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PREVENTION AND MEDICAL MANAGEMENT OF UROLITHIASIS - A REVIEW

General Surgery

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ABSTRACT

Urinary stones are polycrystalline concretions of biominerals occurring in the urinary tract of humans and animals.Their formation is governed by pathoanatomical and physicochemical factors . Around 97% of urinary stones are found in the kidneys and ureters , the remaining 3% in the urinary bladder and urethra .Between 1to15% people globally are affected at some point by urolithiasis in their life time.They occur in one in 11 people at some time in their lifetimes with men affected 2 to 1 over women. Development of the stones is related to decreased urine volume or increased excretion of stone-forming components such as calcium, oxalate, uric acid, cystine, xanthine, and phosphate. Calculi may also be caused by low urinary citrate levels or excessive urinary acidity.Renal calculi present with excruciating pain and most patients present to the emergency department in agony..Nephrolithiasis is a highly prevalent condition with a high recurrence rate that has a large impact on the quality of life of those affected. The recurrence rate ranges from 21% to 53% after 3–5 years. Calcium oxalate (75%–90%) is the most frequent component of calculi, followed by uric acid (5%−20%), calcium phosphate (6%−13%), struvite (2%−15%), apatite (1%) and cystine (0.5%−1%). The incidence of urolithiasis reaches its peak in population aged over 30 years.It also poses a great financial burden on society. There have been great advancements in the surgical treatment of stone disease over the past several decades. The evolution of surgical technique appears to have overshadowed the importance of prevention of stone disease, despite evidence showing medical therapies significantly decreasing stone recurrence rates.

KEYWORDS

nephrolithiasis, kidney, calculi, prevention, medical treament

INTRODUCTION

Nephrolithiasis is one of the most prevalent urologic diseases in world. The worldwide prevalence, incidence and composition of calculi varies and have changed in the last several decades, with prevalence ranging from 7% to 13% in North America, 5%–9% in Europe, and 1%–5% in Asia . The differences among countries reflect several lithogenic factors, including age, gender, dietary habits, fluid intake, climate, occupation and education level, socioeconomic status, racial or national distribution, genetic and metabolic disease. There is high rate of recurrence of urinary calculi, that's why Frère Jacques, that famous lithotomist of the Middle Ages, used to say, 'I have removed the stone, but God will cure the patient'.

Epidemiology in India

Approximate 2 million people in India is affected with nephrolithiasis every year and some parts of country has name denoted as a stone belt that is, Gujarat, Maharashtra, Punjab, Rajasthan, Delhi, Haryana and part of states on North East side. Urinary stone are also found in south India due to high intake of tamarind in regular diet. In upper urinary tract urolithiasis is found mainly in the form of pure calcium oxalate crystals as observed in case studies of AIIMS, New Delhi. The high Kutchha and Saurashtra region of Gujarat also has higher prevalence of renal calculi. In India, approximate 50% of the population is affected with renal calculi, which may end up to renal damage or loss of kidney function. The rate of nephrolithiasis incidence, mainly staghorn calculi is very high in Manipur and some reports indicates that North Western region of India have also increased prevalence. North Eastern part of Bihar in Purnia division showed increase renal stone cases during 1999-2001.In Bhagal pur region of Bihar,there is also high incidence of urolithiasis. In the last few decades occurrence of pediatric renal calculi cases were observed in some epidemiological studies.

Recent decades have witnessed great advances in the surgical treatment of kidney stones. Whereas stones were once treated with invasive open surgical techniques, in the present day almost all urinary calculi are treated either in a completely noninvasive fashion with shockwave lithotripsy or in a minimally invasive endoscopic fashion with ureteroscopy or percutaneous nephrolithotomy

[Pearle et al. 2005]. While there have been great advancements in the surgical treatment of stone forming patients, efforts to prevent the formation of kidney stones with medical therapy have not experienced the same rates of advance. Such a failure is disappointing, as medical therapies have been well demonstrated to significantly decrease stone recurrence rates [Lotan et al. 2005].

Aim and objective of study

Review of Steps for preventing formation of urolithiasis ,its medical management and prevention of its recurrence .

