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Review Article

A comprehensive review of renal calculi (Ḥaṣā al-Kulya): Unani and modern understanding

Ayza-Ur-Rahman[1], Mohammad Shadab[1], Irfan Ahmad[1], Arshad Kafi[1], Junaid M. Hashmi[2]*

[1]Department of Mahiyat-ul-Amraaz, State Unani Medical College, Prayagraj, Uttar Pradesh, India
[2]Department of Kulliyat, State Unani Medical College, Prayagraj, Uttar Pradesh, India

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***Correspondence:**
Dr. Junaid M. Hashmi,
E-mail: drjunaidmalikhashmi@gmail.com

ABSTRACT

Nephrolithiasis, commonly termed urolithiasis, is characterized by the development of calculi anywhere within the urinary tract. The occurrence of this condition varies across different regions and is influenced by multiple factors, including climate, age, gender, dietary patterns, and genetic predisposition. The highest incidence is typically reported during the second and third decades of life, with males being affected more frequently than females in an approximate ratio of 2:1. Stone formation results from supersaturation of urinary solutes such as calcium, oxalate, and uric acid, along with low levels of citrate. This physicochemical imbalance promotes crystal aggregation and subsequent calculus formation. From the perspective of Unani medicine, calculi (*Ḥaṣā*) are believed to develop due to the retention and accumulation of dense, morbid matter *(Mādda Ghalīẓ)* within the organs, coupled with impaired expulsion. Progressive deposition of this material eventually leads to stone formation. The concept of *Hararat* (heat) contributes to the evaporation of *Ratubat* (moisture), resulting in desiccation (*Tajfīf*), condensation, and hardening of the retained matter into calculi.

Keywords: Nephrolithiasis, Urolithiasis, Calcium oxalate, Uric acid, Hararat, Ratubat, *Ḥaṣā al-Kulya*

INTRODUCTION

Nephrolithiasis or urolithiasis, often called kidney stones or renal calculi, are solid masses made of minerals that form in the kidneys. These masses develop when minerals from urine settle and build up over time.[1] Urinary calculi are commonly diagnosed as a consequence of an episode of renal-ureteral colic or gross haematuria.[2] The problem of stone formation is considered a major medical challenge due to its multifactorial etiology and high rate of recurrence, arising from a critical imbalance between urinary promoters and inhibitors.[3] Without timely and proper intervention, kidney stones can cause severe complications, including ureteral obstruction, persistent haematuria, recurrent urinary tract infections, vomiting, or excruciating painful urination, ultimately culminating in permanent functional damage to the renal parenchyma.[4]

EPIDEMIOLOGY

Urinary tract stone disease affects about 8–13% of people in industrialized countries, with significantly lower prevalence rates reported in regions such as Japan and Greenland.[2] Its incidence varies worldwide due to factors like diet, climate, hydration, physical activity, obesity, and socioeconomic conditions.[2] In Europe, prevalence ranges from 9–15%, rising to 23% in the USA and peaking at 27% in Saudi Arabia[2]. In Italy, prevalence is about 19.2%, with an estimated social cost of EUR 200 million per year.[2] The global rate of occurrence is significantly higher in men than women, which is largely attributed to the lithogenic promoting capacity of testosterone and the corresponding inhibiting capacity of oestrogen.[3]

Stone disease is considered a “disease in evolution” because of changing epidemiology, improved diagnostic

techniques, advances in treatment, and better identification of at-risk populations.[2] Most stones are composed of calcium oxalate (60–78%), followed by uric acid, struvite, and cystine.[2] Over recent years, calcium oxalate stones have increased, while infection-related and uric acid stones have declined, likely due to improved living conditions, antibiotic use, and dietary management.[2] There has been a growing prevalence of small ureteral stones (<1 cm), largely due to the widespread clinical use of advanced imaging techniques and extracorporeal shock wave lithotripsy (ESWL).[2] This shift has changed clinical presentation and management, favouring minimally invasive treatments such as ureteroscopy, while ESWL remains suitable for medium-sized stones and percutaneous procedures for larger calculi.[2]

Stone formation results from a complex interaction of genetic, anatomical, metabolic, dietary, and environmental factors. Risk increases with age, male sex, family history, obesity, and comorbidities such as hypertension and hyperuricemia.[2] Climate also plays a role, with higher recurrence in warmer seasons due to dehydration and concentrated urine.[2] Dietary habits particularly high intake of animal protein, calories, oxalate, and insufficient fluid intake are strongly associated with increased stone risk, while adequate hydration remains the most effective preventive measure.[2]

ETIOLOGY

The multifactorial etiology of urolithiasis can be systematically classified into the following primary determinants.[1]

Metabolic factors

Abnormal urine composition leading to crystal supersaturation and subsequent stone formation.

