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Effect of Dietary habbit & fluid intake in patients with urolithiasis

[1]Seema L. Jawalekar, [1]Vasant T. Surve, [2]Anil Bhutey

[1]Department of Biochemistry, Dr. Ulhas Patil Medical College, Jalgaon Maharashtra India
[2]Department of Biochemistry, Panjabrao Deshmukh Medical College
Amravati, Maharashtra, India

ABSTRACT

Diet plays an important role in the pathogenesis of kidney stones as dietary ingredients and fluid intake influence the volume, pH and solute concentration of urine. however, it frequently is ignored in the management and treatment and more emphasis given only on fluid intake and removal of stones. The objective of this study to evaluate the effect on urine chemistry before and after dietary modification in patients with urolithiasis. Urine specimens were collected from 100 normal individuals and 100 kidney stone patients and analysed spectrophotometrically for common stone promoters like oxalate, calcium, uric acid , sodium and phosphate and stone inhibitors like potassium citrate and magnesium before and after dietary modification. To assess the subject's diet we used a semiquantitative food-frequency questionnaire. The results were presented as mean ± SD, student's t test was used for statistical analysis. Urinary Constituents like oxalate, calcium, uric acid and sodium concentration & specific gravity were persistently higher before dietary modification. Urinary citrate and magnesium levels were significantly low in stone formers as compared to normal individuals . But no difference was found in phosphate, Creatinine and potassium excretion.After dietary modification urinary Oxalate, calcium , uric acid & specific gravity significantly declined where as Urinary inhibitor citrate, magnesium rose significantly. Urinary sodium declined and urinary potassium raised when compared with earlier values ,but difference was not statistically significant. Our study shows that certain dietary modifications can limit the risk for stone formation. Restricting sodium, oxalate and animal-protein consumption produces changes in the urinary environment and that should benefit the majority of stone formers, including a decrease in calcium and increase in citrate , Magnesium excretion. To prevent recurrence, dietary modifications along with high fluid intake achieving a urine volume of 2 liters per day should be advised in the long term.

Key words – urolithiasis, diet modification, fluid intake, specific gravity, urinary chemistry

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INTRODUCTION

Urolithiasis, like many other disease processes, is influenced by a number of genetic and environmental factors.Fluid therapy should be a front-line approach for all stone formers, because it is safe, cheap, and effective. Diet is a major environmental component; however, it frequently is ignored in the management of individuals with this disorder, with more emphasis placed on minimally invasive stone-removing procedures and preventive medical therapy. **[1]**

Epidemiological studies and metabolic investigations of the chemical composition of urine have suggested that a number of nutrients may influence the formation of stones in the upper urinary tract. These stones, which are predominantly calcium oxalate in composition, are more common in affluent countries where there is relatively high consumption of protein and fat and low consumption of carbohydrate [2].

The availability of calcium for stone formation depends ultimately on the dietary intake, intestinal absorption, excretion in the faeces, transport across cell membranes from the extracellular to the intracellular components of the body fluids and the renal tubular reabsorption of calcium.

The lithogenic potential of dietary protein is believed to be through different mechanisms. It results from a combination of a higher renal load of lithogenic substances, and a tendency towards their increased precipitation in the kidneys. Animal proteins are rich in sulfur-containing amino acids such as cystine and methionine. Oxidation of sulfur to sulfate generates acid load that aggravates calcium mobilization from bones.[3] Calcium forms soluble complex with sulfate generated from the oxidation of sulfur in proteins. Acid load increases calcium mobilization from the bones, and causes hypercalciuria and low urine citrate levels, which is considered the strongest complexing agent for calcium in urine. [4]

The acid load implemented decreases renal tubular reabsorption and imposes an additional risk for negative calcium balance and osteopenia, since urinary calcium excretion rises further[5,6] . In addition, chronic over consumption of animal protein may increase renal mass and thereby up-regulate calcitriol production [7] . This contributes to hypercalciuria by down-regulating parathyroid hormone secretion with subsequent reduction in renal calcium reabsorption[7] .