Risk factors

Risk factors associated with the formation of urinary calculi can be divided into two main groups, intrinsic or extrinsic factors. The former one includes age, gender, ethnic and familial backgrounds; while the latter group consists of climate and environment, lifestyle and dietary habits, occupation and education level. The most important factors, determining the prevalence, incidence, recurrence rates and constituent of calculi, are climate and dietary habits.

Age

Overall, the incidence of urinary tract stones increased with age, which peaked in the age group of 30–60 years and decreased afterwards . The reason why middle-age population are prone to urolithiasis is because they do more laborious work than others , and subsequently result in less fluid intake and higher rate of dehydration. Besides, they led an unhealthy lifestyle (irregular diet or staying up late) and suffer from occupational stress . Apart from the aging population, western dietary habits and changes of endocrine hormones with age may also be the cause of the upward trend.

Gender

In most countries, males are predisposed to urolithiasis, with male to female ratio ranging from 1.3 to 5 . It may be associated with different dietary habits . Men are more likely to have excessive alcohol and coffee, and consumed more meat than women. In addition, testosterone can promote stone formation, while estrogen appears to inhibit the formation of stones by regulating the synthesis of 1,25-dihydroxy-vitamin D . Additionally, the anatomical differences, with which men are more likely to suffer from posterior urethral valve, benign prostatic hyperplasia and stricture urethra, subsequent obstruction of urethra, can also be a risk factor for bladder stone formation .

Lifestyle and dietary habits

Lifestyle also plays a profound role in stone formation. In developing countries, like India , people fed on cereals and vegetables, which are

high in oxalate and its precursors. In addition, westernized diet, which contains excessive protein, lipid, calcium and sodium, also contributed to stone formation, leading to the rising trend of urolithiasis in many Asian countries, like Japan, China, India, Iran and SaudiArabia . Precursors of oxalate in food (like glycine, hydroxyproline, hydroxyacetic acid and vitamin C) may be metabolized to oxalate in liver, and subsequently increase the oxalate concentration in urine. The combination of lipid and calcium in intestine, forming insoluble substance, can also inhibit calcium absorption, followed by increasing absorption of oxalate . Hyperuricemia, the main risk factor for uric acid stones, can also be the result of excessive consumption of meat .

Besides, there must be another factor, appropriate urine pH, prompting the precipitation of crystals. Urinary tract stones can exist only if two main conditions of urine, ingredients concentration and pH, are fulfilled at the same time . In Southwest and South Asia, rice is the principle food, carbohydrate of which is catabolized to provide the acidic environment of urine . Excessive meat consumption can also lead to acidification of urine , which is in favor of calcium oxalate stone formation. It is typical in Japan, where a rising trend of meat consumption, metabolic syndrome and prevalence of urolithiasis have occurred at the same time

Less fluid intake is a main risk factor for urolithiasis . High levels of fluoride, sodium, calcium, magnesium and phosphates in drinking water are also reported to be associated with calcium oxalate stones. Prevalence of urolithiasis among people drinking high-fluorine water is 4.6 times higher than those in fluoride-free area . The mechanism may be that fluoride in intestine indirectly promotes the absorption of oxalate due to calcium fluoride formation, therefore, calcium availability drops . As a result, increasing of oxalate excretion and insoluble calcium fluoride formation in urine, as well as oxidative stress in renal system are witnessed. In addition, excessive sodium may lead to extra calcium absorption into blood or inhibit calcium absorption from urine into renal tubular epithelial cell, followed by enhanced calcium excretion and precipitation in kidney. Another theory is that high urine sodium may inhibit the citrate excretion in urine, which is crucial in preventing crystal formation. High concentration of magnesium in drinking water and soil may also accelerate development of stones . In contrast, Chandrajith et al. have not found any relationship between water hardness and urolithiasis.