Genetic predisposition

Inherited metabolic disorders, such as cystinuria and primary hyperoxaluria, which alter the excretion of stone-forming substances.

Environmental factors

Arid climates inducing dehydration and concentrated urine; regional water compositions and chemical exposures also act as critical risk factors.

Dietary factors

Excessive intake of oxalates, purines, and sodium, paired with inadequate fluid consumption.

Anatomical factors

Structural abnormalities of the urinary tract causing urinary stasis and obstructing normal flow.

Medical conditions

Underlying systemic diseases, including recurrent urinary tract infections (UTIs), inflammatory bowel disease (IBD), and primary hyperparathyroidism.

PATHOGENESIS

The physicochemical mechanism of urolithiasis progresses through a cascade of distinct phases, triggered primarily by urinary supersaturation (concentration/ solubility ratio >1).[5]

Determinants of supersaturation

Decreased urine volume directly elevates solute concentration.

Furthermore, urinary pH shifts influence crystallization pathways alkaline urine favors calcium phosphate and struvite precipitation, whereas acidic urine promotes uric acid and cystine crystallization.[5]

Crystallization disbalance

A functional deficit in crystallization inhibitors (such as citrate, magnesium, and specialized proteins) combined with an increase in crystal promoters.[5]

Crystal precipitation and nucleation

The initial crystallization phase, often occurring on injured epithelial cell surfaces or localized calcific lesions known as Randall's plaques (calcium phosphate).[5]

Crystal growth and aggregation

Rapid enlargement and binding of calcium oxalate or calcium phosphate crystals.[5]

Nephrolith formation

The consolidation of aggregated crystals into a clinically significant, mature kidney stone.[5]

CLINICAL MANIFESTATIONS

The clinical presentation varies according to the size and anatomical position of the stone within the urinary tract.[1] Early-stage calculi may remain clinically silent.

As the stone enlarges or migrates, patients may experience haematuria, intense colicky flank pain, urinary tract infections, obstruction, and hydronephrosis. Acute renal colic is often associated with nausea and vomiting.[5]

Common clinical symptoms include: severe, intermittent colicky pain, nausea and vomiting, gross or microscopic haematuria, pyuria, dysuria, and oliguria.

CHEMICAL CLASSIFICATION OF KIDNEY STONES

The chemical composition of urine plays a key role in determining the type of kidney stones formed 6. Based on their composition, urinary stones are broadly classified into five major types.

Calcium stones

Accounting for approximately 70–80% of all cases, these are the most prevalent urinary calculi.[6] They manifest as calcium oxalate—occurring either as the highly stable calcium oxalate monohydrate (COM) or dihydrate (COD)—calcium phosphate, or calcium carbonate.[6] Metabolic derangements like hypercalciuria, hyperoxaluria, and hyperuricosuria directly drive their formation.[6]

Uric acid (urate) stones

Comprising 5–10% of diagnosed calculi, these are strongly correlated with a high-purine diet (such as animal proteins), low urinary volume, hyperuricosuria, and a persistently acidic urinary pH.[6]

Magnesium ammonium phosphate (struvite) stones

Also designated as infection or triple phosphate stones, these account for 12–15% of cases.[6] They are etiologically linked to upper urinary tract infections caused by urease-producing microorganisms, which split urea into ammonia and carbon dioxide.[6] The resulting alkaline urinary environment dramatically reduces phosphate solubility, causing rapid stone growth. These are statistically more common in females.[6]

Cystine stones

A rare category comprising less than 3% of cases, arising from cystinuria an autosomal recessive genetic disorder characterized by defective renal tubular reabsorption of dibasic amino acids, leading to poor cystine solubility in urine.[6]

