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Hasaat-Kuliyah (Nephrolithiasis): A review with unani concept

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Abstract

Nephrolithiasis is prevalent in 5% of population and most common type of stones is made up of calcium oxalate (Hissat-e-Tootiyah). Risk of developing recurrent stone is 50% within 5-7 years. Stones are formed from high crystals present in urine like calcium, oxalates, uric acid and low citrates, this imbalance result in stone formation. Chances of renal calculi are more common in men than women because women excrete more citrate and hence have lower incidence of stone formation. There are other risk factors which are associated with formation of stone like environmental factor, genetic factor, dietary factor and different medical conditions like gout, primary and secondary hyperparathyroidism, vit-D deficiency, excess intake of vit-D, obesity, chron's disease. Metabolic disorders like hypercalciuria, hyperoxaluria, hyperuricemia and hyperuricosuria are associated with recurrent kidney stone formation. According to Unani philosophers potential factors responsible for nephrolithiasis are weakness of kidneys, su-e-mizaj kuliyah, Qurooh-e-Kuliyah.

Keywords: Hasaat-kuliyah, calcium stones, oxalates, metabolism

Introduction

Nephrolithiasis is formation of calculi or stone. Its prevalence is globally particularly in some geographic locations such as U.S, South Africa, East Asia and India. Kidney stone effect 5% of total population, seasonal variation are also seen in nephrolithiasis like during summer calcium oxalate saturation is seen in men and in women during early winters. Stones are seen twice more common in males then females, in men peak age is 30 years and in females it is 35-55 years. Once a kidney stone is formed chances of second stone formation is approximately 50%. Renal calculi are clinically characterized by colicky pain (renal colic) as they pass down along the ureter resulting haematuria.

Classification and pathophysiology

Kidney stones are divided in calcareous (Calcium containing stones which are radio-opaque) and non-calcareous which are radiolucent. There are four types of nephrolithiasis ^[4,1], these are as following

- A) Calcium stones.
- B) Cystine stones.
- C) Mixed stones.
- D) Uric acid stones.
- E) Other calculi.

Calcium Stones: Comprise of 75% of all calculi. They may be pure calcium oxalates (50%) or Calcium phosphate (5%) or mixture of calcium oxalate and calcium phosphate (45%). These stones are usually ovoid in shape hard with granular rough surface, stone appear dark brown due to deposition of blood pigments over them, as calculi cause local trauma ^[4].

Etiology: A) 50% of patient with calcium stones have idiopathic hypercalciuria without hypercalcaemia and about 10% of cases are associated with hypercalcaemia with hypercalciuria like in case of hyperparathyroidism or bowel defect (absorptive hypercalciuria) or renal hypercalciuria ^[11].

B) 15% of patient with calcium stones have hyperuricosuria with normal blood uric acid level without any calcium metabolism abnormality.

C) 25% patient with calcium stones have unknown etiology with no abnormality in urinary excretion of uric acid, calcium or oxalate referred as idiopathic calcium stone disease.

Mixed (Struvite Stones): 15% of calculi are made up of magnesium, ammonium-calcium phosphate often called struvite or mixed stones or triple phosphate stones. These stones are yellow-white or grey colour stones soft and friable with irregular in shape [9].

Etiology: Mixed stones are formed due to infection of urinary tract (urinary bladder) with urea-splitting organism that produce urease such as species of proteus occasionally pseudomonas, Klebsiella and enterobacter. These are called infection induced stones, however E-Coli does not produce urease.

Uric acid stones are found in about half of cases. Uric acid stones are smooth, hard yellow-brown and often multiple, 6% of calculi are made up of uric acid. They are radiolucent solubility of uric acid at PH 7 is 200mg/dl while PH at 5 is 15mg/dl as urine become acidic solubility of uric acid in urine decrease and precipitation of uric acid crystal increase resulting in uric acid stone formation [14, 4]. Hyperuricosuria is important factor for formation of uric acid stones while hyperuricemia.

Etiology: Uric acid stones are frequently formed in case with hyperuricemia and hyperuricosuria due to primary and secondary gout, those on chemotherapy and uricosuric drugs e.g. salicylates, probenacid. Other factors are acidic urinary PH (below 6) and decreased urinary volume.

Cystine stones: Cystine stones are less than 2% urinary calculi. Cystine stones are formed due to excessive excretion of Cystine which is least soluble of naturally occurring amino acids leads to formation of crystals and finally Cystine calculi. Cystine stones are small, round, smooth, yellow, waxy and often multiple [1, 12].