Habits, like smoking and high alcohol consumption, can also contribute to stone occurrence . Alcohol and its metabolites can lead to oxidative stress in .kidney tissues, hypercalciuria, and hyperoxaluria, followed by stone formation, especially uric acid stones . However, this idea is not confirmed by other studies . A meta-analysis reveals alcohol consumption may decrease the risk of urolithiasis . Inadequate physical activity, another risk factor, is also implicated by the fact that people with sedentary work in office are more prone to suffer from urolithiasis . In addition, the lack of microelements, such as molybdenum and silicon that play a key role in keeping crystals in solution, may lead to stone formation as well.

Climate or living environment

Climatic and geographical factors are both correlated with urolithiasis. Specifically, temperature, seasons, sunshine hours, humidity, atmospheric pressure and rainfall are included . Countries or regions in tropical and sub-tropical areas have higher prevalence of urolithiasis than that in temperate and frigid zones (5%–10% vs. 1%–5%). Hot dry climate, which is typical in West Asia, can accelerate evaporation of body water from skin, and subsequently results in concentrated urine, which is a risk factor for precipitation of crystals and stone development. In summer and autumn, the prevalence and incidence of urolithiasis are higher than that in spring and winter in many countries or regions, such as India, Pakistan, Saudi Arabia .

Occupation or education level

The risk of urolithiasis in people working outdoors or exposed to high temperatures, such as workers in steel industry, flight attendants, farmers, miners, quarrymen or drivers are twice likely to suffer urolithiasis than people working at room temperature. Tanthanuch et al. and Hussein et al. also find kidney stone patients are more likely to be physical workers with lower education level in Thailand and Malaysia, respectively. The reason for it is that hot temperature may lead to dehydration, and people in these conditions have less access to drinking water. Besides, excessive exposure to sunshine results in more production of vitamin D. After conversion to 1,25-dihydroxy-vitamin D in kidneys, it can promote calcium absorption in the gut .

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Socioeconomic status

Socioeconomic level is also an important factor in epidemiology of urolithiasis. It is also common in higher society.The reason for it may be that chronic metabolic disease, mainly caused by high-calorie intake, is more common among population in the society with higher standard of living .

Racial or national distribution

Whether race and nationality are associated with urolithiasis is still controversial. Generally speaking, dietary habits and gene of various races or nationalities differ from each other, which may be the reason why some studies find statistical differences, while others do not.

Genetics

Cystine stones are caused by mutations in the SLC3A1, SLC7A9 gene or other neighboring genes . In addition, mutation of SLC22A12 and SLC2A9 has also been reported in patients with uric acid calculi . Calcium oxalate stones, the most common type of urinary calculi, are found to be the result of deficiency of enzymes, like alanine glyoxylate aminotransferase (AGT), glyoxylate reductate/hydroxypyruvate reductase (GRHPR) or 4-hydroxy-2-oxoglutarrate aldolase (HOGA1) . Subsequently, synthesis and excretion of oxalate are increased, resulting in calcium oxalate stone formation.

Other risk factors

Apart from urinary tract infection or renal tubular acidosis, patients with urolithiasis are frequently documented to be with metabolic diseases, like diabetes mellitus, hypertension or adiposity, which may be another risk factor for urinary tract stones . Besides, low urine output, hypercalciuria, hyperoxaluria, hyperuricosuria, and hypocitraturia are also reported to be associated with stone formation , though the reason why chemical composition of urine is abnormal is still unclear.

Medical therapy of stone formers

Medical therapy has been demonstrated to significantly decrease stone recurrence rates and may be cost effective as well [Lotan et al. 2005].

There are general recommendations that all stone formers should follow, regardless of diagnosis, to decrease stone recurrence rate. Stone formers should drink enough fluid to maintain a urine output of 2 liters per day and in case of cysteine stones 3 liters per day. A low urine volume is among the most common metabolic abnormality found in stone formers and this is a modifiable risk factor [Porena et al. 2007].

PHARMACOLOGIC INTERVENTION

THIAZIDES

The rationale for the treatment of calcium stone formers with thiazide diuretics is that this class of drugs reduces urinary calcium directly, by enhancing calcium reabsorption in the distal renal tubule, and indirectly by stimulating sodium-dependent calcium reabsorption in the proximal tubule by way of extracellular volume depletion.

