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AN OVERVIEW OF NEPHROLITHIASIS (HISAT UL KULYA) THROUGH THE LENS OF UNANI AND CONTEMPORARY MEDICINE.

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Abstract: **Nephrolithiasis (Hisat e kulya) are among the most prevalent urological conditions encountered in clinical practice, often characterized by recurrent episodes, pain, and potential complications such as infection or renal impairment. Modern treatment ranging from pharmacological agents to surgical intervention offer symptomatic relief but often fall short in addressing recurrence and underlying causative factors.**

Unani medicine provides a distinct perspective rooted in centuries of clinical experience.it attributes stone formation not only to dietary and environmental factors, but also due to weakness of kidney, thick and viscous humor, concentrated and sticky fluid, su -e-mizaj kulya (weakened temperament of kidney), warm -e- Kulya (Nephritis), Qurooh -e- kulya (kidney ulcer), high virulent temperature and a weakning of Quwwat e Dafey'ah (expulsive power). This paper explores the clinical relevance of these concept in the pathogenesis, prevention, and management of nephrolithiasis. A Comprehensive unani management approach including Ilaj bil ghiza (dietary regulation), Ilaj bil Tadbeer (regimental therapy), and Ilaj bid Dawa (pharmacotherapy) emphasizes correction of renal temperament, elimination of lithogenic matter, and strengthening of renal expulsion. Key pharmacological agents with litholytic, diuretic, anti-inflammatory, and nephroprotective actions are highlighted, including both single and compound unani formulations.

Keywords- **Hisat -e-Kulya, Unani medicine, Su -e-mizaj e kulya, Warm -e-kulya, Qurooh-e-kulya, Quwwat e Dafey'ah, Nephroprotective.**

I. INTRODUCTION

Nephrolithiasis is a complex, multifactorial common disease. It occurs in 0.1-0.3%, population males being affected more than female, predominantly in young individual 18 – 45 years withs peaks in the late 20 's and early 30's. The calculous in India though seen in every state but more prevalent in Gujarat Rajasthan, Madhya Pradesh and parts of Andhra Pradesh. contrary to belief, recurrence of stone is common and the recurrence interval between each episode reduces. [(1)]

The word nephrolithiasis comes from ''NEPHRO '', which is the Latin word for KIDNEY and ''LITHIASIS'', which is the medical term used to refer to STONES[(2)].It is most complex process result from imbalance between PROMOTER'S (Calcium, oxalate, phosphorus, urate and hydrogen) and INHIBITOR'S (Magnesium,citrate,nephrocalcin,osteopontin,Tamm-Harsfallprotein,pyrophosphate and glycosaminoglycans) in kidney which cause accumulation of crystalline mass /concretion/solid material in tubal system of the kidney. These urinary concretions vary greatly in size from particles like sand in urinary tract to large round stone in the bladder [(1) (3)].

Renal calculi are one of the oldest known medical conditions dating back to 4800 BC. A bladder stone was discovered in 1901 by English archeologist E. Smith in 4500–5000-year-old mummy at El Amrah Egypt, highlighting early awareness among ancient Egyptians their medical texts from 1500 BC also mention treatment for stones, marking the beginning of scientific understanding and healthcare practices in human history [(4)].

Hippocrates described kidney and bladder disease and in his ''OATH OF MEDICAL ETHICS'', advised physicians not to perform surgery for bladder stones, leaving it to specialized lithotomist and he adamantly stated that wounds of the bladder were lethal [(5)]. Ammonius of Alexandria (276 BC) was the first person to introduce the technique of crushing urinary stones to ease their removal.

According to sheik two main things are responsible for calculi formation i.e., Ghaliz madda and injamat e ghaliz madda. And Samarqandi states that the functional cause of Hisat ul kuliya is Hararat (temperature) and Maddi (morbid matter) cause is ghaliz laisdar rutubat i.e. Balgham, ream, ghaliz khoon and sometimes due to urinary passage constriction in kidney [(6)].
According to unani literature renal calculi are more common in middle aged individuals and bladder stones are more common in adolescence [(7,8)]