Drug-induced stones

Representing approximately 1% of cases, these form when medications (such as triamterene, atazanavir, and sulfonamides) or their metabolites directly crystallize in the collecting system or act as a lithogenic nidus.[6]

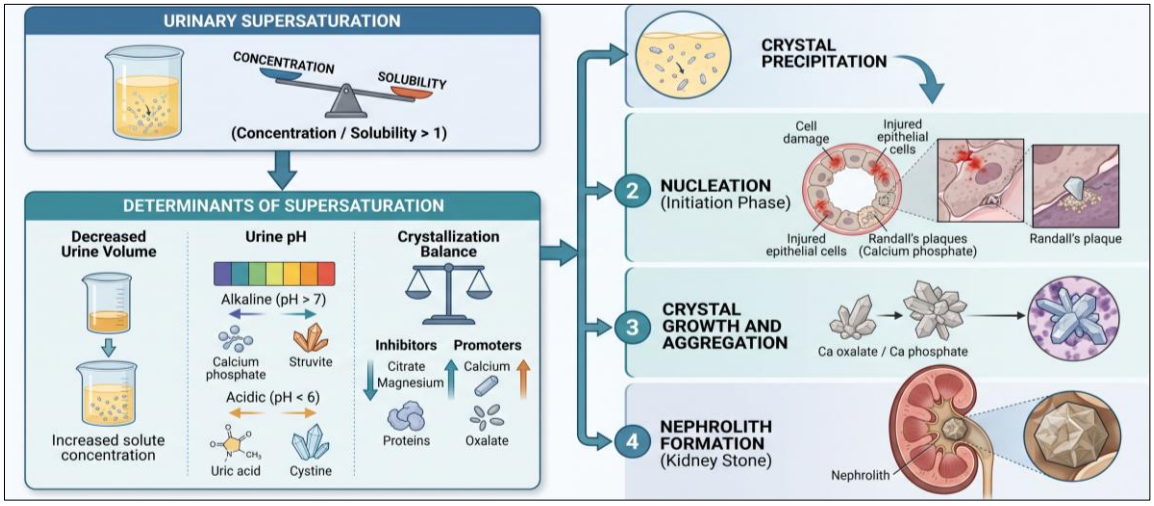

Figure 1: Mechanism of nephrolith (*Ḥaṣā al Kulya*) formation showing urinary supersaturation, nucleation, crystal growth, and nephrolith development.

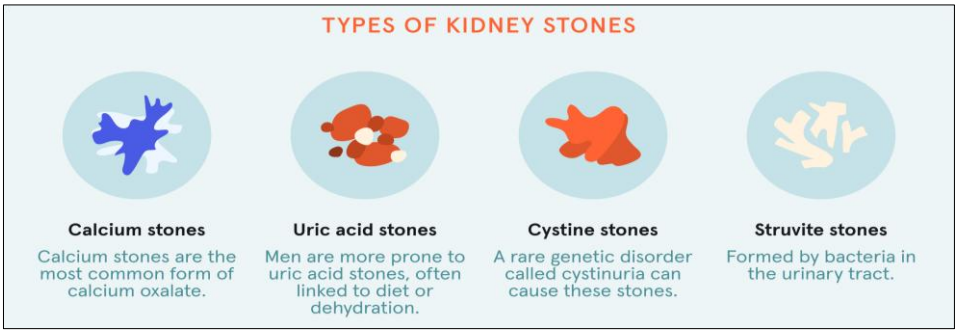

Figure 2: Types of nephroliths (*Ḥaṣā al Kulya*) showing calcium, uric acid, cystine, and struvite stones with their respective causes.

DIAGNOSTIC MODALITIES

Computed tomography

Non-contrast computed tomography (NCCT) remains the gold standard diagnostic modality for urolithiasis, exhibiting superior sensitivity and specificity regardless of calculus dimensions, spatial location, or chemical composition.[7,8] It accurately delineates secondary signs of urinary obstruction (e.g., hydronephrosis, perinephric stranding) and differentiates ureteric stones from pelvic phleboliths.[7,8] Advanced dual-energy CT (DECT) scans allow for the non-invasive determination of stone composition, predicting the efficacy of shockwave therapies.[7,8]