The recommended dosages of commonly used thiazide

diuretics for an average sized adult are hydrochlorothiazide (25-50 mg twice daily), benzoflumethazide (2.5 mg twice daily) and trichlormethiazide (2-4 mg daily). Alternatively, the nonthiazide diuretics Indapamide (1.25-2.5 mg daily) and Chlorthalidone (25-50 mg daily) have similar mechanisms of action to thiazides. Thiazide use may be limited by side effects in up to 30-50% of patients, including fatigue, light-headedness, hypotension, erectile dysfunction and gastro-intestinal effects. In addition, thiazide-induced hypokalemia,

glucose intolerance, hyperuricemia and dyslipidemia have been reported. The degree of hypokalemia is dose-dependent and more pronounced with longer-acting thiazides.

The intra-cellular acidosis associated with thiazide induced hypokalemia promotes citrate reabsorption in the proximal renal tubule, leading to hypocitraturia which can offset the beneficial effect of thiazides.

As such, potassium supplementation is recommended in patients taking thiazides. Hence potassium citrate ,which gives potassium and citrate both is more useful in such condition.

CITRATE

Citrate inhibits calcium stone formation by several mechanisms. First, citrate complexes with calcium, thereby reducing urinary saturation of calcium salts. In addition, citrate directly prevents or inhibits the process of crystallization, growth and/or aggregation of calcium oxalate and calcium phosphate. Compelling evidence justifying the use of citrate as a therapeutic or prophylactic agent in the management of calcium oxalate urolithiasis abounds in the literature.The 3 preparations that have been best studied are potassium citrate, sodium potassium citrate and potassium magnesium citrate.

There is high level (1b) evidence in support of potassium

citrate therapy for the prevention of recurrent calcium stones. Potassium citrate is administered in starting doses of 40-60 mEq daily in divided doses. Potassium citrate can be used for treatment of patients with hypocitraturia or for normocalciuric calcium stone formers regardless of other associated abnormalities.

In patients with hypercalciuria treated with thiazides, potassium citrate is additionally prescribed to prevent thiazide-induced hypokalemia and hypocitraturia. For patients with enteric hyperoxaluria, potassium citrate can raise urine pH and citrate.

However, dosages higher than those used for idiopathic calcium nephrolithiasis (up to 120 mEq daily) may be required. Because of the rapid intestinal transit associated with chronic diarrhoeal syndromes,

a liquid form of potassium citrate may be better absorbed.

Potassium magnesium citrate was developed to combine the beneficial effects of alkali citrate with the additional inhibitory action of magnesium.Potassium magnesium citrate was shown to have a greater citraturic response than either potassium citrate or magnesium citrate in healthy subjects.

ALLOPURINOL

Allopurinol is a xanthine oxidase inhibitor that prevents the conversion of hypoxanthine to xanthine, the precursor of uric acid, thereby reducing uric acid production and urinary uric acid excretion. Uric acid promotes calcium oxalate stone formation by increasing urinary saturation of monosodium urate, a promoter of calcium oxalate crystallization through heterologous nucleation. At pH<5.5, the sparingly soluble undissociated form of uric acid predominates leading to uric acid crystallization and uric acid and/or calcium oxalate stone formation.

At pH>5.5, uric acid promotes calcium oxalate stone formation by way of monosodiumurate-induced calcium oxalate crystallization.

In addition, uric acid reduces the effectiveness of naturally occurring inhibitors of calcium oxalate crystallization.

The rationale for the use of allopurinol for the prevention of calcium oxalate stones is that it reduces serum and urinary uric acid levels, thereby preventing calcium oxalate crystallization.

Dosage of allopurinol is 300 mg once daily., Although the drug is generally well tolerated, rare side effects include reversible elevation of liver enzymes and occurrence of a rash that can progress to Stevens-Johnson syndrome. Either of these side effects should prompt immediate discontinuation of the drug.

MAGNESIUM

Several lines of evidence support the use of magnesium in preventing calcium stones. First, magnesium decreases the crystallization of calcium oxalate by complexing with oxalate ions to form soluble magnesium oxalate, thereby reducing the amount of free oxalate ions in the urine.