Source	Age/Group Affected	Cause/Explanation
Unani Literature	Middle-aged individu Adolescents Adolescems	Renal calculi are more common due to low Hararat (heat) and weak Quwwat-e-Hazima
Ismail Juruani	Bladder calculi Are more common Hararat-e-Ghariziyhah (innate heat) and strong Quwwat e Dafiya	Renal stones more common duo to weak Quwwat-e-Hazima (digestive power)
Hakeem Azam Khan		Nephrolithiasis is common due to strong Quwwat-e-Dafiya, improper diet, thick and viscous urine, high salt content, and increased Hararat

II.RISK FACTOR:

1.Excessive Hararat cause the dryness of rutubat & enhance the ghilzat, which ultimately leads to stone formation.
2.Age -20-30 years
3.Gender – men more than women (Due to higher testosterone level & lower urinary citrate which reduces natural stone inhibition in men's & in women's estrogen reduce calcium oxalate crystal adhesion &urinary oxalate excretion).
4.Geographic factors – Area of highly humidity and elevated temperature, urinary solutes will increase with decrease in colloids, which leads to chelation of solute with calcium forming a nidus for stone.
5.Diet – rich diet of animal protein, oxalate, sodium chloride and refined carbohydrates, low fluid intake and low fibre diet. Citrate level in urine (300-900 mg /24 hrs) maintain the calcium phosphate & carbonate in soluble state & any decrease in citrate level in urine causes stone formation. [9]

III.PRE-DISPOSING FACTOR:

1.**Environmental and dietary**:
Low urine volume, high ambient temperature, low fluid intake, Diet – high protein intake, high sodium, low calcium, High sodium excretion, High vitamin C intake, High urate excretion, High oxalate excretion, Low citrate excretion
2.**Acquired causes:**
Hypercalcemia of any cause, Ileal disease and ileal resection (increase oxalate absorption and urine excretion), Renal tubular acidosis (Distal- type 1)
3.**Congenital and inherited**
Familial hypocalciuric, Medullary sponge kidney, Renal tubular acidosis, Primary hyperoxaluria, Cystinuria, Vitamin B6 deficiency, Anatomical abnormality, Low urine volume, Change and urinary pH, Deficiency of stone inhibitors, stress. [10,19]

IV.AETIOPATHOGENESIS:

According to Ibn-e-Sina (980–1037 AD), kidney stones form due to a disturbance in Quwwat-e-Fa'elah (active power), leading to increased renal temperature and the production of maddat -ul- hisat (lithic matter), a thick, sticky matter comprising phlegm, viscous blood, or pus. When the kidney's expulsive power weakens due to altered temperament, inflammation, or ulceration, this matter fails to be expelled and accumulates in the calyces, resulting in stone formation [2,11].

According to Ibn-e-Zuhr (1091–1161), kidney stones form when the kidney loses its ability to expel thick humors due to weakness. These humors accumulate and gradually undergo crystallization layer by layer, eventually forming stones [12,11].

According to Ali Bin Abbas Majusi (930–994 AD) in Kamilus Sana'ah, renal calculi result from Shadid Hararat (excessive heat) which thickens khilt -e -ghaliz (viscid humors). This leads to drying and hardening of body fluids, forming stones. Increased viscosity of bile contributes to gravel formation, while persistent, thick humor promotes the development of hard and large calculi, which are not easily excreted and eventually crystallize in the kidneys, forming renal stone [13,14].

According to Galen and Avicenna

Galen attributed the formation of renal calculi to the accumulation and solidification of abnormal matter within the kidneys. Galen postulated that Rih (Gaseous matter) get trapped in the renal spaces & hardens, resulting in stone formation. He also noted that ulceration of the kidney (Qurooh-e-Kulya) could lead to pus accumulation, which further solidifies to form calculi.

Avicenna expanded on this view, asserting that the persistence of morbid matter in the urinary tract—particularly thick, viscous substances (madda ghaliz)—contributes to stone formation. He identified dietary factors such as stale milk, cheese, and fried meat as aggravating causes. According to him, weak digestion (zauf-e-hazm) and impaired kidney expulsion capacity (zauf-e-quwwat-

e-Dafey'ah) allow this matter to persist and form calculi. Conditions like cold temperament of the kidneys (baroodat-e-kulya) and inflammation (warm-e-haar) were seen as contributing factors [8,14].