Ultrasonography

Transabdominal ultrasound is an invaluable, radiation-free first-line screening tool, particularly indicated in paediatric and pregnant patient populations.[9] While highly effective at detecting hydronephrosis and large proximal or vesical calculi, its diagnostic sensitivity is limited in evaluating mid-ureteral stones compared to CT imaging.[9]

Magnetic resonance urography

Magnetic resonance urography (MRU) represents a sophisticated alternative to CT imaging when ionizing radiation must be strictly avoided (e.g., during pregnancy or childhood) or when iodinated contrast is contraindicated due to renal impairment.[10] It provides excellent anatomic resolution of the collecting system and nearby soft tissues, successfully differentiating pathological obstructive hydronephrosis from physiological gestational uropathy.[10]

CONVENTIONAL MANAGEMENT AND PREVENTION

The modern management of urolithiasis necessitates a multimodal therapeutic strategy encompassing dietary modification, botanical pharmacotherapy, medical therapy, and advanced surgical interventions.[11]

Dietary risk factors

High intake of dietary oxalates (found in spinach, rhubarb, legumes, and wheat bran), excessive sodium consumption, and high-purine alcoholic beverages (such as beer and wine) significantly elevate the metabolic risk of calculus formation.[11]

Dietary prevention

Increased consumption of citrus fruits rich in citrate—a potent natural crystallization inhibitor along with optimal hydration, is highly effective.[11] Traditional intake of horse gram, radish, and beetroot is also widely utilized.[11] Long-term recurrence is minimized by adopting a diet low in animal proteins and sodium, supplemented with adequate dietary fiber, magnesium, and vitamins B6 and E.[11]

Medical therapy

Pharmacological management focuses on preventing stone growth, mitigating symptoms, and chemo-prophylaxis.[11] Standard options include thiazide diuretics (for hypercalciuria), potassium citrate (for urinary alkalinization), allopurinol (for hyperuricosuria), sodium cellulose phosphate, penicillamine, bisphosphonates, and specialized probiotics like *Oxalobacter formigenes*.[11]

Herbal therapy

Over-the-counter phytotherapeutic preparations (formulated as syrups, tablets, and decoctions) are widely used. Mechanistically, these agents induce diuresis to flush out small calculi, exert anti-inflammatory and analgesic effects to relieve colic, and display lithotripsic properties that help break down the crystalline lattice.[7] Well-established proprietary formulations include Cystone, Calcuri, Chandraprabha Vati, and Ber Patthar Bhasma.[11]

Surgical therapy

Interventions are mandatory for large, symptomatic, or highly obstructive stones.

Extracorporeal shock wave lithotripsy

A non-invasive modality preferred for small-to-medium renal stones (<20 mm), achieving stone-free rates of 70–80%.[12,13]

Ureterorenoscopy

An endoscopic approach offering excellent stone-free rates for ureteric and lower pole calculi, characterized by rapid recovery.[13]

Percutaneous nephrolithotomy

The definitive treatment of choice for large renal calculi (>2 cm) and complex staghorn stones, yielding stone-free rates between 85–93%.[9]

UNANI PERSPECTIVE AND CLASSICAL CONCEPTS

The Unani system of medicine, originating in ancient Greece and formalized by Hippocrates (460–377 BC), is underpinned by the Humoral Theory (*Akhlāt*).[14] Human health is governed by the equilibrium of four primary humors: *Dam* (blood), *Balgham* (phlegm), *Safrā* (yellow bile), and *Saudā* (black bile). Any qualitative or quantitative disturbance in this humoral homeostasis leads to a disease state.[15] This balance fundamentally dictates an individual's unique temperament (*Mizāj*), characterized by

the core cosmic qualities of heat (*ḥarārat*), cold (*burūdat*), dryness (*yubūsat*), and moisture (*ruṭūbat*).[16]

The academic advancement of urology was profoundly shaped by medieval Islamic physicians, notably al-Rāzī (Rhazes), al-Majūsī (Haly Abbas), and Ibn Sīnā (Avicenna).[17] Ibn Sīnā's monumental work, *Al-Qanun fi al-Tibb* (The Canon of Medicine), served as the supreme European medical textbook for centuries, providing advanced, systematic data on the diagnosis and treatment of renal and vesical disorders.[18]