Etiology: Cystine stones are associated with cystinuria due to genetically determined defect in Cystine and other amino acid transportation across the cell membrane of renal tubule and mucosa of small intestine [4].

Other types of stones like: Xanthine, Indigo, indinavir.

Risk Factor for Stone Formation

Precise etiology is not identified in most of the cases. Family history of kidney stones (increase risk three times), Insulin resistant states, Primary hypothyroidism, Hypertension, Gout, Chronic metabolic acidosis, surgical menopause are all associated with increased risk of kidney stones. Incidence is higher in anatomical abnormality of urinary tract, most patient with calcium stone or recurrent history of calculi have metabolic risk factors like Hypercalciuria, Hyperuricosuria, Hypocitriuria and Hyperoxaluria [1, 4, 11].

Hypercalciuria: It is defined as excretion of urinary calcium more than 200 mg in 24 hours or 4mg/kg/24 hours. This is most common metabolic abnormality in patient with calcium stone. Different mechanism is responsible for hypercalciuria like:-

(a) Absorptive hypercalciuria: Increased level of circulating calcium due to increased renal filtration load. This is common condition and most patients remain asymptomatic and do not have any history of stone formation.

(b) Renal hypercalciuria: Renal excretion of calcium is increased due to abnormal tubular absorption of calcium. This occurs in 2% of patient with history of recurrent stone formation.

(c) Resorptive hypercalciuria: Occur in 5% of patients with recurrent stone formation, commonest cause is primary hyperparathyroidism as there is increased resorption of bone [9, 7].

Hyperuricosuria: End product of purine metabolism is uric acid, it is derived either from exogenous source (dietary) or endogenously during cell turn over. Low urinary PH <5.5 is the most important factor in uric acid type of stone formation and history of gout increases the risk of kidney stone formation especially in men [5,9,6].

Hyperoxaluria: It is defined as increase in excretion of urinary oxalate i.e. more than 45 mg/dl, different mechanism cause hyperoxaluria which are as follows:

(a) Enteric hyperoxaluria: Increase in intestinal absorption due to (a) Ileal disease e.g., chron's disease, Ileal bypass etc.

(b) Decreased calcium intake.

(c) Gastrointestinal decolonization of oxalobacter formigenes [5, 11].

(b) Increased ingestion: Dietary oxalate is inversely proportional to calcium intake in healthy people, it contributes to about half of urinary oxalate e.g. Rhubarb, beets, nuts, tea, wheat bran, soya, and spinach increases the oxalate contribution [4].

(c) Primary Hyperoxaluria: is congenital error of oxalate metabolism.

Hypocitriuria: It is defined as urinary citrate excretion of <250mg in 24 hours. Citrate form soluble complex with calcium that inhibits stone formation and promote crystallization. Women excrete more citrate and have less incidence of stone formation than man. Fruits such as oranges, grapes are exogenous source of urinary citrate [1, 7].

Anatomical abnormality that increases the risk of stone formation

(A) Obstruction of pelviuretric junction.

(B) Horseshoe kidney.

(C) Ureterocoele.

(D) Vesicoureteral reflex

(E) Ureteral stricture.

(F) Hydronephrotic renal pelvis or calices.

(G) Tubular ectasia (medullary spongy kidney)

Unani concept of hassaat-e-kuliyah

According to Unani medicine morbid material and stagnation of this morbid material are responsible for renal calculi. This morbid material (Ghaleez madaa) is produced by Ghaleez Aghzia e.g. Ghaleez meat, fish meat, concentrated milk those dietary items which are not easily digested. Su-e-Mizaj kuliyah, warm-e-kuliyah, zof Qoowat-e-Dafey'ah, Qurooh-e-kuliyah are considered potential cause of Hassat-e-Kuliyah [10, 2].

Different theories of Unani philosopher are: According to Ibn-e-Abbas Majoosi (930-994 AD) States that more concentrated humours and highly viscous fluids get adhere to calyces of kidney, these humours and fluids are dried by high virulent temperature to form crystals result in stone formation. Jaleenous describe that nephrolithiasis is caused

by ulcer of kidney [3].