According to Nuh bin Mansoor, the formation of kidney and bladder stones is attributed to the narrowing at the neck of these organs. This constriction, along with increased bodily heat (hararat), draws thick, viscid matter (madda) toward the urinary tract. When excessive movement (harkat) occurs, it transforms the moisture (rutubat) into a dense form. Over time, these particles accumulate and solidify, eventually leading to the development of renal stones [15].

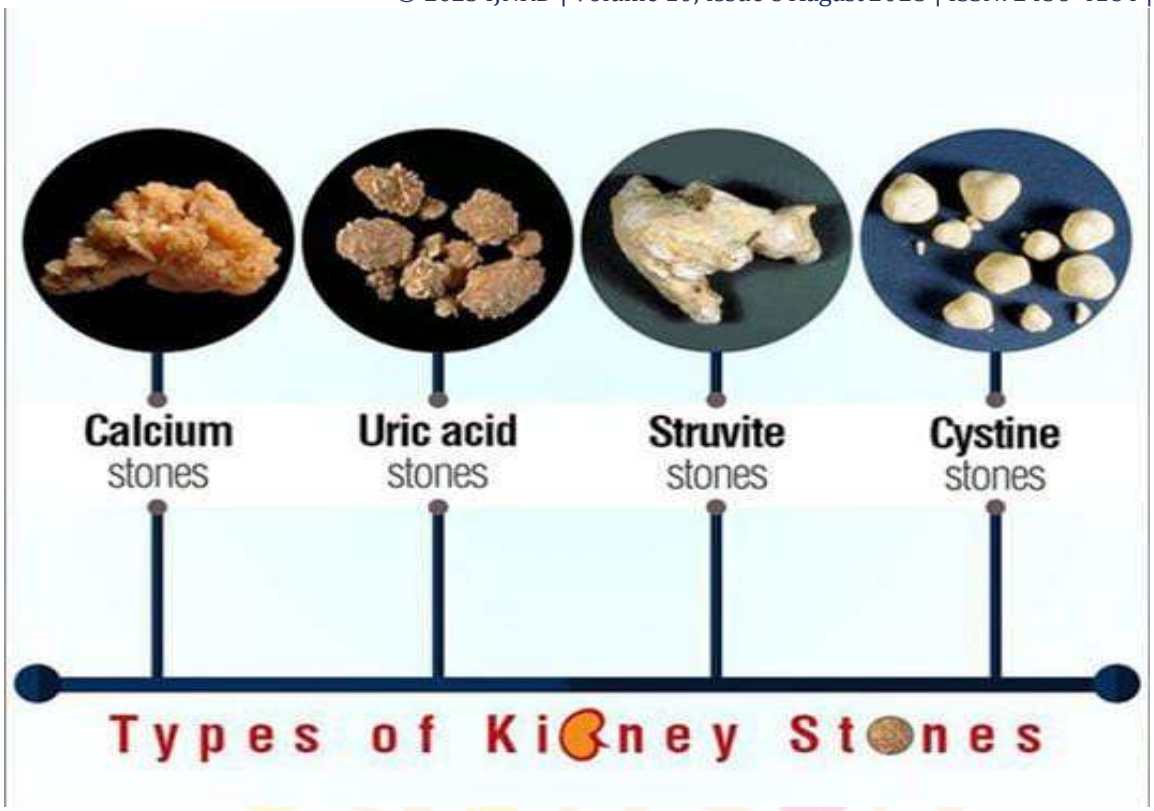
V.CAUSES

There are many causes for urinary-tract stone disease which can be classified into the following groups:

I) Vitamin A Deficiency -it causes desquamation of epithelium which act as a nidus for stone formation.
ii) Idiopathic calcium urolithiasis
iii) Hypercalciuric disorders
    1. Hyperparathyroidism
    2. Prolonged immobilization
    3. Milk alkali syndrome
    4. Sarcoidosis
    5. Other causes include disseminated neoplastic disease, Cushing disease, hypervitaminosis D
iv) Renal tubular acidosis (RTA) only types 1
v) Cystinuria
vi) Uric acid lithiasis
vii) Enzyme disorder
    1. Hyperoxaluria
    2. Xanthinuria
    3. 2,8-dihydroxyadeninuria
viii) 2° urolithiasis
    1. 2° hyperoxaluria
    2. dietary excess
    3. infection
    4. obstruction & stasis
    5. medullary sponge kidney
ix) urinary excretion
x)Drugs
    1. Acetazolamide → stimulates RTA
    2. Allopurinol →precipitate xanthine stone
    3. Thiazide diuretic → ↓ Ca^{+} excretion → ↓ Uric acid stone formation
xi) other factors
    i) geography
    ii) climate & season
    iii) water intake
    iv) diet
    v)occupation especially sedentary Jobs in hot environment. [15] [17]

