal mitochondrial membrane. They also function as metal chelators, radical scavengers, and indirectly influence enzymes and transcription factors [7]. Catechin decreased oxidative damage and crystal deposition markers (MDA, osteopontin, and 8-OHdG) and raised superoxide dismutase (SOD) activity in rats with ethylene glycol-induced nephrolithiasis and calcium oxalate monohydrate (COM)-treated NRK-52E renal proximal tubular cells . This limited renal calcium crystallization. Consuming catechin has also been shown to slow the development of COM papillary deposits and renal papillary calcification [80]. In keeping with the antilithiatic activity of green tea mentioned in Section 12 , these results suggest catechin, particularly EGCG, as a preventative agent for urinary stones.[7]

11.2 Epigallocatechin

The main catechins in green tea (Camellia sinensis), together with epicatechin gallate and epicatechin, are epigallocatechin (EGC) and its gallate ester, epigallocatechin-3-gallate (EGCG), which shield renal cells from oxalate-induced damage . Although green tea's anti-lithogenic and antioxidant properties have made it a dietary supplement of interest in nephrolithiasis, it is not advised for those with calcium oxalate stones due to its high oxalate content . Supplementing with green tea in rats inhibited kidney crystal growth, decreased oxalate excretion and brushite supersaturation, downregulated osteopontin, increased superoxide dismutase activity, elevated Bcl-2 expression, and decreased the apoptotic index; these effects were primarily attributed to the tea's catechins. Additionally, EGCG directly affects the interaction between crystals and cells: by reducing the surface expression of the crystal-binding protein α-enolase, it decreased the binding of calcium oxalate monohydrate (COM) crystals to renal tubular (MDCK) cells [84]. EGCG is thought to be a promising dietary agent for avoiding urolithiasis, similar to catechin (Section 11.1), albeit the majority of the evidence comes from cell and animal research.[81]

11.3 Diosmin

Citrus fruits and vegetables contain the flavonoid glycoside diosmin . It shields the kidneys against oxidative stress, nephrotoxicity, and diabetic nephropathy and is frequently used as a natural remedy for renal disorders. Suppression of lipid peroxidation, enhancement of antioxidant enzymes, and regulation of Bax and p53 protein expression are all thought to contribute to its renoprotective properties .[7] Diosmin prevented the

development of calcium oxalate stones in a rat model of ethylene glycol-induced nephrolithiasis by decreasing capillary hyper-permeability, slowing down glomeruli and tubule degeneration, and increasing the diameter of capillaries and arteries in the cortex .[82] Additionally, by maintaining an acidic urine pH, it inhibited the crystallization of stone-forming promoters, increased urine volume, elevated urine potassium and magnesium, and lower urine protein.[7]

11.4 Quercetin

Numerous fruits and vegetables contain quercetin, a bioflavonoid with anti-inflammatory, antioxidant, diuretic, and hypouricemic qualities. Phyllanthus niruri, Bryophyllum pinnatum, and Aerva lanata (Sections 9.1, 9.4 and 9.6) also contain quercetin. Quercetin's antioxidant and anti-apoptotic actions, which were mediated by elevated catalase and SOD activity, protected renal tubular cells in a rat model of calcium oxalate stone formation.[7] When taken orally in experimental hyperoxaluria, it decreased MDA and nitric oxide levels as well as oxidative and inflammatory alterations in renal tissue. Free-radical scavenging, metal-ion chelation, prevention of lipid peroxidation, and enhanced intracellular glutathione and antioxidant enzyme activity via the p38-MAPK pathway are some of its antioxidant mechanisms. Therefore, quercetin might be a good treatment for recurrent calcium oxalate stones, but further research and clinical validation are needed.[83]

11.5 Rutin

Apple peel, black tea, medicinal herbs including Herniaria hirsuta and Cynodon dactylon, and P. niruri have all been shown to contain rutin, a flavonoid antioxidant (Sections 9.1 and 9.5) . It inhibits xanthine oxidase, an enzyme that produces reactive oxygen species, increases glutathione production, catalase and SOD expression, and directly scavenges reactive oxygen species . Rutin, either by itself or in combination with curcumin, reduced lipid peroxidation, affected COM crystal aggregation and glomerular filtration rate, and corrected urinary calcium and oxalate levels in rats with calcium oxalate urolithiasis . Like the other phytochemicals mentioned here, the evidence supporting rutin as a plant bioactive for urolithiasis prevention is mostly preclinical.[7]