PATHOPHYSIOLOGY ACCORDING TO UNANI MEDICINE

Mechanism of stone formation

In *Kāmil al-Ṣināʿah al-Ṭibbyah*, Ibn Abbās al-Majūsī states that urolithiasis is a product of intense pathological heat (*shadīd ḥarārat*) acting upon a thick, viscous humor (*khilṭ ghalīẓ*).[19,20] This interaction causes the evaporation of its fluid component (*ruṭūbat*), leading to the desiccation (*Tajfīf*), precipitation, and hardening of a gritty residue into a calculus . While mildly thick humors result in transient, fine gravel, highly viscous material aggregates into large, fixed stones within the urinary tract, which undergo further consolidation over time.[19,20] Al-Majūsī also observed that bladder stones (*ḥiṣāt al-mathānah*) occur frequently in adolescents due to their naturally moist temperament (*raṭab mizāj*), lifestyle, and dietary habits, whereas in adults, it is driven by heavy, viscid foods (*ghalīẓ al-kaimūs ghidhā*).[21]

Galen originally postulated that stones develop either due to the condensation of trapped gas (*rīḥ*) within the renal spaces or the calcification of inflammatory pus from renal ulcerations.[22] Ibn Sīnā later synthesized these concepts, attributing stone formation to the prolonged retention of morbid matter (*mādda ghalīẓ*) resulting from defective primary digestion (*ḍuʿf quwwat-i hāḍimah*).[18] This un-concocted, thick humor accumulates in the collecting system and, due to weakened renal expulsive force (*ḍuʿf quwwat-i dāfiʿya*), abnormal renal temperament (*sūʾ-i mizāj kulliyā*), or local structural lesions, undergoes calcification under the influence of metabolic heat.[22] Al-Rāzī similarly identified a systemic failure to eliminate viscid matter (*lesdār mawād*) as the root cause, leading to gradual mineral deposition and desiccation by innate heat.[23]

Age and gender distribution

Classical Unani literature documents that renal calculi are more prevalent during middle age, whereas bladder calculi are frequently encountered in childhood and adolescence.[21] In middle age, the natural decline in innate heat (*ḥarārat-i-gharīziyā*), accumulation of viscid phlegm (*khilṭ-i-balgham*), and dominance of coldness (*burūdat*) structurally constrict the narrow urinary passages, trapping larger particles within the kidney where they mature into renal stones.[24]

Conversely, in adolescents, the renal passages are relatively wider, allowing micro-calculi to flow freely into the bladder.[21] However, the narrow adolescent urethral outlet acts as a mechanical barrier, leading to vesical retention. The high intrinsic heat of the young bladder subsequently hardens this material into a vesical stone.[24] Young adult females are considered less prone to stone disease due to an anatomically wider, shorter urethra that facilitates the easy evacuation of thick urinary sediments.[21]

In *Dhakhīra-i-Khwārazm Shāhī*, Ismāʿīl Jurjānī notes that thick, viscid urine and excessive urinary sediments from difficult-to-digest, lithogenic foods such as beef, camel meat, fried eggs, unrefined wheat products, milk, rice, paneer, and citrus fruits significantly increase the incidence of stone formation.[25] Ḥakīm Aʿẓam Khān further observed that renal calculi are more common in obese and elderly populations due to an age-related decline in digestive strength (*ḍuʿf-i quwwat-i hāḍimah*).[22] Ibn Sīnā also noted the morphological differences: kidney stones are typically small, soft, and reddish, while bladder stones present as larger, dense, and sandy-white or black masses.[24] Classical physicians also noted that lithogenic matter is systemic and can manifest as calculi in the joints, liver, intestines, or colon.[21]

DIAGNOSIS AND SYMPTOMATOLOGY IN UNANI MEDICINE

The classical Unani diagnosis relies on a comprehensive evaluation of the pulse (*Nabḍ*), stool (*Barāz*), and critically, the urine (*Baul*).[26] Traditional qualitative urinalysis involved collecting urine overnight to examine the sedimentation layer; the presence of distinct red or yellowish crystalline deposits indicated active urolithiasis.[26]

Key diagnostic indicators described in classical texts include the following.

A sudden shift in urine character from thick and turbid to clear and watery, signalling the trapping and accumulation of morbid matter (*Mādda*) within the renal pelvis.