TYPES OF RENAL CALCULI:

According to chemical composition, approximately 80% of renal stones are comprised of calcium-containing compounds, predominantly:
Calcium oxalate monohydrate (40–60%)
Calcium oxalate dihydrate (40–60%)
Calcium phosphate stones Collectively known in Unani terminology as (Hist-e-Qalmooqiyah).
Other types include:
Hydrogen phosphate (Brushite) (2–4%)
Calcium orthophosphate (<1%)
Uric acid and urate stones (Hisat-e-Bauliyah) — formed from uric acid or its salts like ammonium/sodium urate (10%)
Struvite (magnesium ammonium phosphate) — ~1%, associated with infections caused by urease-producing bacteria



Cystine stones (Hisat-e-Zubaniyah) — ~1%, containing sulfur
Xanthine stones (Hisat-e-Laiyyinah) — rare (~1%)
Mixed compositions can account for 50–60% of cases. [10,17,18,20]

VI.PATHOGENSIS:

Renal stone formation results from a delicate imbalance between the solubility and precipitation of urinary salts. Although the kidneys conserve water effectively, they also excrete substances with low solubility. This necessitates a balance influenced by dietary habits, climate, and physical activity. Under certain conditions, especially dehydration or high solute excretion, the urine becomes supersaturated with insoluble substances like calcium, oxalate, phosphate, or uric acid, which can lead to crystal formation and aggregation.

SUPERSATURATION AND METASTABILITY

Supersaturation is a state in which urine contains a higher concentration of stone-forming salts than it can stably hold in solution. When this limit is exceeded, the urine becomes meta stably supersaturated, meaning it hold excess solutes without immediate crystallization. However, if crystals or nucleating agents (e.g., cell debris) are present, crystallization may initiate once the upper limit of metastability is crossed. This excessive supersaturation is a key contributor to stone formation.

CHEMICAL INTERACTIONS AND PH INFLUENCE

Stone-forming ions such as calcium, oxalate, and phosphate exist in urine as part of complex soluble compounds. However, their free ionic activity increases with the loss of binding ligands like citrate, enhancing supersaturation. Urine pH plays a critical role in determining the type of crystals formed. For example
Acidic urine (pH < 5.5) favors uric acid crystal formation.
Alkaline urine promotes precipitation of phosphate-containing stones like brushite and apatite.
Fluctuations in hydration status, urinary pH, and transient spikes in solute excretion (e.g., postprandially) can significantly increase supersaturation, heightening the risk of stone formation**.**

CRYSTALLIZATION AND STONE GROWTH

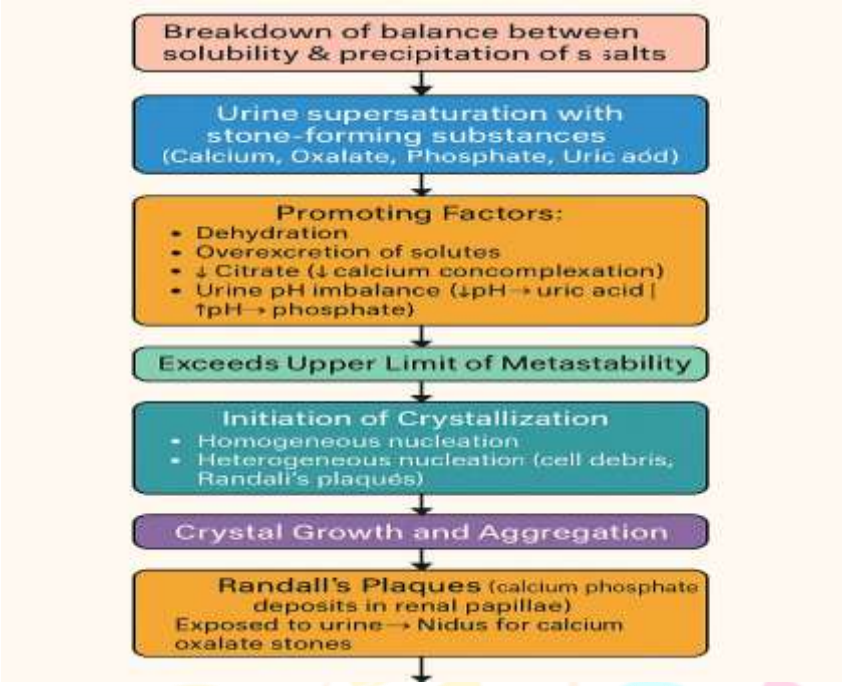
Once the upper metastable limit is surpassed, nucleation occurs, particularly through heterogeneous nucleation, where existing particles (e.g., cell debris or Randall's plaques) serve as nucleation sites. This (e.g., cell debris or Randall's plaques) act as templates. This reduces the energy required for crystal formation. If the urine remains supersaturated, these nuclei grow and aggregate into clinically significant stones.