12. RECENT LITERATURE:

Recent reviews broadly agree on the promise and limits of phytotherapy. Chobhe et al. (2026) classified synthetic and herbal anti-urolithiatic agents and the models used to test them, highlighting plants and phytoconstituents as safer, cost-effective alternatives [31]. Mutomba et al. (2025) reviewed 68 articles, mainly randomized trials, of single extracts (*P. niruri*, *N. sativa*, *Phaseolus vulgaris*) and formulations (Cystone®, Subap, Wu-Ling-San, Renalof®); most reported reduced stone size, better expulsion and favourable urinary parameters, but Cystone® and Wu-Ling-San showed no benefit in some trials, and the authors cautioned that “natural” does not mean “safe” [2]. Allam et al. (2025) systematically reviewed 14 randomised trials; many herbal treatments raised urinary citrate, lowered urinary calcium and increased stone passage, *Alhagi maurorum* acted like hydrochlorothiazide, and *N. sativa* equalled or bettered tamsulosin for pain and stone passage [12]. The European Association of Urology guidelines give herbal treatments little attention for lack of strong evidence [15], and Balawender et al. (2024) stress fluid intake (urine output ≥2.0–2.5 L/day), calcium intake of 1000 –1200 mg/day, reduced sodium and individualised diet as priorities for reducing recurrence [19]. Oswal et al. (2023) found antilithiatic effects for pomegranate, green tea, *N. sativa*, lemon, cranberry, *P. niruri*, *A. lanata*, *A. racemosus* and *T. terrestris*, mostly in preclinical studies [7].

13. FUTURE PERSPECTIVES AND RESEARCH GAPS:

Most evidence comes from cell-line and rat models, with few controlled human trials, and the literature has mainly reported litholytic effects and urinary parameters; oxidative stress and inflammation, central to stone formation, have been studied in very few reports. The pathogenesis itself, including the role of Randall’s plaque and matrix molecules, is incompletely understood, and the active compounds of plants such as *P. niruri*, and why urinary calcium falls only in hypercalciuric patients, remain unclear. Available evidence

suggests these plants may prevent stone growth rather than dissolve preformed stones, and clinical results are inconsistent. Heterogeneity in plant parts, extraction methods, doses and phytochemical characterization prevents comparison, safety and toxicity data are incomplete, and many studies lack quality or risk-of-bias assessment. Epidemiological estimates are weakened by non-standardized diagnostics, few CT-based studies and under-representation of regions such as Africa and the Caribbean, while individualised and microbiota-based dietary strategies and paediatric urolithiasis remain underexplored.

Future work should therefore combine well-designed preclinical studies and randomized controlled trials to confirm efficacy and prevention of recurrence [2,84]; standardize extracts with validated phytochemical markers and reproducible extraction and quality-control methods [7,85]; clarify molecular mechanisms, bioavailability, pharmacokinetics, long-term toxicity and drug interactions; and use bioassay-guided fractionation, biotechnology, nanotechnology and novel drug-delivery systems to discover and improve plant-derived agents [2,7,84,85].

CONCLUSION:

Urolithiasis is a common, multifactorial disease. Calcium oxalate is the most frequent stone type, and recurrence is high, at about 60% over ten years. Conventional treatments such as medical expulsive therapy, shock wave lithotripsy, ureteroscopy and percutaneous nephrolithotomy remove stones effectively. They are costly, carry procedural risk and do not reliably prevent recurrence. Medicinal plants are therefore a promising complementary option, acting through several mechanisms: diuresis, inhibition of crystal nucleation, growth and aggregation, antioxidant and anti-inflammatory effects, and smooth-muscle relaxation. Among the eight plants reviewed, *Phyllanthus niruri* has the most human data. *Nigella sativa* and *Ammi visnaga* also show useful activity, while the evidence for the remaining plants is mostly preclinical. Phytochemicals such as catechin, epigallocatechin, diosmin, quercetin and rutin help explain these effects. Polyherbal formulations such as Subap Plus and Mofatet gave encouraging trial results, but Cystone® showed no efficacy, so results are inconsistent. Most evidence comes from cell-line and rat studies, extracts are not standardized, and long-term safety and herb–drug interaction data are lacking. Standardized extracts, well-designed randomized trials and long-term safety evaluation are needed before clinical recommendation. Until then, herbal agents should support, not replace, proven urological treatment.