The passage of dark, black coloured urine accompanied by severe abdominal colic and lumbar pain.[25]

Pain radiating from the lumbar region toward the pubic area indicates active ureteral migration, while a sudden relief in flank pain denotes that the calculus has reached the urinary bladder.[25]

Bladder stones characteristically present with acute dysuria, incomplete emptying, urethral pruritus, and the appearance of a whitish, muddy urinary sediment.[27]

Ibn Sīnā highlighted that renal calculi are distinguished by a profound feeling of heaviness in the lumbar region, exacerbated when the colon is full, paired with yellowish-red urine.[28]

Al-Rāzī noted that bilateral renal calculi present with synchronized loin pain, painful micturition, nausea, and severe constipation.[23]

PRINCIPLES OF TREATMENT (UṢŪL-I-ʿILĀJ)

Acute and preventive pharmacotherapy

The classical management of urolithiasis (*Ḥaṣā al-Kulya wa Mathānah*) is strictly divided into two distinct phases: acute management of renal colic and long-term curative/preventive therapy initiated post-attack.

During acute renal colic, treatment aims to alleviate pain and induce muscle relaxation without dampening the body's innate evacuative power (*Quwwat-i-Dāfiʿah*). External non-invasive therapies are prioritized, including the following.

Nuṭūl (medicated irrigation)

Continuous irrigation using decoctions of *Butea monosperma*, *Adiantum capillus-veneris*, and the diuretic seeds of *Cucumis sativus* and *Cucumis melo*.

Ḍimād (medicated paste) and Qairūṭī (poultice)

Applied locally using *Pisum sativum* flour and soothing oils.

Ābzan (sitz bath)

Hydrotherapy with hydro-decoctions of *Tribulus terrestris*, *Matricaria chamomilla*, *Melilotus officinalis*, and *Althaea officinalis* to reduce ureteral spasms.

Once the acute spasm resolves, therapy advances to oral lithotriptic (*Mufattit-i-Ḥaṣā*) and purgative (*Mushil*) administrations to fragment and expel the stone.[22,23] Long-term therapy focuses on eliminating the source of *Mādda Ghalīẓah* (morbid matter) through emetics, customized purgatives, and gastric tonics (*Muqawwi Miʿdah*) combined with strict avoidance of cold water during meals, fatty foods, and lithogenic diets.[22,23]

The temperamental dynamics of lithotriptics

In *Kitāb al-Kulliyāt*, Ibn Rushd notes a profound pharmacological principle: lithotripsic (*Mufattit*) drugs must possess only a mild, balanced degree of heat (*ḥarārat*).[29] He argues that excessive, abnormal heat (*ḥarārat-i-gharībah*) actually worsens desiccation and hardens the stone matrix. Therefore, morbid matter dominated by heat and dryness is optimally corrected using cooling (*burūdat*) and moisturizing (*ruṭūbat*) dynamics, allowing the body's innate heat (*ḥarārat-i-gharīziyā*) to naturally dissolve the stone. He cited *Halyun*, *Chana*, and *Bādām* as primary examples.[29]

Ibn Rushd further explained that these drugs must possess a bitter (*talkh*) taste and stone-fragmenting (*taqṭīʿ*) properties.[29] Notably, *Ḥajr al-Yahūd* (Jew's stone) was identified as highly specific for renal stones, while compound formulations like *Mājūn Ḥajr al-Yahūd* and *Mājūn Aqrab* are premier compound lithotriptics. Mildly warm diuretic agents (*Mudirr-i-Bāwl*) such as *Karafs* (*Apium graveolens*), *Bādiyān* (*Foeniculum vulgare*), and *Dūqū* (*Peucedanum grande*) are concurrently administered to maximize urinary wash-out.[29-31]

Conservative and surgical milestones

As documented in the *Hidāyat of Al-Akawaynī*, initial conservative protocols combined warm balneotherapy with lumbar oil massages and rigorous physical exercises (jumping, horseback riding) to physically dislodge the stone.[17] Stepwise pharmacotherapy utilized mild lithotriptics up to potent agents like Cantharides, which was strictly controlled due to its risk of causing bladder ulceration. To safeguard the tract, mucosal dilating herbs like marshmallow seeds were administered concurrently.[17]

