

## **Kidney Stones**

**By Thomas A. Kruzel, ND**

Development of kidney stones is often associated with affluent cultures and annually accounts for 175,000 to half a million cases per year in the United States. About 1 of every 1000 hospital admissions is related to kidney stone disease with men afflicted more often than women usually in the third to fifth decade of life. About 1% of all autopsies reveal some form of nephrocalcinosis. [Merck] While kidney stone formation in children is considered relatively rare, they are more often associated with inborn errors of metabolism or genetic predisposition. [Kempke] In one study conducted on 32 children from India about half demonstrated an underlying disorder such as idiopathic hypercalciuria, hyperoxaluria and renal tubular acidosis, primary hyperparathyroidism and hyperuricosuria. [Hari, P. et al. ] Prematurely born infants given Furosemide are at a greater risk for development of renal stones [smith pg289]. Nephrolithiasis during pregnancy, although uncommon, occurs more frequently during the later stages of gestation resulting in a higher incidence of urinary tract infection. [Maikranz]

The incidence of bladder stones is more likely to occur in less affluent cultures which interestingly, have a lower incidence of renal stones.

### **Signs & Symptoms**

Generally two types of renal pain can accompany the onset of nephrolithiasis. Renal colic results from a stretching of the ureter while the heavy, tensive non-colicky pains are due to a distention of the renal capsule. Renal colic is almost always associated with obstruction of the ureter. These symptoms may overlap, making a differentiation difficult. Often a kidney stone "attack" is experienced by an abrupt onset of extreme flank pain, which radiates to the groin and hips. Concomitantly the person experiences nausea and vomiting, gross or microscopic hematuria which heralds the passage of a stone. Localized inflammation, edema, mucosal irritation and hyperperistalsis contribute to the pain and discomfort experienced by the patient. Stone size does not necessarily contribute to the severity of pain.

The patient often moves in response to the pain and to help afford relief from it. An absence of movement accompanied by a rigid abdomen, suggests peritoneal inflammation rather than stone passage.

Hematuria and pyuria [see lab] are present to varying degrees and may range from frank hematuria and pyuria to an aseptic urine and few symptoms. A urinary microscopic exam which is void of WBC's suggests pyonephrosis. Pyonephrosis, pus accumulation in the renal pelvis, carries with it the danger that a renocutaneous fistula may develop as the infection spreads.

The presence of a fever signifies an accompanying infection and constitutes an emergency due to the increased chance of sepsis. Tachycardia, hypotension and flushing of the skin may also accompany the condition.

Costovertebral angle tenderness may be absent or markedly tender and is not a reliable sign for the presence of a kidney stone. Kidney punch test may or may not be present for the same reasons. If the kidney is hydronephrotic it may be palpable if the obstruction is upper ureter and acute. It can not be relied upon in chronic obstruction. Elicitation of a neurological pain reflex may occur by pressing on the region over the kidneys and noting a retracting of the abdominal muscles or of the reactive leg. This is nonspecific however and may be due to other conditions as well.

The presence of nausea and vomiting usually signifies upper urinary tract obstruction as similar spinal reflex centers are stimulated by the irritation. Intravenous fluid replacement is in order if the condition is on-going and severe, and signs of dehydration and electrolyte imbalance are present. Intravenous fluid replacement should not be used to increase urinary flow however, only to achieve a euvolemic state and correct an electrolyte imbalance.

During an acute passage of a kidney stone the body will initiate several defense mechanisms. There will often be an increase in the thirst mechanism as the body attempts to flush out the stone. Secondly, there will be an increase in frequency of urination for the same reasons, and thirdly, the body will attempt to change the urinary pH in response to the presence of an infection. This latter mechanism may be detrimental to the condition as in the case of triple phosphate stones which thrive in an alkaline environment.

## **Stone Formation**

Kidney stones are most often composed of a mixture of calcium oxalate and calcium phosphate. Those being composed primarily of calcium phosphate suggest that the urine is primarily alkaline or that a primary hyperparathyroidism, renal tubular acidosis, alkali therapy or milk-alkali syndrome is present.

Stones measuring less than 5 mm in diameter are more likely to be passed with there being less chance of stone passage if it is greater than 7 mm. Under certain conditions renal failure can occur with nephrocalcinosis from hyperparathyroidism, renal tubular acidosis or staghorn calculus formation. Obstruction of a unilateral kidney takes place if the stone becomes lodged in the ureter or at the vesicle/ureter junction, its smallest point.