REFERENCES

1. Tiselius HG. Epidemiology and medical management of stone disease. BJU Int. 2003;91 (8):758-767. doi:10.1046/j.1464-410X.2003.04208.x.
2. Mutomba WF, Symeonidis EN, Mykoniatis I, Tzelves L, Tsaturyan A, Juliebo-Jones P, et al. Phytotherapy in urolithiasis: an updated overview of current knowledge. J Clin Med. 2025;14 (9):2885. doi:10.3390/jcm14092885.
3. López M, Hoppe B. History, epidemiology and regional diversities of urolithiasis. Pediatr Nephrol. 2010;25 (1):49-59. doi:10.1007/s00467-008-0960-5.
4. Kusz M, Alzubedi A, Popiołek J, Konopelko M. Prevalence, diagnosis and treatment in urolithiasis. J Educ Health Sport. 2018;8 (8):1143-1148. doi:10.5281/zenodo.1433726.
5. Vera-Ponce VJ, Sanchez-Tamay NM, Ballena-Caicedo J, Zuzunaga-Montoya FE, De Carrillo CIG, Poemape Mestanza RL. Global prevalence of urolithiasis: a meta-analysis accounting for methodological heterogeneity. Front Urol. 2025;5:1705953. doi:10.3389/fruro.2025.1705953.
6. Ferraro PM, Bargagli M, Trinchieri A, Gambaro G. Risk of kidney stones: influence of dietary factors, dietary patterns, and vegetarian-vegan diets. Nutrients. 2020;12 (3):779. doi:10.3390/nu12030779.
7. Oswal M, Varghese R, Zagade T, Dhatrak C, Sharma R, Kumar D. Dietary supplements and medicinal plants in urolithiasis: diet, prevention, and cure. J Pharm Pharmacol. 2023;75 (6):719-745. doi:10.1093/jpp/rgac092.