When conservative measures failed, surgical lithotomy was indicated.[17] Al-Masīḥī advocated precise surgical incision for refractory urinary obstructions.[32] While Abū al-Qāsim al-Zahrāwī designed highly specialized surgical instruments for urethral stone extraction and vaginal lithotomy.[33]

Ibn Sīnā described pioneering localized urethral drug instillations, and Ibn Zuhr documented the use of a diamond-tipped lithotrite, representing a monumental historical milestone in minimally invasive endourology.[33]

OPERATIVE PROTOCOL OF SURGICAL LITHOTOMY

As detailed by Al-Masīḥī in *Kitāb Al-Umda fi'l Jarāhat*, the historical surgical protocol was highly systematic.

Pre-operative preparation

An enema (*ḥuqnā*) was administered to completely evacuate the bowel. The patient performed structured jumping motions to encourage the calculus to descend into the bladder neck.

Patient positioning and localization

The patient was secured with hands bound to the thighs to optimize perineal exposure. A digital rectal examination lubricated with Roghan Banafsha (*Viola odorata* oil) was performed to manually guide the stone toward the urethral outlet.[32]

Incision and extraction

The surgeon made a precise, oblique perineal incision slightly to the left of the midline. Superficial stones were removed manually, while deep stones were extracted using blunt forceps. Multiple calculi were extracted individually, and massive stones were carefully crushed intra-operatively to prevent wide tissue laceration.[32]

Hemostasis and wound management

Hemostasis was achieved by applying powdered alum (*Phitakrī*). The surgical wound was dressed with a specialized cicatrizing powder composed of *Kundur* (*Boswellia serrata*), *Damm al-Akhawain* (*Dracaena cinnabari*), and *Sibr* (*Aloe barbadensis*). Post-operatively, the thighs were kept bound together for three days, and the wound was maintained with *Roghan Gul* and *Marham Aswad*.[32]

CONCLUSION

While modern medicine has made remarkable technological advances in diagnostic imaging and the surgical removal of urolithiasis, preventing long-term metabolic recurrence remains a major medical challenge. Unani medicine approaches stone disease from a holistic, systemic perspective by addressing primary humoral imbalances, digestive errors, dietary habits, and impaired elimination of morbid matter. Its therapeutic principles successfully combine targeted lifestyle modifications with mild, non-toxic lithotripsic and diuretic remedies to dissolve existing stones and effectively prevent recurrence. Integrating the time-tested concepts of Unani medicine with modern scientific research offers a promising pathway toward developing comprehensive, evidence-based strategies for the long-term management of urolithiasis.