About 40% of all persons who develop a stone will suffer a recurrence within the next 5 years and an additional 40% within the following 25 years. Certain individuals who have a genetic predisposition toward developing kidney stones (i.e.. a positive family history) may develop them at frequent and varying intervals.

Kidney stone formation depends upon several factors which, when found in combination, allows for stone formation. Most importantly is an increased concentration of urinary crystalloid substances either through increased out put or low urinary volume. Urinary pH, ionic strength of the solute and complexation are required for stone nucleation and subsequent formation. A decrease in inhibitors such as magnesium, citrate, pyrophosphate and nephrocalcin [ref] or an increase in promoter substances is also needed.

Tamm-Horesfall protein (THP), a mucoprotein secreted in the kidney, acts to reduce formation of aggregates of stone complexes and is found in lower amounts in chronic stone formers. Other studies have found that THP has a dual role as a modifier of crystal aggregation. In solutions with high pH and low concentrations of calcium and THP, THP acts as a powerful inhibitor of calcium oxalate crystal aggregation. Conversely, solutions with low pH and high concentrations of calcium and THP, favor self-aggregation of THP molecules which lowers their inhibitory activity against calcium oxalate crystal aggregation. [Hess, B.]

Several theories of stone formation have been proposed such as the stone matrix, nucleation and crystal inhibitor theories. Each seems to provide reasonable scenario's for

stone formation, but exceptions to all of them can be found. Generally a combination of all of them is felt to be part of stone formation. Nucleation generally begins on the epithelium once the solubility product constant [ $K_{sp}$ ] of the substance has been reached. Stone growth then proceeds either by growth of the original nidus or attachment of aggregates formed in other locations. Anatomical conditions such as polycystic kidneys, horseshoe kidney and medullary sponge kidney increase the risk of stone formation.

### **Types of Stones**

**Calcium oxalate/phosphate stones** are the most common variety accounting for about 75% of all episodes. The chemical composition will vary slightly from occurrence to occurrence but generally has a matrix comprised of calcium, phosphate and oxalate. These are always seen in an acid urine (pH less than 6.5).

**Struvite stones** (Ammonium-Magnesium-Phosphate) occur about 15% of the time, are primarily found in women and may frequently reoccur. Their formation is associated with chronic urinary tract infections. Urea splitting microorganisms such as Klebsiella, Mycoplasma, Proteus, Pseudomonas, Providencia, and Staphylococci increase the urine pH to greater than 6.8 leading to stone formation. The danger with Struvite stone formation is that they can form and enlarge rapidly resulting in obstruction of the renal calyx.

**Uric acid** stone formation (5% to 10%) is usually found in males and is associated with a chronically low urinary pH (less than 5.5). Increases in serum uric acid levels do not usually accompany stone formation as does the presence of gout. Higher incidences are seen in patients with myeloproliferative disorders or cancer patients on cytotoxic drugs. Uric acid stones are not visible on x-ray due to the lack of calcium in the matrix.

**Cystine** stone formation (1% to 2%) is due to an inborn error of metabolism resulting in excess intestinal and renal tubule absorption. High pH levels are needed (greater than 7.5) in order for cystine stones to form, with the process being independent of crystal inhibitors. Despite treatment, high recurrence rates are often seen.

**Xanthine** stone formation is associated with a deficiency of xanthine oxidase, occurring in about 25% of persons with this deficit. While higher levels of xanthine are found in the urine of patients on allopurinol for the treatment of gout, xanthine stone formation has not been reported.

## **Differential Diagnosis of Nephrolithiasis**

Kidney stones confined to the renal pelvis will radiate to the flank and the upper anterior abdominal quadrant which may mimic the pain of cholecystitis if on the right and gastritis, pancreatitis or colitis if on the left. Upper to mid-ureter stones may also be confused for an acute appendicitis or diverticulitis as they radiate towards the inguinal canal or lower abdominal quadrant. Distal ureter stone pain radiates into the ilioinguinal region and testis in males and labia majora in females. A consideration of testicular torsion, epididymitis, torsion of the ovary, salpingitis or a ruptured ovarian cyst must be part of the differential diagnosis.

### **RISK Factors:**

A low urinary output does not necessarily predispose the patient to stone formation as the ability of the kidney to concentrate as well as an availability of solutes also contributes. Some studies suggest that the incidence of calcium stone formation rises if the total urinary output is less than 1500 ml/day.

