International Journal of Medicine Research
ISSN: 2455-7404
Impact Factor: RJIF 5.42
www.medicinesjournal.com
Volume 3; Issue 4; October 2018; Page No. 85-87

A single case study of nephrolithiasis under the effect of Unani medicine

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Abstract
A study was carried out on 34 year old married male patient, who was diagnosed with bilateral nephrolithiasis associated with some metabolic disturbances like hypercalciuria, hyperoxaluria, hyperuricemia and vitamin D deficiency. Patients have history of recurrent stone formation. All mentioned said metabolic disorders are interlinked with each other. Patient was put on Unani medicine like Mudri-e-Boul Advia, Muffatit-e-Hissat Advia, Vitamin-D and Vitamin-B_6 supplement and some strict dietary advices for two months and patient got improved.

Keywords: nephrolithiasis, oxalates, calcium, Unani medicine

1. Introduction

Nephrolithiasis is a condition in which stone are formed in kidneys, this condition is prevalent in 5% of population, 90% stones are calcium carbonate and main cause of recurrent stone formation is hyperparathyroidism. Stones are formed from crystals present in urine, crystal forming substance like calcium, oxalates and uric acid are high and Citrate (inhibits stone formation) are low, this imbalance result in formation of stone and stone formation is more in males then females, however genetic factor, environmental exposure and dietary factor are associated with risk of developing nephrolithiasis and following medical conditions are associated with nephrolithiasis like primary or secondary hyperparathyroidism, intestinal ailments (Chron's disease, ileal resection etc), Gout, Vitamin-D deficiency, Obesity, different types of kidney anomalies (horse shoe kidney, medullary sponge kidney) Different types of nephrolithiasis are Calcium oxalate, Calcium phosphate, uric acid, struvite and Cystine kidney stones. Clinically patient present with bilateral flank pain, nausea, vomiting, frequent urination or blood in urine, sometime patient are asymptomatic [1, 2, 3, 4].

1.1 Pathogenesis

Patient with recurrent kidney stone usually have different biochemical abnormalities like hypercalciuria, hyperoxaluria, hyperuricosuria, hyperuricemia. It is very important to understand the pathophysiology of above said metabolic disorders [1, 4]. Most of the nephrolithiasis have calcium as main constituent. calcium homeostasis is physiologically under tight control, so to maintain adequate blood levels. Main three sites of control are (a) Bones (b) Kidney (c) GIT. 30-40% dietary calcium are absorbed via small intestine (highest rate of calcium absorption occur in ileum) and colon, out of which 90% is absorbed via small intestine and 10 % via colon. Coming towards the calcium excretion, it is excreted via kidney and gut daily fecal calcium loss is same as that of urinary excretion of calcium. Important component for calcium homeostasis is vitamin-D, source of vitamin-D is diet and UV radiation to skin. Vitamin-D activation involves 2 steps, Liver convert vitamin-D into 25(OH) D_3 via cytochrome P45 system and 25(OH)D_3 is converted into calcitriol in proximal tubules of kidney by enzyme 1-alpha hydroxylase [3, 5]. This calcitriol has significant action at skeletal level it provides sufficient calcium to bone. Another important aspect of calcitriol is its relation with parathyroid gland function. If serum 25(OH) D_3 is decreased PTH secretion is stimulated resulting increase level of serum calcium (hypercalcaemia) and end effect is excretion of more calcium via kidney (hypercalciuria) ultimately result is crystallization of calcium in kidney [2, 3].

Increase level of uric acid in blood is called hyperuricemia, and it is the marker of metabolic abnormality. Uric acid is the end product of purine metabolism and is derived from diet and Vitamin-C metabolism, normally uric acid is excreted via urine (70%) and feces (40%), so to regulate normal level of uric acid in blood [4, 5] Pathophysiology aspect of uric acid is obscure however hyperuricemia occurs due to increased production or decreased excretion of uric acid or combination of both which usually occurs in alcoholic consumption. Globally prevalence of hyperuricemia has increased in recent decades, in males is 5.9% and 2% in females but after post menopause incidence increases. Increase level of uric acid result in deposition of uric acid crystal in joints causing pain and swelling and kidneys resulting stone formation [6, 7]. Another metabolic disorder related to recurrent nephrolithiasis is Hyperoxaluria, this condition is characterized by increase in excretion of oxalates. Primary and secondary are two main types, primary hyperoxaluria is a metabolic error and secondary hyperoxaluria is caused by increased dietary intake of oxalates or excessive absorption of oxalates from intestines like in Ileal resection, Roux-en-Y gastric bypass and crohn's disease. Hyperoxaluria is associated with recurrent calcium oxalate stone because oxalate binds with calcium easily. If the condition remain untreated it lead to deposition of oxalates in

human organs called systemic oxalosis, as oxalates are not destroyed or metabolized by the cells of human body resulting in accumulation.

2. Method

2.1 Participant Information

In this single case study participant is 34 year old married male, occupationally patient has table work (exposure to sunlight is very less). Patient experienced intermittent bilateral flank pain radiating to groins, nausea, and general body weakness, frequent and burning urination. On physical examination renal angles and lumbar were tender on both sides. After complete physical examination and investigation patient was diagnosis with bilateral nephrolithiasis with some metabolic disturbances that is hypercalciuria, hyperoxaluria and Hypovitaminosis.

2.2 Past history of illness

Sudden onset of weakness at of age 13 followed by intake of Arachitol (VIT-D) injection (7-10 Shots) advices by doctor. History of renal stone on left kidney with spontaneous expulsion after taking medicine. Gastric upset.