Dietary sodium increases the risk of urolithiasis. Salt intake expands intravascular volume, which can increase urinary calcium level, likely by decreasing renal tubular calcium reabsorption. Increase in salt intake can induce mild systemic metabolic acidosis, which can lower urinary citrate levels, and increases the risk of calcium precipitation in kidneys.[8]

On the other hand, potassium-rich foods decreases stone formation through a decrease in urinary calcium excretion & high urinary potassium is believed to increase renal tubular phosphate absorption and consequently inhibit 1, 25-dihydroxyvitamin synthesis.[9] Decrease in 1, 25-dihydroxy vitamin slows intestinal calcium absorption. Potassium-rich foods offer the additional advantage of high citrate content thus decreasing the precipitation of urinary calcium.[10]

Its logical that increased fluid intakes should result in an increased volume of more dilute urine and increased frequency of urination and reduces urinary super saturation, thereby reducing the driving force for crystallization and stone formation. Low urine volume thus, considered as independent risk factor for stone formation.

Urine specific gravity, which measures urine density function, may be a better indicator of urinary dilution and a stronger predictor of stone formation. [11] study results suggest that maintaining urine specific gravity below a certain level might reduces the occurrence of urinary stones, along with appropriate fluid intake. Increased fluid intake has been advocated as a dietary management of the prevention of any type of stones. The hydration status, nevertheless, could be monitored by urine specific gravity.

Urine P^H varies throughout the day and is influenced by the amount and type of food consumed. A diet high in animal proteins results in higher acid excretion and lower urinary P^H compared with a vegetarian diet. The formation of calcium phosphate crystals is highly dependent on the urinary P^H (Robertson 1975), where as the formation of calcium oxalate in a solution seems to be independent of variations in P^H within the physiological range. The increase of urinary calcium oxalate saturation was influenced by changes in urinary P^H.

The aim of this study to evaluate the effect on urine chemistry before and after dietary modification in patients with urolithiasis.

MATERIALS AND METHODS

The study included 100 patients with stone disease age ranges from 16- 60 yrs. Kidney stone patients were selected among those attending the local clinics . 100 healthy persons age ranges from 15- 58 yrs who served as controls,with no recent report of ill health of any kind and had no past history of urolithiasis, including that in the family.

The diagnosis of urolithiasis was based on plain abdominal X – ray, ultrasonography or intravenous pyelography. Patient who had history of bowel disease, renal tubular acidosis and urinary tract anomalies were excluded from the study.

Inclusive criteria decided for the patients, who is only suffering with urolithiasis and the patients with urolithiasis and along with other disease are excluded from the study. The procedures were approved by the local ethics committee.

To assess the subject's diet we used a semiquantitative food-frequency questionnaire that inquired about the average use of foods and beverages during the previous year. Information was also collected on the amount of supplemental calcium (such as calcium carbonate) ingested, either alone or in multivitamin preparations.

Twenty four hours of urine samples were collected from both patients and controls were analysed for urine chemistry. Urine volume and concentration of urine calcium, phosphate, uric acid, creatinine was estimated by normal standard colorimetric procedures and in addition urinary citrate ,magnesium and oxalate were also quantitatively estimated by using standard methods.[13,14,15] before modification of diet, and 6 monthly thereafter dietary modification.

Total energy intake for a given person tends to be fixed within a very narrow range, changes in nutrient intake must be made primarily by altering the composition of the diet, not the total amount of food consumed. The mean daily intake of animal protein,oxalate content , sodium decreased & magnesium , potassium,phosphorus and total fluid increased with increasing dietary citrate intake. The average daily alcohol intake decreased with increasing citrate intake. The subjects were classified into 3 groups:

Group I- Purely vegetarian . Total patients 42 out of which 31 are males and 13 are female.
Group II-Frequently non vegeterian. Total patients 58 out of which 45 are males and 12 are female.
Controls - Total patients 100 out of which 75 are males and 25 are female.

Table: 1 showing Laboratory findings of Urinary Constituents before dietary modification.