According to Ali Ibn-e-sina (980-1037 AD) nephrolithiasis is formed by active power (Qoowat-e-Taayelah) which raises temperature inside the kidney, also states that stone producing substance is viscous (sticky matter) that may be either phlegm or viscous blood or pus, when Qoowat-e-Daafeah become weak they get retained in calyces of kidney. According to Ibn-e-Zohr (1091-1162 AD) inability of kidney to excrete the thick humour due to weakness, resulting in deposition inside the kidney. According to Zakariya Razi (850-932 AD) States that body produce abnormal humours which are excreted in form of viscid fluid that moves towards kidney and form stone [8].

Clinical manifestation of nephrolithiasis

Colicky Pain-stones in kidney cause pain in lumbar region. Haematuria gross or microscopic in urine occur in 90% of cases. Dysuria, Burning micturation, Urgency, Frequency of micturation, Strangury, renal angle tenderness, lumbar region tenderness are positive.

Systemic symptoms like restlessness, writhing in distress, nausea and vomiting or both, fever and chills or both it is often associated with infection [11].

Investigation

Urine examination, total protein, albumin, bicarbonate, urate. Kidney function test (KFT), Serum iPTH, Thyroid hormones, and serum electrolytes. 24 hours urinary creatinine, protein, oxalates, calcium, urate [9, 4, 11].

Imaging- Abdominal 'KUB' film, Ultrasonography, CT scan, IVP.

General Measures to prevent stone formation

- Fluid intake 2-3 liters/day.
- Decrease salt intake <5 moles/day.
- Decrease in take of animal proteins, as it produces metabolic acids.
- Decrease intake of oxalate rich diet.
- Normal calcium intake >30 mol/day, as low calcium diet increase the urinary oxalate production [7, 6].

Specific treatment to prevent nephrolithiasis [11, 6, 7]:

Hypercalciuria:- Thiazides decrease the urinary excretion of calcium by augmenting tubular reabsorption of calcium. Oral potassium citrate- controls diuretic induced hypokalemia.

Potassium phosphate –Suppress calcitriol synthesis and thereby decreases calcium absorption.

Hyperuricaemia or hyperuricosuria: Allopurinol- inhibits uric acid synthesis and decrease urinary uric acid excretion.

Hyperoxaluria: No specific drug is available to control oxalate excretion in urine.

Pyridoxine- reduces production of oxalate by inducing enzyme activity, as it is a co-factor in alanine-glycoxylate pathway.

Calcium supplement- to control enteric hyperoxaluria.

Cholestyramine decreases the intestinal absorption of oxalates.

Probiotic treatment with Oxalobacter formigenes.

Hypocitriuria

Potassium citrate is advised to increase citrate excretion.

Struvite Stone

Acetohydroxamic acid is urease inhibitor, it reduce the urinary saturation of struvite. Their uses have been limited due to its side effect like DVT and hemolytic anemia.

Cystine Stones

Alkalinization of urine with potassium citrate and increasing urine output.

Specific agents like- α -mercaptopyrionylglycine that form soluble complex with Cystine.

Renal colic: IV fluids or oral fluids, Analgesics (oral, intramuscular, suppository), Antibiotics.

Surgical Treatment

80-90% of stones measuring 5mm pass spontaneously, 50% of stones measuring between 5-10mm need conservative treatment, average time for spontaneous passage of stone is one week- three week. 10-20% of all kidney stone need surgical intervention to remove stone. Large calculi can be treated with ureteroscopy, extracorporeal shock wave lithotripsy, Percutaneous Nephrolithotomy.

Unani Treatment for Hassat-E-Kuliyah

In Unani system of medicine goal for the treatment for renal calculi is to make morbid and abnormal humours easily out of body through excretory system. It involves three types of therapy which are as follows:-

(a)-ILAJ Bil-Ghiza (Dietotherapy)

(B)-ILAJ Bil-Tadbeer (Regimental Therapy)

(C)-ILAJ Bil –Dawa (Pharmacotherapy)

ILAJ BIL-GIZ

Plenty of fluid and easily digestible food is indicated like Aab-e-Naryl (coconut water), carrot, chicken, pear, Almond, goats heart (qalab-e-ghenam) and Sparrow (Asaafeer). High oxalate diet like Amlah, tomato, cashew nuts, pumpkin, spinach, amaranth leaves, mushrooms, cauliflower, brinjal etc should be avoided [13].

ILAJ Bil-Tadbeer

Basic aim of Ilaj bil-tadbeer is to change the consistency (soft) of morbid matter (talteef-e-maddah), Purgation (Mushilat), Huqna (Enema), venesection (Fasad) Sitz bath, (Aabzan), is indicated.