Urinary calcium levels greater than 300 mg/day in males and greater than 250 mg/day in females (or greater than 4 mg/kg) is considered abnormal and a predisposing factor to stone formation. Ideally less than 200 mg/day is considered normal on a dietary intake of 600 to 800 mg/day.[Jacobs et al.] A number of conditions predispose the person to excess calcium excretion such as renal tubular acidosis, sarcoidosis, primary hyperparathyroidism and various familial hypercalcuric syndromes. Primary hyperparathyroidism is more often found in middle-aged women or older. A determination of hyperparathyroidism need not be considered unless the serum calcium level is consistently high, the serum calcium/phosphorus level is less than 1.5 and the calcium X phosphorus value is greater than 50.

Urinary excretion of calcium and oxalate in children varies throughout the first few years of life then stabilizes by about age 6 years. In children who are stone formers, higher amounts of calcium are excreted relative to oxalate and restriction of calcium intake helps to correct the disproportion. More recently normal values in pediatric patients based on urinary calcium/creatinine (Ca/Cr) and oxalate/creatinine (Ox/Cr) ratios has been established in order to monitor dietary intakes better. [Reusz, G. S. et al.]

Idiopathic hypercalcuria is usually found in men in their 40's to 60's and often associated with hypertension, obesity and familial tendencies. A correlation with possible affluence has been noted. Dietary sodium and high animal protein intake has been known to contribute to excess calcium excretion and contribute to idiopathic hypercalcuria.

An increased dietary intake of protein has also been shown to contribute to nephrolithiasis. However, the type of protein has not been determined and a restricting of protein intake does not seem to decrease stone formation. (protein @ 60 to 70 gms/d) Dietary calcium intake has also been shown to contribute to an increase in stone formation. Diets moderate in calcium intake ( 2 to 3 servings/day) have been proposed but no specific guidelines as to amounts have emerged.

In one study 124 out of 282 patients with calcium oxalate stones were found to be hypercalcuric on either their normal diet or one containing 1,000 mg of calcium. About half who were hypercalcuric on their normal diets exhibited a calcium excretion that fell markedly or normalized on the high calcium diet. An analysis of other factors suggested that dietary sodium was at least as important as dietary calcium and more important than other risk factors for calcium excretion. [Burtis, W. et al.] This study suggests that in at least some incidences calcium supplementation may help lower excretion levels. While the mechanism is unknown, a tie in with parathyroid levels, bone loss and other minerals should be examined.

Vitamin D seems to play an essential role in the pathogenesis of idiopathic hypercalcuria in part via intestinal hyperabsorption of calcium. Hyperabsorption of calcium, is thought to also enhance the intestinal uptake of free oxalate, thus leading to hyperoxaluria. In one study it was noted that while the serum 1,25-dihydroxyvitamin D concentration remained in the normal range, it was higher in the hypercalcuric groups. When the same study analyzed data from the 75 stone-formers, a positive correlation between the serum concentration of 1,25-dihydroxyvitamin D and urinary calcium and urinary oxalate excretion was seen. The conclusions seem to confirm a relevant role for the vitamin D system in the pathogenesis of calcium nephrolithiasis due to increased intestinal calcium absorption, but also because of a greater intestinal absorption of oxalate, conditions leading to the occurrence of hyperoxaluria. [Giannini, S., Nobile, M. et al. ]

As noted, hyperoxaluria also contributes to kidney stone formation. Normally oxalate is excreted at between 15 to 40 mg/day and is the end product of metabolism.

Hyperoxaluria may be due to excess synthesis or excess dietary intake. Excess synthesis may be due to a congenital enzyme deficiency while excess absorption may also occur due to Crohn's disease or following ileal surgery. Magnesium supplement will help to decrease absorption of oxalate.

Citrate acts to inhibit nucleation through the formation of insoluble complexes with calcium with normal citrate excretion being approximately 300 to 900 mg/day. Higher amounts are found in premenopausal and pregnant women than in men. Idiopathic hypocitrauria is found in 10% to 40% of stone formers. Lower amounts of citrate may be associated with potassium deficiency, acidosis, renal failure, renal tubular acidosis, chronic diarrhea and chronic malabsorption. Potassium citrate can be used to treat hypocitraturia and/or renal tubular acidosis and is more useful than sodium citrate or sodium bicarbonate as excess sodium increases urinary calcium levels. Thiazide diuretics decrease urinary citrate levels possibly because of potassium depletion.