Parameters	Group I (Purely vegetarian)	Group II (Frequently Non vegetarian)	Controls	P Value
Oxalate (mmol/24 hrs)	0.48 ± 0.29	0.37 ± 0.12	0.29 ± 0.07	0.000
Citrate (mmol/24 hrs)	1.32 ± 0.35	1.0 ± 0.28	1.94 ± 0.29	0.000
Calcium (mmol/24 hrs)	10.5 ± 9.0	12.5 ± 7.0	4.00 ± 0.96	0.0001
Uric acid (mmol/24 hrs)	3.1 ± 1.6	5.4 ± 1.0	2.58 ± 0.64	0.01
Phosphate (mmol/24 hrs)	31.2 ± 8.7	33.4 ± 5.6	31.80 ± 6.06	NS
Creatinine (mmol/24 hrs)	13.6 ± 3.0	13.9 ± 2.8	13.20 ± 2.17	NS
Magnesium (mmol/24 hrs)	2.26 ± 1.20	2.32 ± 1.09	3.82 ± 1.72	0.01
Sodium (mmol/24 hrs)	190.0 ± 59	206.0 ± 66	173.0 ± 56	0.01
Potassium (mmol/24 hrs)	66.6 ± 32.8	64.6 ± 26.5	71.6 ± 22.8	NS

NOTE:- Values expressed Mean ±SD , P value <0.05= significant.

Table: 2 showing Laboratory findings of Urinary Constituents after dietary modification.

Parameters	Group I (Purely vegetarian)	Group II (Frequently Non vegetarian)	P Value
Oxalate (mmol/24 hrs)	0.34 ± 0.13	0.31 ± 0.09	0.001
Citrate (mmol/24 hrs)	1.87 ± 0.33	1.76 ± 0.82	0.001
Calcium (mmol/24 hrs)	6.6 ± 2.0	8.8 ± 4.0	0.001
Uric acid (mmol/24 hrs)	2.7 ± 0.69	3.8 ± 1.2	0.01
Phosphate (mmol/24 hrs)	32.4 ± 6.9	33.7 ± 3.8	NS
Creatinine (mmol/24 hrs)	13.6 ± 2.5	13.5 ± 2.8	NS
Magnesium (mmol/24 hrs)	3.76 ± 1.36	3.21 ± 1.11	0.01
Sodium (mmol/24 hrs)	181.0 ± 34	193.0 ± 52	NS
Potassium (mmol/24 hrs)	73.1 ± 23.8	74.7 ± 26.3	NS

NOTE:- Values expressed Mean ±SD , P value <0.05= significant.

RESULTS

Value of each constituent was compared between controls and stone formers. The value of each urinary constituents was expressed as Mean ± SD and the statistical analysis of the data was performed by students t – test. The analysis was carried out using SPSS 12 . P values < 0.05 were regarded as statistically significant.

Effects of dietary modification before and after on urinary chemistries were compared in study group.

Table 1 shows laboratory findings of Urinary Constituents before dietary modification. The urinary oxalate, calcium, uric acid and sodium concentration were persistently higher in group I & group II of stone formers when compared with normal individual (P < 0.000, P < 0.0001, P< 0.01 and P < 0.01 respectively). Urinary citrate and magnesium

levels were low in stone formers as compared to normal individuals (P < 0.000, P< 0.01). But no difference was found in phosphate, Creatinine and potassium excretion.

Table 2 shows laboratory findings of Urinary Constituents after dietary modification Urinary Oxalate, calcium , uric acid significantly declined in group I & group II after dietary modification (P < 0.001, P < 0.001,P < 0.01) when compared with before dietary modification.

Urinary inhibitor citrate, magnesium rose significantly (P < 0.001 ,P < 0.01), where as no difference was found in phosphate, Creatinine excretion. Urinary sodium declined and urinary potassium raised when compared with earlier values but difference was not statistically significant.

Urine volume, mean for controls was (1586 ± 97) and stone formers was (1671 ± 646) before modification which is statistically not significant.Even after modification there is no appreciable change in volume which was (1734 ± 124) in stone formers.

Urine PH of stone formers (6.09 ± 0.64) was not significant when compared with controls (6.14 ± 0.31) and and no variation in PH after modification.

The urine specific gravity of stone formers (1.021±0.007) was significantly higher than the controls (1.02 ± 0.00) (*P*<0.001), Urine specific gravity after modification significantly reduced (1.010 ± 0.004) in stone formers.

DISCUSSION

Diet plays a crucial role in the treatment of stone diseases. Diet can contribute to the etiology, management or prevention of recurrence of kidney stone because dietary ingredients and fluid intake influence the volume, pH and solute concentration of urine.