ROLE OF RANDALL'S PLAQUES

Recent studies highlight that many calcium oxalate stones originate from Randall's plaques, which are subepithelial deposits of calcium phosphate located in the renal papillae. These plaques provide a nidus for heterogeneous nucleation of calcium oxalate crystals. When exposed to urine due to urothelial damage, they become active sites for stone development.

INHIBITORS OF CRYSTAL FORMATION:

Urine naturally contains several inhibitors that prevent stone formation by interfering with nucleation, growth, and aggregation of crystals;



RENAL STONE FORMATION

Citrate is a major inhibitor that reduces supersaturation by chelating calcium, thereby preventing crystal growth.
Inorganic pyrophosphate inhibits calcium phosphate crystallization more effectively than calcium oxalate.
Glycoproteins and other urinary macromolecules also inhibit calcium oxalate crystallization
Despite the presence of these protective factors, stone formation can still occur when inhibitory mechanisms are overwhelmed by high supersaturation or other promoting factors.[16,17]

RENAL DYSFUNCTION IN RENAL CALCULI

Renal stones can cause renal dysfunction through obstruction, infection, or direct damage to kidney tissue. Persistent obstruction due to stones leads to hydronephrosis, reduced glomerular filtration, and eventual renal parenchymal damage. Recurrent infections associated with stones further aggravate renal injury. In cases of bilateral stones or solitary kidney involvement, even partial obstruction can significantly impair renal function. Timely intervention is crucial to prevent progression to chronic kidney disease.[6,21]

VII.CLINICAL FEATURES:

Symptoms of nephrolithiasis depend on their size, shape and their location in urinary tract
1)Colicky pain (pain radiating from loin to groin)
2)Hematuria -due to damage to wall of ureter and urethra
3)Dysuria
4)Oliguria
5)Nausea and vomiting
6)Fever
7)Burning micturition
8)Deep tenderness present in lion region
9)Pulse rate and BP increase due to pain. [10,17,19,21]

VIII.COMPLICATIONS:

Hydronephrosis
Perinephric abscess
Anuria
Infection
Renal injury
Malignancy
Chronic kidney Disease [19]

IX.DIFFERENTIAL DAIGNOSIS: [4,19,23]

CONDITION	PAIN CHARACTERISTICS	DFFERENTIAL POINTS FROM RENAL CALCULI
Acute Appendicitis	Dull periumbilical pain shifting to sharp RLQ pain	Localized to RLQ; no radiation to groin; no hematuria
Acute Pyelonephritis	Dull, constant flank pain	Associated with fever, chills, and pyuria; no colicky nature
Acute Intestinal Obstruction	Crampy, intermittent colicky abdominal pain	Central abdominal pain; bowel disturbances; no urinary symptoms
Biliary Colic	Intense, colicky pain post fatty meal	RUQ pain; related to meals; no hematuria or urinary complaints
Renal Tuberculosis (TB)	Dull, persistent flank pain	Chronic symptoms; sterile pyuria; systemic TB signs
Renal Infarction	Sudden, severe, sharp flank pain	Associated with hypertension, hematuria, and embolic history
Transitional Cell Tumor	Mild or dull pain (often late)	Painless hematuria is common early; no colicky nature
Renal Adenocarcinoma	Dull flank or lumbar pain	Palpable mass, weight loss, and systemic features