Uric acid stones occur with excessive urinary output of uric acid but may also occur with persistently acidic urines. Uric acid is much more soluble in alkaline [pH 7] than acid [pH 5] urine therefore alkalization [pH 6.5 to 7.5] of the urine becomes an effective part of the therapy. The source of the uric acid is primarily due to excesses in dietary intake. Having a low solubility constant ( $K_{sp}$ ) excess uric acid acts primarily as a nidus for nucleation and formation of uric acid stones. Dietary restriction of foods high in purines usually corrects the problem.

Renal calculi which are formed from chronic kidney infections can lead to other sequelae such as progressive renal failure, sepsis, perinephric abscess, pain and bleeding and constitute a more severe form of nephrolithiasis. Usually the stones are Struvite [ammonium-magnesium-phosphate] and apatite [calcium phosphate] and are found with Klebsiella, Proteus, Pseudomonas, Mycoplasma, Staphylococcus or Providencia species infections. These are urease splitting micro organisms which convert urea to ammonia resulting in an alkaline pH [pH 8 or greater] which results in the precipitation of Struvite-apatite stones. Any other micro organism found will be due to a secondary infection and is not the causative agent. Formation of Struvite stones resulting in "staghorn calculi" can occur quickly once the infection has taken hold and may be asymptomatic initially, especially in the elderly. About 40% of persons who form Struvite stones have an underlying metabolic abnormality which predisposes the person to infection and stone

formation. These stones tend to be larger over all than others and can pose severe problems with renal function.

Cystine stones are usually associated with an inherited disorder resulting in abnormal reabsorption of cystine, ornithine, arginine and lysine. [hospital medicine dec 1980] An abnormality of amino acid metabolism and impaired renal excretion are predisposing factors [Kempe pg 600]. Increasing the urinary out-put usually reduces the concentration of these amino acids, maintaining solubility and decreasing the formation of stones. Higher than normal incidences of cystinuria and subsequent formation of stones, are found in mentally retarded children. Shock wave lithotripsy does not seem to be as effective with cystine stones as with other types [Knorr et al].

Persons who live in the Southwestern or Southern United States are at greater risk for stone formation due to the loss of fluids through the skin and lungs. The incidence increases from July to October. Low fluid intakes or intake of diuretics such as alcohol, soft drinks with caffeine, coffee or black teas also increases the risk. In a study of 45,289 men 40 to 75 years old, who had no history of kidney stones, the rates of incident stone formation within the United States were determined by region. Controlling for other risk factors, including age and relevant nutrients, it was found that the prevalence of stone disease was significantly greater in the Southeastern United States. Using the Southeast as the comparison region, a decreased risk of having a history of kidney stones was found, with the Mid-Atlantic and Northwestern regions having the lowest incidences. The authors concluded that while the Southeast region had the highest incidence, the prevalence compared to other areas of the United States was not as high as previously thought. [Curhan, G.C. et al.]

Other factors such as persons with a family history of stone formation are at greater risk. Low magnesium intake has been linked to an increase in development of kidney stones by increasing the solubility of calcium oxalate stones.[Wunderlich, W]. Diets high in red meat and fats, [the beef and brew set], high oxalate containing foods such as chocolate, tomatoes, spinach, and peanuts also predispose to stone formation. There is some evidence that the increase in kidney stone formation in children is related to excesses of dairy product intake. In one study, recurrent calcium stone formers exhibited magnesium deficiencies in erythrocytes and in total serum magnesium. [Schmiedl, A & Schwille, P]

Males between the ages of 30 and 50 have higher rates of stone formation as do persons with some previous insult to the kidney or GU tract such as previous infections, scarring or abnormal kidney development. It has also been noted that persons who are more sedentary have a greater tendency toward stone formation as opposed to persons who exercise or are involved in active labor. Medications such as diuretics, antihypertensives and long term use of antacids which contain silica have been implicated as well.

### **Laboratory Evaluation of Chronic Stone Formation**

Evaluation of single incidence stone formers has to this point not thought to be necessary if there has been only one episode. This is because the side effects of the various treatments is low and there is a limited morbidity rate in most patients. However, in a retrospective analysis of stone formers, Parks and Coe [Parks JH Coe FL] found that the number of stones per episode were a predictor of whether or not the person would suffer further episodes. The study suggests that intervention and prevention may lessen recurrence of nephrolithiasis.