2.3 Dietary Habbit

Patient was fond of oxalate and protein rich food like red meat, chicken, beverages, barbeques, fruit juice, oranges, grapes etc. Spicy peppery and hot food and habitual of taking less water.

2.4 Family History

Father died because of renal failure, Mother Hypertensive, one brother has history of renal stone.

3. Investigation

Table 1: Pre-treatment investigations

S.No.	Investigations	Results
1.	Colonoscopy	Infective colitis
2.	Eye examination	Normal findings
3.	Serum uric acid	6mg/dl
4.	Serum phosphorus	2.41mg/dl
5.	Albumin serum	4.47g/dl
6.	Alkaline phosphate serum	109U/L
7.	Serum creatnine	1.12mg/dl
8.	Blood urea	39mg/dl
9.	Serum potassium	4.5mmole/L
10.	Serum sodium	136mg/dl
11.	Serum uric acid	7mg/dl
12.	Serum calcium	9.37mg/dl
13.	PH blood	7.38
14.	HCO_3	29.6
15.	Intact serum PTH	78pg/ml
16	24 hours urinary oxalates	107mg/dl
17.	24 hours urinary calcium excretion	387.10mg/dl
18.	24 hours urinary uric acid	442mg/dl
19.	24 hours urinary citrate	5.45mmol/24hrs
20.	25 Hydroxy Vitamin D	10 mmol/L
21.	USG	Shows bilateral multiple renal stones measuring 6mm and 3mm in right kidney and 4mm in left kidney.

4. Intervention

Patient was put on Unani medicine for a period of two month including some supplements and regimental therapy which is discussed below:

4.1 Unani Medicine

1. Diuretic and Alkaliser:-Sharbat Bazoori Motadil. Mufatti-e-hissat Advia: Majun Hajrul yahud half table spoon once in a day.
2. Botanical: Sang Sarmahi, Hajrul yahud, (20 gms) Habul Qilt, Jawakhara, tukhm shibbat (6gms) as powder 5gms twice daily with Sharbat Bazoori 20 ml.
3. Diuretic and Nephroprotective: Jawarish Zarooni half tablespoon once in a day.
4. Supplements: Vitamin-D 2000U daily.
5. Vitamin-B_6: 100mg once in a day.
6. Regimental Therapy: Sun Bath for one hour daily.

4.2 Dietary Advices

Patient was put on moderate calcium diet, as urinary oxalates are inversely proportional to calcium intake. Omega fatty acids, VIT-B6 and VIT-D rich diet is very beneficial. Fish twice in a week, chicken piece once in a week, rest of the day patient was on vegetable diet. Abstains from Oxalate, VIT-C and fat diet like spinach, rhubarb, beets, potato, onion, tomato, chips, nuts and nut butter.Patient was advised to boil vegetables before cooking because it cut down the oxalate content of the stuff. Coming towards the fruits most of fruits are rich in VIT-C but few have negligible amount of VIT-C like Apple, watermelon, pear, and are advised to take daily. In metabolic process of oxalate formation vitamin-c (Ascorbic acid) converts in oxalic acid that adds the hyperoxaluria disorder. Patient was advised to void urine in a container to rule out sedimentation or renal stone and follow up after every eight days. During follow up visits subject was asked to repeat few investigation like USG, spot urine etc so to assess the calcium or protein loss, to rule out the dimension of renal stone which was gradually reducing in each scan.

Table 2: Post treatment investigations

S. No.	Investigations	Results
1.	Serum uric acid	5.2 mg/dl
2.	Serum phosphorus	2.41mg/dl
3.	Serum albumin	4.40g/dl
4.	Serum alkaline phosphate	106U/L
5.	Serum creatnine	1mg/dl
6.	Blood urea	17mg/dl
7.	Serum potassium	4.5mmole/L
8.	Stone analysis	Calcium mono-oxalate
9.	Serum sodium	136 mg/dl
10.	Serum calcium	9.27 mg/dl
11.	Intact serum PTH	44 pg/ml
12.	24 hours urinary oxalates	52 mg/dl
13.	24 hours urinary calcium excretion	320mg/dl
14.	Urine culture	No bacterial growth was found
15.	25 Hydroxy Vitamin D	45mmol/L
16.	USG	No stone found in right kidney, 3mm stone found in lower calyx of left kidney.
17.	Urine culture	No bacterial growth was found

5. Discussion and Conclusion

The single case study was on 34 year old married male who was diagnosed with recurrent nephrolithiasis, which was calcium oxalate stone. Kidney stones are prevalent globally, they are formed due to deposition of crystals like calcium, oxalates and uric acid in kidney, if remain untreated it can lead to complication. USG technique is important for evaluation for renal stones. 24 hours urinary protein, calcium, uric acid creatnine and oxalates, blood biochemistry are important to identify the metabolic disorders and renal impairment. In this case study it seems that renal stone was the outcome of Hypovitaminosis-D that results in hyperoxaluria and disturbance of calcium homeostasis and consequence was hypercalciuria respectively. Patient was put on lithotriptic, diuretic and Alkaliser and Nephroprotective drugs for crushing and spontaneous expulsion of stone. Well planned dietary advice was given to patient so to avoid excess intake of oxalates, vit-C and proteins. Symptomatically patient was experiencing gradually decrease in pain sensation, nausea and backache. Low oxalate, protein, VIT-C, moderate calcium intake and adequate intake of fluid are important to avoid the recurrent renal stone formation.

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