X.INVESTIGATIONS:

1: -Plain Abdominal x-ray
2: - CT KUB (Non-contrast -gold standard)
3: - USG

BLOOD SAMPLE -TO RULE OUT

Calcium
Phosphate
Uric acid
Urea and electrolytes
Parathyroid hormone
Bicarbonates

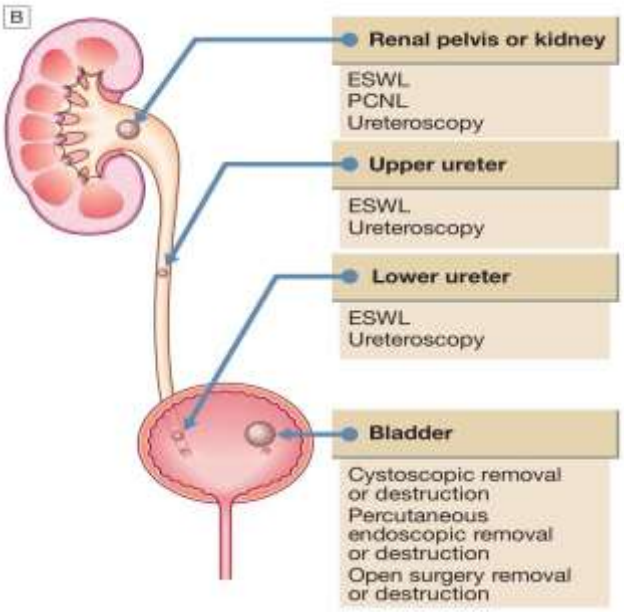
URINE SAMPLE- Dipstick for protein
Blood glucose
Amino acids

24 HOUR URINE -yield more detailed investigation which is reserved for young patients those with recurrent /multiple stones or those with complicated presentation. [10,17]

XI.MANAGEMENT:

→ Immediate treatment of renal colic is with analgesic & antiemetic's.
→ Around 90% of stones of less than 4mm diameter pass spontaneously
→ Pt's with renal / ureteric stones are at higher risk of infection is surgery is contemplated, the pt should be covered with appropriate antibiotics.

→ Immediate action is required if infection occurs in the stagnant urine proximal to the stone (pyonephrosis)and in pt.'s with a solitary kidney who develop anuria in association with stone in the ureter. Stones that don't pass spontaneously through urinary



tract may need to be removed surgically i.e.

-PCNL
-ESWL
-CYSTOSCOPY
-URETEROSCOPY. [10]

XII.USOOL E ILAJ

Unani physician adopted a well-organized line of treatment in the management of Hisat ul Kulya (nephrolithiasis)
1: -Izala-e-sabab
2: -Remove the Asbabe Maddi
3: -Use of taskeen e dard advia (pain relief drugs)
4: - Use of muftit e-hissat advia (lithotriptic drugs)
5: - Use of mudir-e-baul advia (diuretic drugs)
6: -Hifz-e-mataqaddum (preventive measure for recurrence) [7,8,24,25]

XIII.ILAJ (TREATMENT)

In the Unani system of medicine, the principal aim of the treatment for Hisatul Kulya (Nephrolithiasis) is to make morbid and abnormal humor's easily extractible from the body through excretory system.
It basically provides 3 types of therapy as follow:
1. Ilaj bil Ghiza (dietotherapy)
2. Ilaj bit Tadbeer (Regimental therapy)
3. Ilaj bil dawa (Pharmacotherapy)

ILAJ BIL GHIZA

→ Prevention of Renal Calculi focuses on ↑ water intake & dietary changes which also help to maintain healthy lifestyle.
→ ↑ fluid intake helps to flush away the kidney stone out of body.
→ Coffee, alcohol, tea & soda consumption should be avoided, esp. during stone flushing as they can produce dehydration and this may worsen the pain.
→ Kidney beans are rich sources of fiber, minerals & Vit-B that help in cleaning the kidney.
→ Easily digestible foods like — Aab -e- Naryal (coconut water), jau, barley, naspati(pears), magz e badam (almonds), carrot, citrus juices (lemon, orange), olive oil &pomegranate help dissolve or pass stones due to their urate & potassium content.
→A high quantity of oxalate contains diet such as spinach, amaranth leaves, tomato, amla, cheeko, cashew nut, cucumber & uric acid containing diet such as cauliflower, pumpkin, mushroom & brinjal should be avoided.
→Dietary calcium intake helps in binding with oxalates, therefore low Ca^{2+} diet do not prevent kidney stone. A hard & late digestive diet should also be avoided. [20,24]