Second, in vitro crystallization experiments demonstrated that it inhibits calcium oxalate nucleation, growth and aggregation. In addition, magnesium oxalate is more soluble than calcium oxalate. Some studies have also reported lower levels of urinary magnesium in stone formers compared with healthy controls.

Finally, magnesium supplementation has been shown recently to reduce intestinal oxalate absorption.

SODIUM

A high salt intake has been associated with an increased risk of stone formation through a variety of effects. First it decreases renal calcium reabsorption and increases calcium excretion. It also reduces urinary citrate resulting in reduced inhibitory activity against the crystallization of stone-forming calcium salts. Finally it increases monosodium urate formation which promotes calcium oxalate crystallization. Curhan and colleagues confirmed a positive correlation between sodium consumption and risk of first-time stone formation in women but not in men. No prospective clinical trials have investigated the role of sodium restriction as an independent variable in reducing the risk of stone formation, although the Borghitrial found a lower rate of recurrent stones in men adhering to a normal calcium, low animal protein, low sodium (50 mmol/day) diet than men on a low calcium diet. Calcium stone formers should be advised to avoid adding salt to foods and to limit their intake of sodium to 2000 to 3000 mg daily. For hypercalciuric stone formers taking thiazide diuretics, sodium restriction is particularly critical as high urinary sodium attenuates the hypocalciuric action of these drugs.

Amino acids are freely filtered by the glomerulus and reabsorbed in the proximal tubule. Proximal tubule reabsorption of sodium and water provide the energy and chemical driving force for the reabsorption of cystine. High dietary intake of sodium, as assessed by urine sodium excretion, is associated with increased urine cystine excretion.

Low sodium diets, which lead to slight volume depletion and subsequent increased proximal tubule reabsorption of sodium, have been used to lower urine cystine excretion.

Cystinuric patients are routinely instructed to increase fluid intake and maintain a high urine flow rate in order to lower urine cystine saturation.

Drug therapy for cysteine stone

Thiol-binding drugs

Tiopronin, d-penicillamine, and captopril are drugs that contain thiol groups. When present in solution with cystine, a disulfide exchange reaction occurs and a drug-cysteine complex forms. The drug-cysteine complexes for all three of these thiol-binding drugs are much more soluble than cystine, effectively increasing cystine solubility. These medications have been shown to lower urine cystine saturation when studied in vitro and clinically.

However, the significant level of side effects of the drugs generally restricts their use to patients who are unable to control stone formation with high fluid intake, dietary modification and urine alkalinization.

Common side effects include nausea, rash, fatigue, fever, and proteinuria. The side effects appear to be dose related and tiopronin is reported to have lower rates of side effects than d-penicillamine.

Captopril is generally better tolerated than tiopronin and dpenicillamine, but that is likely due to the much lower dose used, 150 mg per day (0.7 mmol/day) versus 1000 to 2000 mg/day (6 to 12 mmol/day) for thiola and d-penicillamine.captopril should be considered the preferred thiol binding drug, in the patient who has hypertension also.

Vitamin B6 –high doses of vitamin B6(40mg/day) decreases oxalate production where as vitamin C can be metabolized to oxalate.The relation between the intake of vitamin B6 and C and the risk of symptomatic kidney stone were studied in a cohort study in women with no history of kidney stone by G C Curhan et al J Am Soc Nephrol.1999Apr.Large doses of B6 may reduce the risk of kidney stone formation in women.Routine restriction of vitamin C to prevent stone formation appears unwarranted

Summary of prevention and treatment

Moderate animal protein intake is recommended, as purine gluttony is a known risk factor for uric acid stone formation.

A strict low-salt diet is also advised, as elevated sodium excretion in the urine can induce or exacerbate hypercalciuria. Increased sodium excretion can also blunt effectiveness of a thiazide-type medication used to treat hypercalciuria

Lastly, intake of a normal recommended daily allowance of calcium

(1200mg per day) is advised. A common misconception among stone formers is that a low calcium diet is a treatment for calcium stone disease. However, such a diet can actually increase stone risk, as demonstrated in a randomized, controlled trial performed by Borghi and colleagues [Borghi et al. 2002)

In addition to these generic recommendations for all stone formers, recommendations are based on the results of the workup discussed. Treatment of the diagnosed underlying etiology of the stone disease should be pursued.