8. Butterweck V, Khan SR. Herbal medicines in the management of urolithiasis: alternative or complementary? Planta Med. 2009;75 (10):1095-1103. doi:10.1055/s-0029-1185719.
9. Moe OW. Kidney stones: pathophysiology and medical management. Lancet. 2006;367 (9507):333-344. doi:10.1016/S0140-6736 (06)68071-9.
10. Sorokin I, Mamoulakis C, Miyazawa K, Rodgers A, Talati J, Lotan Y. Epidemiology of stone disease across the world. World J Urol. 2017;35 (9):1301-1320. doi:10.1007/s00345-017-2008-6.
11. Alelign T, Petros B. Kidney stone disease: an update on current concepts. Adv Urol. 2018;2018:3068365. doi:10.1155/2018/3068365.
12. Allam AT, El-Dessouki AM, El-Shiekh RA, Abou-Hussein D, Mahmoud MA, Marcus WH, Ruby HA. A holistic guide to effective prevention and treatment for kidney stones: a systematic review exploring anti-urolithiasis approaches. Naunyn Schmiedebergs Arch Pharmacol. 2026;399 (3):3235-3284. doi:10.1007/s00210-025-04658-y.
13. Dawson CH, Tomson CR. Kidney stone disease: pathophysiology, investigation and medical treatment. Clin Med (Lond). 2012;12 (5):467-471. doi:10.7861/clinmedicine.12-5-467.
14. Shastri S, Patel J, Sambandam KK, Lederer ED. Kidney stone pathophysiology, evaluation and management: Core Curriculum 2023. Am J Kidney Dis. 2023;82 (5):617-634. doi:10.1053/j.ajkd.2023.03.017.
15. Skolarikos A, Geraghty R, Somani B, Tailly T, Jung H, Neisius A, et al. European Association of Urology Guidelines on the diagnosis and treatment of urolithiasis. Eur Urol. 2025;88 (1):64-75. doi:10.1016/j.eururo.2025.03.011.
16. Joshi A, Tallman JE, Calvert JK, Brewer T, Miller NL, Yang L, et al. Complementary and alternative medicine use in first-time and recurrent kidney stone formers. Urology. 2021;156:58-64. doi:10.1016/j.urology.2021.05.084.
17. Singh N, Agarwal S, Sarpal R. Prospective evaluation of extracorporeal shockwave lithotripsy in renal and upper ureteric stone treatment: clinical assessment and results. Cureus. 2024;16 (5):e61102. doi:10.7759/cureus.61102.
18. Jiang P, Xie L, Arada R, Patel RM, Landman J, Clayman RV. Qualitative review of clinical guidelines for medical and surgical management of urolithiasis: consensus and controversy 2020. J Urol. 2021;205 (4):999-1008. doi:10.1097/JU.0000000000001478.
19. Balawender K, Łuszczki E, Mazur A, Wyszyńska J. The multidisciplinary approach in the management of patients with kidney stone disease—a state-of-the-art review. Nutrients. 2024;16 (12):1932. doi:10.3390/nu16121932.
20. Özdemir M, Çığşar G, Bağcıoğlu M, Çiftçi H, Günal E. Comparison of the analgesic effects of intravenous dexketoprofen, ibuprofen and fentanyl in patients suffering from renal colic pain in the emergency department. Eurasian J Emerg Med. 2023;22 (1):18-23. doi:10.4274/eajem.galenos.2022.20633.
21. Spradling K, Ganesan C, Conti S. Medical treatment and prevention of urinary stone disease. Urol Clin North Am. 2022;49 (2):335-344. doi:10.1016/j.ucl.2021.12.007.
22. Siener R. Nutrition and kidney stone disease. Nutrients. 2021;13 (6):1917. doi:10.3390/nu13061917.
23. Liu M, Hou J, Xu F, Du H, Liu J, Li N. Minimally invasive nephrolithotomy versus retrograde intrarenal surgery in surgical management of lower calyceal stones: a systematic review with meta-analysis. Int J Surg. 2023;109 (5):1481-1488. doi:10.1097/JS9.0000000000000394.
24. Setthawong V, Srisubat A, Potisat S, Lojanapiwat B, Pattanittum P. Extracorporeal shock wave lithotripsy (ESWL) versus percutaneous nephrolithotomy (PCNL) or retrograde intrarenal surgery (RIRS) for kidney stones. Cochrane Database Syst Rev. 2023;8 (8):CD007044. doi:10.1002/14651858.CD007044.pub4.
25. Shafi H, Moazzami B, Pourghasem M, Kasaeian A. An overview of treatment options for urinary stones. Caspian J Intern Med. 2016;7 (1):1-6.