Purgation: Mild purgatives like Anjeer (Ficus Carica), Sapistan (Cordia latifolia), Aslussoos (Glyzyrrhiza glabra), and Maghz-e-Amaltas (cassia fistula).

Huqna (Enema): Huqna of mulayyin and muzliq (Laxative and Emollient) like Tukhm-e-Katan (Linum usitatissimum), Tukhm-e-Khatami (Althoea Officinalis) [2, 10].

Fasad (venesection): Rag-e-Basaleeq (Baselic vein).

Aabzan (Sitz bath): Decoction containing Murakhkhi and Musakkin drugs such as Khatami (Althoea Officinalis), Shibt (Anethum sowa kutz), Hulba (Trigonella foenumgraece), Baboona (Althoea Officinalis), Kurfah (Portutaca oleracea), and Banafshah (Viola odorata), to relive pain. After Sitz bath Rogn-i-banafshah (Violent herb oil) or Rogn-i-soya (Dill oil) should be applied locally [3, 10].

ILAJ-Bil-Dawa (Pharmacotherapy)

(A) Mufatit-E-Hisat (Litholytic/ Lithotriptic)(B)- Mudirr-E-Baul (Diuretics), (C)- Mohalil-E-Waram(Resolvent), (D)- Muqawwiyat-E-Kuliyah (Nephroprotective). Aim is to prevent stone formation and expel calculi out of body [8, 10].

Table 1: Above mentioned single Unani / herbal drug are advised in in renal stone/ nephrolithiasis

Unani Name	Botanical Name	Origin
Habb-ul-Qilt	Dolichos biflorus	Plant origin
Khar-e-Khasak	Tribulus terrestris	Plant origin
Dooqu	Peucedanum	Plant origin
Aalu Balu	Prunus cerasus linn	Plant origin
Beekh-e-Gh"ar	Prunus Laurocersus	Plant origin
Charchatah	Achyranthes aspera	Plant origin
Habb-e-Kaaknaj	Physalis alkekengi	Plant origin
Qurtum	Carthamus tinctorius	Plant origin
Tukhm-e-Khayaar	Cucumis sativus linn	Plant origin
Tukhm-e-Kharpaza	Cucumis melo linn	Plant origin
Hajr-ul-Yahood	Lapis judaicus (Jewes stone)	Mineral origin
Jawakhar	Potassium carbonate	Mineral origin
Shorah qalami	Potassium nitrate	Mineral origin
Sang –e-sarmahi	Fish stone	Animal origin
Aqrab Sokhata	Burnt scorpion	Animal origin
Tukhm-e-Gazar	Daucus carota linn	Plant origin

Unani Advia Murakkab (Compound Pharmacopeia formulations)

Qurs kaaknaj, kushta Hajr-ul-yahood, Majoon Aqrab, Qurs kushta Hajr-ul-yahood, Sharbat Aaloo Balu, Jawarish Zarooni saada, Jawarish Zarooni Ambari, Kushta Hajr-ul-Yahood, Majoon Aqrab, Ikseer-e Gurdah.

Discussion and conclusion

Nephrolithiasis is prevalent globally and is considered common painful condition its incidence is substantially increasing that is one in eleven persons. Although men are at higher risk of developing nephrolithiasis than females. When evaluating patient with kidney stone dietary habits, medication, family history and medical illness is to be evaluated. Calcium oxalate stone are most common type among all renal calculi and risk of developing recurrent stone is 50% more within 5-7 years especially in those who have metabolic disorder like hypercalciuria, hyperoxaluria, hyperuricemia etc. Increase intake of fluids, low salt, low oxalate and low animal proteins food reduce the chance of stone formation. Low calcium diet is not recommended as a calcium stone prevention measure because calcium binds with oxalate in intestinal lumen so it reduces the availability of soluble oxalates for absorption. Animal protein metabolism produce acid in body that reduce urinary PH which increase the risk of uric acid stone formation and high salt intake increase urinary calcium. Drugs that crystallize in urine or alter urinary PH may predispose to stone formation like quinolones, acyclovir, sulfadiazine, acetazolamide etc. Imaging modalities are used to confirm final diagnosis like IVP, KUB, CT SCAN, and Ultrasonography. 90% of stones measuring 5mm pass spontaneously, 50% of stones measuring 5-10mm need conservative management, Unani medicines are beneficial like mufatit-e- hasat and mudri-e-baul advia for crushing and spontaneous expulsion of calculi. Large stone especially those obstructing the urine flow need surgical interference.

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