Hyperparathyroidism causes resorptive hypercalciuria as elevated levels of parathyroid hormone stimulates uptake of calcium from the gut.

Primary hyperparathyroidism is usually treated surgically. Idiopathic hypercalciuria can be treated with a thiazide-type medication [Lipkin andShah, 2009]. Whether the etiology of idiopathic hypercalciuria is absorptive or resorptive, thiazide medications will increase tubular reabsorption of calcium in the kidney, thereby reducing the degree of calcium in the urine. Supplemental potassium may be necessary for patients receiving thiazide therapy, as these medications may promote hypokalemia, which can induce an intracellular acidosis and hypocitraturia.

Primary hyperoxaluria is an hereditary condition that is associated with stone disease. High-dose pyridoxine, magnesium, orthophosphate, glycosaminoglycans,and hydration are all used to manage this condition. Ultimately, however,liver and kidney transplantation is required for cure [Cochat and Basmaison, 2000]. Enteric hyperoxaluria can also occur for many different reasons but mal-absorption or malnutrition are the most common causes. This can occur as a result of intestinal disease and/or chronic diarrhea. In addition, these patients often have great intestinal losses that result in low urine volume contributing an additional risk factor for kidney stone formation. The central treatment point toreduce urinary oxalate is dietary modification, hydration, calcium supplementation, magnesium,and reduction of other coexistent risk factors [Penniston and Nakada, 2009]. Cholestyramine and potassium citrate is also sometimes used.

Hyperoxaluria can also be idiopathic.

Citrate therapy is used for many metabolic abnormalities that are associated with an increased risk of stone disease. It is used to raise the urine pH in uric acid stone formers. Citrate itself is an inhibitor of stone formation and hypocitraturia is among the most common metabolic abnormalities that are discovered in stone formers [Coe and Kavlach, 1974]. Hypocitraturia is often idiopathic, although other disease states, such as distal RTA(Renal tubular acidosis), hypokalemia, chronic diarrhea,urinary tract infection, thiazide medication, and a low-alkali, high-protein diet can induce this disorder [Zuckerman and Assimos, 2009].

Again, hypocitraturia should be addressed by treating the underlying abnormality, but citrate replacement therapy is often the mainstay of therapy for this situation, such as those patients with distal RTA in which profound hypocitraturia is present. Dietary modifications with lemonade and other citrus-juice-based therapies can also help increase citrate in the urine [Zuckermanand Assimos, 2009]. One caveat to citrate therapy is to maintain vigilance for calcium phosphate stone formation, as the alkali nature of citrate medication will raise urinary pH. Finally, the presence of urease-producing bacteria, which are associated with struvite stone formation, should be addressed. Elimination of the infection is important, and it should be noted that eradication of the bacteria cannot be achieved if a stone burden is present. The central components of therapy for such patients are both surgical stone clearance as well as antimicrobial treatment of the offending bacteria

CONCLUSION

Nephrolithiasis remains life threatening disorder and cover almost all regions of the world. Exhaustive understanding of this disorder is necessary as lot of factors can induced this disease.

The main treatments for calcium stone prevention,thiazides, potassium citrate, allopurinol, high urine volume, and control of dietary sodium and protein intakes, are supported by prospective randomized trials of sufficient power to draw conclusions of merit. In addition, surgery for primary hyperparathyroidism, alkali for uric acid stones, high fluid, alkali, and thiol agents for cystinuria are obvious treatments with no formal trials. Likewise, the patchwork of remedies for enteric hyperoxaluric states for which there is fragmentary evidence, and liver transplantation for primary hyperoxaluria complete the summary of what can be offered to a patient by way of stone prevention. As a rule, all of these treatments in one way or anotheraim to reverse pathogenetic factors of stone formation,have a rationale in science and are meant to be long lasting in their effects.

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