The primary goal of dietary manipulation to alter urinary specific gravity and solute concentration is to achieve urine that is undersaturated with calculogenic crystalloids. In patients with urolithiasis, the mean of total daily intake of energy was significantly higher than that of controls, and so as the mean daily intake of dietary animal protein and oxalate content . All patients were consuming the diet of North Maharashtra region, which specifically consist of tomatoes, Spinach, whole-wheat flour and Bajara roti, and frequently non-vegetarian diet.

Diet plays an important role in the pathogenesis of kidney stones. Correlation of the dietary pattern with the incidence of kidney stone disease in the Indian subcontinent had revealed that kidney stone occurred more frequently in the areas where the staple diet has been wheat than among the rice eaters [16]

Whole-wheat flour when consumed as a staple food leads to the production of urine supersaturated with uric acid, which appears to be an essential pre-requisite for the formation of primary stone. In addition to this increased animal-protein intake increases the excretion of uric acid and calcium and lowers urinary citrate excretion, all of which predispose a person to the formation of calcium stones.[17]

Increased uric acid saturation may favour the nucleation and growth of calcium oxalate and / or ammonium acid urate by blocking the action of acid mucopolysaccharide inhibitors. Another possibility could be that whole wheat flour when eaten as a staple food for a long period may lead to the production of some unknown chemical factor that inhibits the reabsorption of uric acid in the proximal renal tubule and thus causes increased urinary concentration of uric acid. Whether or not such a mechanism is genetically determined requires further studies.[16]

In view of our findings that whole-wheat flour and increase animal protein produces saturation of urine with uric acid, it is evident that saturation of urine with uric acid is an essential pre-requisite for the formation of primary kidney stone. It also implies that wheat, when used, as a staple cereal, is the major causative factor in the etiology of kidney stone.

Dietary protein intake increases net fixed acid production and acid excretion, thus inhibiting renal tubular reabsorption of calcium and imposes an additional risk for negative calcium balance and osteopenia, since urinary calcium excretion rises further. [18]

The dietary intake of calories, protein, fat, carbohydrates, purines, calcium and dietary fibre was markedly higher in stone formers than in controls. Distribution by social class was different in stone formers and controls, so the greater affluence among stone formers could have led to a “richer” diet than that consumed by the controls.

Vegetarian diets have been associated with increase excretion of calcium, oxalate and uric acid and a significant increase in oxalate excretion. Dietary intake of oxalate also varies among individuals based on food choices and it is especially high in vegetarians because green leafy vegetables contain large amounts of oxalate .It is present in wide range of food and drinks, including tea, coffee, chocolate and fruit and vegetables. Dietary fibre might increase the risk of stone formation by increasing hyperoxaluria as many foods high in fibre are also high in oxalate The concentration of oxalate voided in the urine plays an important role in urolithiasis, the formation of calcium oxalate kidney stones. On the other hand calcium restriction increases the absorption of oxalate in the gastrointestinal tract leading to an increase of urinary oxalate excretion.,suggesting that the inverse relation between dietary calcium and kidney stones may be due to increased binding of oxalate by calcium in the gastrointestinal tract. Urinary oxalate may be more important than urinary calcium for stone formation, because calcium oxalate saturation of urine increases rapidly with small increases in the oxalate concentration. Therefore, calcium restriction could actually be harmful in that it may lead to increased urinary oxalate excretion.[19]

Sodium intake is another significant dietary risk factor for kidney stone disease and hypercalciuria because urinary sodium excretion is directly correlated with urinary calcium excretion, such that increasing the excretion of one leads to an increase in excretion of the other.

High sodium intake contributes to stone formation in several ways, first it increases the urinary calcium level by reducing renal tubular reabsorption of calcium. Second, high sodium intake can cause a mild reduction in urinary citrate level by provoking mild bicarbonaturia and metabolic acidosis. Third it can increase urinary saturation of monosodium urate, causing urate induced calcium oxalate crystallization.[20] ,the contrary, potassium-rich foods reduces calcium excretion , an effect that would decrease the risk of stone formation lower. High urinary potassium is believed to increase renal tubular phosphate absorption and consequently inhibit 1, 25-dihydroxyvitamin synthesis.[9] Decrease in 1, 25-dihydroxy vitamin slows intestinal calcium absorption. Potassium-rich foods offer the additional advantage of high citrate content thus decreasing the precipitation of urinary calcium.[10]

Before modification of diet citric acid excretion was very low in stone formers, after advice to include citrus fruit in diet and increase in potassium intake and decrease animal protein intake the net and combine effect is significant increase in citric acid excretion, which is considered as urinary stone inhibitor because it reduces urinary super saturation by complexing calcium ions. In addition, it inhibits growth and aggregation of calcium oxalate and calcium phosphate crystals.