26. Rammah AM, Khaled F, Zamel S, Elkady A, Morsy S, Abdelwahab M. The efficacy of flexible ureteroscopy for large volume stones and hazards of ureteral access sheath usage: a prospective randomized study. Arab J Urol. 2025;23 (1):62-69. doi:10.1080/20905998.2024.2398378.
27. Cassell A 3rd, Jalloh M, Ndoye M, Mbodji M, Gaye O, Thiam NM, et al. Surgical management of urolithiasis of the upper tract: current trend of endourology in Africa. Res Rep Urol. 2020;12:225-238. doi:10.2147/RRU.S257669.
28. De Lorenzis E, Zanetti SP, Boeri L, Montanari E. Is there still a place for percutaneous nephrolithotomy in current times? J Clin Med. 2022;11 (17):5157. doi:10.3390/jcm11175157.
29. Paludo AO, Gorgen ARH, Araldi M, Tavares PM, Batezini NS, Rosito TE. Laparoscopic pielolitotomy: an option for the management of pelvic kidney stones. Int Braz J Urol. 2020;46 (3):489-490. doi:10.1590/S1677-5538.IBJU.2019.0148.
30. Bahmani M, Baharvand-Ahmadi B, Tajeddini P, Rafieian-Kopaei M, Naghdi N. Identification of medicinal plants for the treatment of kidney and urinary stones. J Renal Inj Prev. 2016;5 (3):129-133. doi:10.15171/jrip.2016.27.
31. Chobhe A, Sable S, Pawar PY, Gaikwad PM, Kakade. A comprehensive review on anti-urolithiatic agents: mechanisms, therapeutics, and future perspectives. Int J Pharm Sci. 2026;4 (4):3568-3582.
32. Coe FL, Evan A, Worcester E. Kidney stone disease. J Clin Invest. 2005;115 (10):2598-2608. doi:10.1172/JCI26662.
33. Khan SR. Reactive oxygen species, inflammation and calcium oxalate nephrolithiasis. Transl Androl Urol. 2014;3 (3):256-276. doi:10.3978/j.issn.2223-4683.2014.06.04.
34. Calixto JB, Santos ARS, Cechinel Filho V, Yunes RA. A review of the plants of the genus Phyllanthus: their chemistry, pharmacology, and therapeutic potential. Med Res Rev. 1998;18 (4):225-258.
35. Bagalkotkar G, Sagineedu SR, Saad MS, Stanslas J. Phytochemicals from Phyllanthus niruri Linn. and their pharmacological properties: a review. J Pharm Pharmacol. 2006;58 (12):1559-1570.
36. Murugaiyah V, Chan KL. Antihyperuricemic lignans from the leaves of Phyllanthus niruri. Planta Med. 2006;72 (14):1262-1267.
37. Barros ME, Schor N, Boim MA. Effects of an aqueous extract from Phyllanthus niruri on calcium oxalate crystallization in vitro. Urol Res. 2003;30 (6):374-379.
38. Campos AH, Schor N. Phyllanthus niruri inhibits calcium oxalate endocytosis by renal tubular cells: its role in urolithiasis. Nephron. 1999;81 (4):393-397.
39. Freitas AM, Schor N, Boim MA. The effect of Phyllanthus niruri on urinary inhibitors of calcium oxalate crystallization and other factors associated with renal stone formation. BJU Int. 2002;89 (9):829-834.
40. Nishiura JL, Campos AH, Boim MA, Heilberg IP, Schor N. Phyllanthus niruri normalizes elevated urinary calcium levels in calcium stone forming (CSF) patients. Urol Res. 2004;32 (5):362-366.
41. Micali S, Sighinolfi MC, Celia A, De Stefani S, Grande M, Cicero AF, et al. Can Phyllanthus niruri affect the efficacy of extracorporeal shock wave lithotripsy for renal stones? A randomized, prospective, long-term study. J Urol. 2006;176 (3):1020-1022.
42. Boim MA, Heilberg IP, Schor N. Phyllanthus niruri as a promising alternative treatment for nephrolithiasis. Int Braz J Urol. 2010;36 (6):657-664.
43. Ali BH, Blunden G. Pharmacological and toxicological properties of Nigella sativa. Phytother Res. 2003;17 (4):299-305.
44. Ahmad A, Husain A, Mujeeb M, Khan SA, Najmi AK, Siddique NA, et al. A review on therapeutic potential of Nigella sativa: a miracle herb. Asian Pac J Trop Biomed. 2013;3 (5):337-352.
45. Darakhshan S, Bidmeshki Pour A, Hosseinzadeh Colagar A, Sisakhtnezhad S. Thymoquinone and its therapeutic potentials. Pharmacol Res. 2015;95-96:138-158.