IlAJ BIT TADBEER (REGIMENTAL THERAPY)

Basic aim of Regimental therapy is to change the consistency of morbid matter into soft & easily digestible expulsive form & resolving disease matter (taqtee-e-maddah).
Various therapies are performed for the above purpose:
1.Purgation (Mushilat)
2. Vomiting (Emesis)
4. Fasd (Venesection)
5. Takmeed - Abzan (Sitz bath), Nutool, Qatoor

MUSHIL (PURGATIVES)
like sapistan, Anjeer, Asalusoos & Khatmi can be used as a mild purgative.

QAI (EMEMSIS)
The emesis serves dual purpose – it is an instant measure – can be repeatedly given & secondly it can be used to expel the entire morbid matter from GIT in a shorter time.

HUQNA (ENEMA)
Luaab - e tukhm-e-khatmi (Althoea officinalis), luaab-e-Hulba (trigonella foenumgraecum) & luaab e katan (linum usitatissimum) can be given as huqna.
Orally Roghan e Badam (almond oil) with maghz e amaltas (cassia fistula) can be given in obstruction as enema.

FASD (VENESECTION)
Should be done in Rag e basaleeq (Basilic vein) when severe pain arises.

TAKMEED, ABZAN, NUTOOL
To relieve the pain the pt is advised following –

Therapies which sedate the pain:
It should be prepared using a decoction containing musakkin wa murakhi advia unani (sedative) drugs such as:
Karafs (Apium graveolens), Qurtum (Carthamus tinctorius), Baboona (Maticaria chamomilla), shibt (Anethum Sowa), Hulba (Trigonella foenum), Pershioshan (Adiantum capillus), Banafshah (Viola odorata) and Beekhe-e-kibr (Capparis spinosa) can be used. [6,13,20,24,25]

ILAJ BIL DAWA (Pharmacotherapy)
The recommended principles of treatment to control nephrolithiasis & to expel out the destroyed stones are illustrated as:
Tafteet e hisat (Litholytic / lithotriptic), Idrar -e-baul (diuresis), Tahleel-e-Wasam (Resolution), along with Taqwiyath e kulya (Nephroprotective)
Advia e mufradah (Single drugs) [6,11,13,24,26,27]

UNANI NAME	SCIENTIFIC NAME	FUNCTION
Aalu Balu	Prunus cerasus Lin	Diuretic, Lithotriptic, Anti-inflammatory
Khare-e-Khasak	Tribulus terrestris Linn	Diuretic,
Charchatah	Achyranthes aspera Linn	Lithotriptic, reduces swelling
Kulthi	Dolichus biflorus Linn	strong lithotriptic
Tukhm-e-Gazar	Daucus carota Linn	Diuretic, supports renal function
Tukhm-e-Karafs	Apium graveolens Linn	Diuretic, carminative
Tukhm-e-Kurfah	Portulaca oleracea Linn	Diuretic, anti-inflammatory
Tukhm-e-Kharpazah	Cucumis melo Linn	Diuretic, cooling effect
Tukhm-e-Khayar	Cucumis sativus Linn	Diuretic, soothing for urinary tract
Tukhm-e-Kasni	Cichorium intybus Linn	Detoxifies kidney, mild diuretic
Tukhm-e-Turb	Raphanus sativus Linn	Dissolves stones, improves urine flow
Qurtum	Carthamus tinctorius Linn	Diuretic, blood purifier
Beekh-e-Neil	Ipomoea nil Linn	Diuretic, anti-inflammatory
Beekh-e-Ghar	Prunus laurocerasus	Renal tonic, supports excretion
Beekh-e-Kibr	Capparis spinosa	Anti-inflammatory, supports liver and kidney function
Dooqu	Peucedanum graveolens	Diuretic, reduces obstruction in urinary tract
Beekh-e-Halyoon	Asparagus officinalis Linn	Strong diuretic, nephroprotective
Habb-e-Balsan	Commiphora opobalsamum Linn	Anti-inflammatory, mild diuretic
Persiya Shah	Adiantum capillus veneris Linn	Renal detoxifier, supports urine output
Sang-e-Sarmahi	Fish stone	Lithotriptic
Hajrul Yahood	Lapis judaicus	Strong lithotriptic
Aqrab Sokhtah	Burnt scorpion	Lithotriptic (used in minute amounts)
Shorah Qalmi	Potassium Nitrate	Diuretic
Kharateen	Earth worm	Lithotriptic, Anti inflammatory