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REFERENCES

1. Yamgar PV, Nimbalkar VV, Pagar HJ. Urolithiasis: Mechanism of stone formation, diagnostic modalities and treatment protocols. IP Int J Compr Adv Pharmacol. 2024;9:227-35.
2. Shavkatovich BB. Urolithiasis: Epidemiology and Risk Factors. Ethiop Int J Multidisciplin Res. 2026;13(03):671-5.
3. Vijaya T, Sathish Kumar M, Ramarao NV, Naredra Babu A, Ramarao N. Urolithiasis and Its Causes-Short Review. J Phytopharm. 2013;2.
4. Wang Z, Zhang Y, Zhang J, Deng Q, Liang H. Recent advances on the mechanisms of kidney stone formation (Review). Int J Mol Med. 2021;48:4982.
5. Shastri S, Patel J, Sambandam KK, Lederer ED. Kidney Stone Pathophysiology, Evaluation and Management: Core Curriculum 2023. Am J Kidney Dis. 2023;82:617-34.
6. Sangolli A, Ghagane SC, Nerli RB. Role of Dietary Supplements in Prevention of Renal Stones: An Update. In. IntechOpen. 2021.
7. Shokeir AA. Renal Colic: New Concepts Related to Pathophysiology, Diagnosis and Treatment. Curr Opin Urol. 2002;12:263-9.
8. Andrabi Y, Patino M, Das CJ, Eisner B, Sahani DV, Kambadakone A. Advances in CT imaging for urolithiasis. Indian J Urol. 2015;31:185-93.
9. Masch WR, Cronin KC, Sahani DV, Kambadakone A. Imaging in Urolithiasis. Radiol Clin North Am. 2017;55:209-24.
10. Regan F, Kuszyk B, Bohlman ME, Jackman S. Acute ureteric calculus obstruction: Unenhanced spiral CT versus HASTE MR urography and abdominal radiograph. Br J Radiol. 2005;78:506-11.
11. Sah AN. Urolithiasis: An Update on Diagnostic Modalities and Treatment Protocols. Indian J Pharm Sci. 2017;79(2):164-74.
12. Petrides N, Ismail S, Anjum F, Sriprasad S. How to maximize the efficacy of shockwave lithotripsy. Turk J Urol. 2020;46:S19-26.
13. van Besien J, Uvin P, Hermie I, Tailly T, Merckx L. Ultrasonography Is Not Inferior to Fluoroscopy to Guide Extracorporeal Shock Waves during Treatment of Renal and Upper Ureteric Calculi: A Randomized Prospective Study. BioMed Res Int. 2017;2017:7802672.
14. Israili AH. Humoral theory of Unani Tibb. Indian J Hist Sci. 1981;16:95-9.
15. Hussain I, Baig MF, Hashmi JM, Zulkifle Mohd. Role of Temperament in Habitat Selection: Insights from Unani Medicine. Tradit Med Mod Med. 2025;8:179-85.
16. Kabiruddin M. Ifada-i-Kabir Mufassal. 1st edition. New Delhi, India: Central Council for Research in Unani Medicine (CCRUM); 2001:90.
17. Ardalan MR, Shoja MM, Tubbs SR, Eknoyan G. Diseases of the kidney in medieval Persia: The Hidayat of Al-Akawayni. Nephrol Dial Transplant. 2007;22:3413-21.
18. Madineh SMA. Avicenna's Canon of Medicine and Modern Urology. Part I: Bladder and its diseases. Urol J. 2008;5:284-93.
19. Majoosi AIA, Qantoori GH. Kamil us Sana'a. Urdu translation. Lucknow, India: Matba Munshi Naval Kishore; 2010:53.
20. Al-Din SN, Ibn 'Iwad KN, Kabiruddin. Sharh al-Asbab wa al-'Alamat. New Delhi, India: Idara Kitab-ul-Shifa; 2008:419.
21. Al-Majusi IA. Tarjuma-i Kamilus Sana. Volume 1. 1889:525.
22. Kabiruddin HM. Al-Iksir. Volume 2. Delhi: Ejaz Publication House; 2003:1201.
23. Razi Z. Al-Hawi. Volume 10. New Delhi: Central Council for Research in Unani Medicine; 2002:376.
24. Ibn-e-Sina. Al-Qānūn fi'l Ṭibb. 1st edition. Delhi, India: Idara Kitab-ul-Shifa; 2014:174.

25. Jurjani I. Tarjuma-i Zakhira-i Khwarzam Shahi. Volume 2. Lucknow: Munshi Naval Kishore Press; 1903:245.
26. Baqar SM. Ikseer-al-Qulūb. 1st edition. Central Council for Research in Unani Medicine (CCRUM); 2010:268.
27. Al-Din NB, Kabiruddin. Kulliyat Nafisi. Urdu translation. Volumes 1 and 2. New Delhi, India: Idara Kitab-ul-Shifa; 1934:223.
28. Arzani HMA. Mizan-i Tibb. Delhi: Idare Kitab al-Shafa; 2002:182.
29. Rushd I. Kitab Al-Kulliyat. New Delhi: Central Council for Research in Unani Medicine; 1987:224.
30. Farooqi A, Sheikh N, Muqaddam M. Evaluation of efficacy of Unani compound formulation Saffofe Hissat in the management of Hissatul Kulliya (Nephrolithiasis). Natl Medicos. 2012;18:15-8.
31. Kumar R. Study of Hissat-i Kulliya (Renal Calculus) with therapeutic evaluation of Unani formulation in its management. Rajiv Gandhi University of Health Sciences; 2011.
32. Ibn Al-Qaff. Kitab Al-Umda fi'l Jarahat. Volume 2. New Delhi: Central Council for Research in Unani Medicine; 1986:235.
33. Haddad FS. Highlights on the history of medicine in the Middle East. T Klin J Med Ethics Law Hist. 2002;10:243-57.

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