Increase intake of of magnesium is favorable as it decreases calcium absorption and increases magnesium absorption which as an inhibitor reduces risk factors of the disease.Magnesium a divalent cation is a complexing agent for oxalate. Magnesium inhibits oxalate absorption and excretion thus prevents its supersaturation. Normally magnesium is complexed with calcium as well as oxalate and decreases its excretion. Thus decreased magnesium in nephrolithiasis results in increased urinary oxalate level, as sufficient magnesium is not available to form the magnesium oxalate complex.[21]

Intake of increased phosphorus has an effect it decreases calcitriol production and enhance urinary excretion of a natural inhibitor of oxalate precipitation, pyrophosphate. Phosphorus has been shown to partially protect the kidney against calcium-induced damage. Phosphorus acts as a urinary acidifier and helps prevent stones from forming in the kidney.[22]

The beneficial effect of increased fluid intake and the subsequent dilution of urine is well known. There was no appreciable change in volume of 24-hour urine between normal individual and stone formers and even after modification. The increased urine volume in patients as compared to normal individuals and with increasing number of episodes could be due to the advice given to the patients to consume large volumes of water in order to avoid recurrence of stone formation.

Enhancement of urine volume may have the added benefit of increasing the frequency of urination, and hence reducing retention time. Urine dilution by increased water intake may increase ion activity coefficient and hence, urinary crystallization, water diuresis also reduces the average time of residence of free crystal particles in urine, dilutes the components of urine that may crystallize and does not reduce the activity of natural inhibitors.[12]

In present study urinary P^H of stone formers was 6.09 ± 0.64, which indicates that at this P^H calcium oxalate solubility is minimum and hence urine get supersaturated with calcium oxalate. Hence P^H 6.09 ± 0.64 is the optimum P^H for stone formation. The P^H was favorable for control group (6.14 ± 0.31), which is not statistically significant even though the stone was not formed, it may be due to the high level of urinary inhibitors such as citrate, magnesium and phosphate.

There was a significant difference found in specific gravity of urine after dietary modification which declines from (1.021±0.007) to (1.010 ± 0.004) in stone formers . Urine specific gravity, which measures urine density function, may be a better indicator of urinary dilution and a stronger predictor of stone formation. If large volumes of urine are excreted, the specific gravity is usually low, where as if small quantities are being eliminated, the specific gravity is generally high. Even though there is no appreciable change in urine volume and pH but significant change in specific gravity observed may be because of the reduction of amount of calcium , oxalate & increase of degree of tubular reabsorption or concentration by the kidney and increase in urinary stone inhibitors like citrate and magnesium.Increasing water consumption to achieve a urine specific gravity <1.021 dilutes the urine concentration decreases risk of stone recurrence.

CONCLUSION

We conclude that the dietary components influence the biochemical parameters such as oxalate, uric acid, calcium and sodium etc. Accordingly, quantitative as well as qualitative dietary modifications especially for oxalate, animal protein, and minerals may play an important role in reducing the likelihood of recurrent stone formation.

As in North Maharashtra region diet contains tomatoes, spinach, animal protein etc. use of more salt in dietary preparations and bore well water for drinking purpose. Thus these factors may serves as risk factors in addition to other risk factors for stone formation in the North Maharashtra region, and that the dietary advice to use a mixed cereal diet and not wheat alone as a staple food would be useful.

The normal calcium, low protein, low salt & low oxalate diet decreases urinary excretion of both calcium and oxalate which in combination with an increase in urinary volume causes a marked reduction in the calcium oxalate molar product and in the relative calcium oxalate saturation.

Dietary modifications could play an important part in the management of stone disease in the region. Keeping in perspective the social and cultural environment of the stone formers. The dietary modifications should be advised in the long term.

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