46. Hadjzadeh MA, Khoei A, Hadjzadeh Z, Parizady M. Ethanolic extract of Nigella sativa L. seeds on ethylene glycol-induced kidney calculi in rats. Urol J. 2007;4 (2):86-90.
47. Shakeri N, Mehrabi S, Paymard A. Comparison efficacy of oral Nigella sativa seeds and tamsulosin on pain relief and passage of 4 to 10 mm stones of kidney and ureter: a randomized clinical trial. J Nephropharmacol. 2022;11 (1). doi:10.34172/npj.2022.08.
48. Chhatre S, Nesari T, Somani G, Kanchan D, Sathaye S. Phytopharmacological overview of Tribulus terrestris. Pharmacogn Rev. 2014;8 (15):45-51.
49. Dinchev D, Janda B, Evstatieva L, Oleszek W, Aslani MR, Kostova I. Distribution of steroidal saponins in Tribulus terrestris from different geographical regions. Phytochemistry. 2008;69 (1):176-186.
50. Joshi VS, Parekh BB, Joshi MJ, Vaidya AB. Herbal extracts of Tribulus terrestris and Bergenia ligulata inhibit growth of calcium oxalate monohydrate crystals in vitro. J Cryst Growth. 2005;275 (1-2):e1403-e1408.
51. Aggarwal A, Tandon S, Singla SK, Tandon C. Diminution of oxalate-induced renal tubular epithelial cell injury and inhibition of calcium oxalate crystallization in vitro by aqueous extract of Tribulus terrestris. Int Braz J Urol. 2010;36 (4):480-489.
52. Anand R, Patnaik GK, Kulshreshtha DK, Dhawan BN. Activity of certain fractions of Tribulus terrestris fruits against experimentally induced urolithiasis in rats. Indian J Exp Biol. 1994;32 (8):548-552.
53. Sangeeta D, Sidhu H, Thind SK, Nath R. Effect of Tribulus terrestris on oxalate metabolism in rats. J Ethnopharmacol. 1994;44 (2):61-66.
54. Al-Ali M, Wahbi S, Twaij H, Al-Badr A. Tribulus terrestris: preliminary study of its diuretic and contractile effects and comparison with Zea mays. J Ethnopharmacol. 2003;85 (2-3):257-260.
55. Kamboj A, Saluja AK. Bryophyllum pinnatum (Lam.) Kurz.: phytochemical and pharmacological profile: a review. Pharmacogn Rev. 2009;3 (6):364-374.
56. Harlalka GV, Patil CR, Patil MR. Protective effect of Kalanchoe pinnata Pers. (Crassulaceae) on gentamicin-induced nephrotoxicity in rats. Indian J Pharmacol. 2007;39 (4):201-205.
57. Hasanudin K, Hashim P, Mustafa S. Corn silk (Stigma maydis) in healthcare: a phytochemical and pharmacological review. Molecules. 2012;17 (8):9697-9715.
58. Maksimović Z, Malenčić D, Kovačević N. Polyphenol contents and antioxidant activity of Maydis stigma extracts. Bioresour Technol. 2005;96 (8):873-877.
59. Velazquez DVO, Xavier HS, Batista JEM, de Castro-Chaves C. Zea mays L. extracts modify glomerular function and potassium urinary excretion in conscious rats. Phytomedicine. 2005;12 (5):363-369.
60. Grases F, March JG, Ramis M, Costa-Bauzá A. The influence of Zea mays on urinary risk factors for kidney stones in rats. Phytother Res. 1993;7 (2):146-149.
61. Soundararajan P, Mahesh R, Ramesh T, Begum VH. Effect of Aerva lanata on calcium oxalate urolithiasis in rats. Indian J Exp Biol. 2006;44 (12):981-986.
62. Vetrichelvan T, Jegadeesan M. Anti-diabetic activity of alcoholic extract of Aerva lanata (L.) Juss. ex Schultes in rats. J Ethnopharmacol. 2002;80 (1):103-107.
63. Shirwaikar A, Issac D, Malini S. Effect of Aerva lanata on cisplatin and gentamicin models of acute renal failure. J Ethnopharmacol. 2004;90 (1):81-86.
64. Alok S, Jain SK, Verma A, Kumar M, Mahor A, Sabharwal M. Plant profile, phytochemistry and pharmacology of Asparagus racemosus (Shatavari): a review. Asian Pac J Trop Dis. 2013;3 (3):242-251.
65. Wiboonpun N, Phuwapraisirisan P, Tip-pyang S. Identification of antioxidant compound from Asparagus racemosus. Phytother Res. 2004;18 (9):771-773.