ADVIA MURAKABAT (COMPOUND FORMULATIONS)
1) Majoon e Hajrul Yahood
2) Majoon e Aqrab
3) Qurs e kushta Hajrul Yahood
4) Qurs e kaknaj
5) Majoon e Sang e sarmahi
6) Sharbat e Bazoori Motadil
7) Sharbat e Annanas
8) Sharbat e Aalu Balu
9)Jawarish e Zarooni Sada
10) Jawarish e Jalinoos

11) Jawarish Zarooni Ambari Ba nuskha e kalan
12) Akseer e gurda
13) Hab e dard e gurda. [28,29]

XIV.PREVENTIVE MEASURES

The Unani system of medicine provides insight into managing Renal Calculi through herbal & dietary measures.
1. Fluids – at least 2L output/day (intake 3-4 L)
– Intake should be throughout the day
– Clear & colorless urine excretion
ii) Sodium – Restrict intake
iii) Protein – moderate, not high
iv) Ca^{2+} – Maintain good Ca^{2+} intake (Ca^{2+} forms an insoluble salt & dietary oxalate, lowering oxalate absorption & excretion)
– Avoid Ca^{2+} supplements (separate from meals)
(↑ Ca^{2+} excretion without altering oxalate excretion)
v) Oxalate – Avoid food that are rich in oxalate
(Spinach, Strawberries)
DRUGS
vi) Thiazide diuretics → ↓ Ca^{2+} excretion
valuable in recurrent stone formation & hypercalciuria
Allopurinol -
– if urate excretion is high
AVOID
– Vit D supplements (↑ Ca^{2+} absorption & excretion)
– Vit C supplements (↑ oxalate excretion) [10,16,19]

XV.COURSE AND PROGNOSIS:

Renal calculi often recur, Prognosis depends on underlying systemic or metabolic factors and the extent of renal damage. Nephrocalcinosis significantly worsens the outcome, especially when both kidneys are involved, even if the primary cause is treatable. [22]

XVI.RECURRENCE:

Renal calculi have a high recurrence rate, with up to 50% of patients developing another stone within 5–10 years. Recurrence is more common in men due to persistent metabolic and dietary risk factors. Calcium oxalate stones are most prone to recur, especially in those with hypercalciuria or low fluid intake. Lack of preventive measures and poor compliance with medical advice further increase the risk. [31]

XVII.CONLUSION

Renal calculi (Hisat-e-Kulya) remain a common and challenging clinical condition, with high recurrence and significant patient morbidity. While conventional medicine often focuses on surgical and pharmacological removal, the Unani system provides a more comprehensive view by addressing both the formation and recurrence of stones. A key insight in Unani medicine is the role of Su-e-Mizaj (disordered Mizaj or temperament) and the weakening of Quwwat-e-Dafiyah (expulsive power), which allow the accumulation and retention of lithogenic substances.

As a Moalijat scholar, it becomes essential to bridge traditional principles with clinical application. Unani pharmacotherapy (Ilaj bil Dawa) using litholytic (Mufateet-e-Hisat), diuretic (Mudir-e-Baul), anti-inflammatory, and nephroprotective (Muqawwi-e-Kulya) drugs shows promising outcomes, especially in early stages and recurrent cases. Regimental and dietary therapies further strengthen the Unani approach.

Thus, the Unani system not only aims at symptomatic relief and stone expulsion but also focuses on correcting the underlying renal dysfunction, thereby reducing recurrence and improving renal health. The integrative and preventive approach offers a valuable aid to mainstream nephrology, particularly in regions where the disease burden is high and access to advanced interventions is limited.

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