66. Christina AJM, Ashok K, Packialakshmi M, Tobin GC, Preethi J, Murugesh N. Antilithiatic effect of Asparagus racemosus Willd on ethylene glycol-induced lithiasis in male albino Wistar rats. Methods Find Exp Clin Pharmacol. 2005;27 (9):633-638.
67. Khalil N, Bishr M, Desouky S, Salama O. Ammi visnaga L., a potential medicinal plant: a review. Molecules. 2020;25 (2):301.
68. Duarte J, Pérez-Vizcaíno F, Torres AI, Zarzuelo A, Tamargo J. Vasodilator effects of visnagin in isolated rat vascular smooth muscle. Eur J Pharmacol. 1995;286 (2):115-122.
69. Khan ZA, Assiri AM, Al-Afghani HM, Maghrabi TM. Inhibition of oxalate nephrolithiasis with Ammi visnaga (Al-Khillah). Int Urol Nephrol. 2001;33 (4):605-608.
70. Vanachayangkul P, Byer K, Khan S, Butterweck V. An aqueous extract of Ammi visnaga fruits and its constituents khellin and visnagin prevent cell damage caused by oxalate in renal epithelial cells. Phytomedicine. 2010;17 (8-9):653-658.
71. Gürocak S, Küpeli B. Consumption of historical and current phytotherapeutic agents for urolithiasis: a critical review. J Urol. 2006;176 (2):450-455. doi:10.1016/j.juro.2006.03.034.
72. Kieley S, Dwivedi R, Monga M. Ayurvedic medicine and renal calculi. J Endourol. 2008;22 (8):1613-1616. doi:10.1089/end.2008.0020.
73. Patankar SB, Mujumdar AM, Bernard F, Supriya P. Safety and efficacy of an herbal formulation in patients with renal calculi: a 28 week, randomized, double-blind, placebo-controlled, parallel group study. J Ayurveda Integr Med. 2020;11 (1):62-67. doi:10.1016/j.jaim.2018.12.006.
74. Ansari R, Karimzade I, Nimrouzi M, Ezatzadegan S, Hosseini MM, Zarshenas MM. Safety and efficacy of a polyherbal formulation from traditional Persian medicine in patients with calcium kidney stones: a randomized, double-blinded clinical trial. J Res Med Sci. 2024;29:12. doi:10.4103/jrms.jrms_670_22.
75. Ahmad W, Khan MA, Ashraf K, Ahmad A, Daud Ali M, Ansari MN, et al. Pharmacological evaluation of Safoof-e-Pathar Phori—a polyherbal Unani formulation for urolithiasis. Front Pharmacol. 2021;12:597990. doi:10.3389/fphar.2021.597990.
76. Goyal PK, Verma SK, Sharma AK. Antiurolithiatic potential of Neeri against calcium-oxalate stones by crystallization inhibition, free radicals scavenging, and NRK-52E cell protection from oxalate injury. Pharmacogn Mag. 2017;13 (Suppl 3):S549-S554. doi:10.4103/pm.pm_551_16.
77. Shirfule AL, Racharla V, Qadri SSYH, Khandare AL. Exploring antiurolithic effects of Gokshuradi polyherbal Ayurvedic formulation in ethylene-glycol-induced urolithic rats. Evid Based Complement Alternat Med. 2013;2013:763720. doi:10.1155/2013/763720.
78. Mote D, Chaudhari N, Maurya MN. A prospective, open-label non-randomized clinical trial to evaluate the safety and efficacy of Calcury tablets in treatment of renal calculi. J Ayurveda Integr Med Sci. 2025;10 (7):40-48. doi:10.21760/jaims.10.7.6.
79. Erickson SB, Vrtiska TJ, Lieske JC. Effect of Cystone® on urinary composition and stone formation over a one year period. Phytomedicine. 2011;18 (10):863-867. doi:10.1016/j.phymed.2011.01.018.
80. Zhai W, Zheng J, Yao X, et al. Catechin prevents the calcium oxalate monohydrate-induced renal calcium crystallization in NRK52E cells and the ethylene glycol-induced renal stone formation in rat. BMC Complement Altern Med. 2013;13:228. doi:10.1186/1472-6882-13-228.
81. Nirumand MC, Hajialyani M, Rahimi R, Farzaei MH, Zingue S, Nabavi SM, et al. Dietary plants for the prevention and management of kidney stones: preclinical and clinical evidence and molecular mechanisms. Int J Mol Sci. 2018;19 (3):765. doi:10.3390/ijms19030765.
82. Noorafshan A, Karbalay-Doust S, Karimi F. Diosmin reduces calcium oxalate deposition and tissue degeneration in nephrolithiasis in rats: a stereological study. Korean J Urol. 2013;54 (4):252. doi:10.4111/kju.2013.54.4.252.

83. Guzel A, Yunusoglu S, Calapoglu M, et al. Protective effects of quercetin on oxidative stress-induced tubular epithelial damage in the experimental rat hyperoxaluria model. Medicina (Kaunas). 2021;57:566. doi:10.3390/medicina57060566.

84. Allam EAH, Sabra MS. Plant-based therapies for urolithiasis: a systematic review of clinical and preclinical studies. Int Urol Nephrol. 2024;56 (12):3687-3718. doi:10.1007/s11255-024-04148-9.

85. Patle A, Hatware KV, Patil K, Sharma S, Gupta G. Role of herbal medicine in the management of urolithiasis—a review for future perspectives. J Environ Pathol Toxicol Oncol. 2019;38 (2):97-118. doi:10.1615/JEnvironPatholToxicolOncol.2019029075.

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