During an acute crises, or following ESWL, the urine should be strained for recovery of the stone. Evaluation of the sediment will reveal the nature of the stone matrix and help the physician to prescribe the appropriate dietary and fluid changes. With chronic stone formers further laboratory data needs to be obtained. A 24 hour urine for calcium, oxalate, citrate, uric acid, creatinine, total volume, pH, urea nitrogen and sodium should be obtained to help determine what imbalance exists. Preferably, conclusions about specific mineral excretion should be made after several samples are measured as these values will fluctuate with time, fluid and diet changes. The information provided is useful in developing long term dietary strategies.

Normal 24 hour urine creatinine 15-25 mg/kg  
Normal 24 hour calcium <200 mg/day  
Normal 24 hour citrate <320 mg/day  
Normal 24 hour cystine <200 mg/day  
Normal 24 hour oxalate <44 mg/day  
Normal 24 hour uric acid <600 mg/day

If hyperparathyroidism is suspected, or there is an underlying kidney disease, periodic serum calcium and phosphorus levels will help screen for fluctuations in the calcium and

phosphate balance. Using the following calculations will be helpful in monitoring this balance.

$$\begin{aligned} \text{Ca} / \text{PO}_4 &= 2.5 \quad (\text{N} = 2.2 \text{ to } 2.8) \\ \text{Ca} \times \text{PO}_4 &= < 40 \quad (\text{N} = < 40) \end{aligned}$$

A urinary sodium/potassium ratio on a first morning urine has been shown to be significantly and independently associated with the prevalence of urinary stone disease in males and females between the ages of 25 to 74 years of age. Additionally, the urinary sodium/creatinine ratio was positively related to urinary stone disease while the potassium/creatinine ratio was inversely related. (There is an association between high urinary sodium and high urinary calcium as well as high urinary potassium and low urinary calcium.) [Stamler, et al.]

First morning fasting urinary pH levels have been suggested to have some predictive value for the formation of nephrolithiasis. Urine pH levels greater than 6.10 were found to be higher in known stone formers which the authors felt to be abnormal. [Chafe, L., Gault, M. H.]

### **Medical Management**

Increased fluid intake will help pass most stones which are 5 mm or less in size. An initial abdominal x-ray should be obtained in order to locate the stone and measure its size. This will help in making management decisions initially as larger stones may need surgical or ESWL therapy.

ESWL therapy is most effective with stones measuring 5 mm up to 2 cm but begins to lose its effectiveness the larger they become. Struvite stones, depending upon the location and size may need surgical intervention, especially if they are causing renal impairment. While ESWL has had some success in eliminating stones there has been some concern that it has limitations in the pediatric population and that its role needs to be redefined. In a study examining ESWL's role in pediatric nephrolithiasis, surgery was ultimately needed in a large number of cases following treatment. [Losty, P. et al.] Despite the widespread clinical use of ESWL, the margin of safety for the kidney during shock wave application is largely unknown. In a study done on rabbits, dose-dependent moderate damage (subcapsular hemorrhage, interstitial hemorrhage, capsular tension and

perirenal hemorrhage) were noted in all kidneys at 24 hours following treatment. Evidence of permanent changes (some fibrosis, tubular and glomerular damage, chronic inflammatory alterations) was noted in long-term follow up, while a complete necrosis of the treated kidney was not encountered. [Karalezli, G. et al.] While ESWL is often the first choice of therapy its limitations are also well established: silent calyceal stones, calyceal diverticula stones, nephrolithiasis in horse-shoe kidneys, medullary sponge kidney, and residual fragments after ESWL. Other methods and further refinement of ESWL are currently being examined to decrease morbidity and the need for further invasive treatments. [Eisenberger F., Schmidt A.]

For smaller stones less than 5 mm , medical management is usually all that is necessary. Increasing the fluid intake and alkalizing the urine, if it is acidic, or acidifying it if it is alkaline, will help to make the stone more soluble and easier to pass. Demulcents such as Althea or Ulmus, coupled with a mild diuretic such as Galium or Zea may also be useful. An anti infective such as Uva ursi or Barosma added will help to decrease the risk of infection.

In cases where there is severe colic from passage of the stone certain homeopathic preparations can help in alleviating the pain and with passage. Dioscorea administered in drop doses along with Piscidia and Belladonna will help with pain relief. Hot packs to the affected flank is also useful for pain relief and ureteral dilation.

In cases presenting with, or the development of sepsis, blood cultures are needed to identify the organism so that appropriate antibiotic therapy can be initiated. Blood cultures taken just before a rise in temperature and/or every 3 hours for a total of 3 blood draws is recommended. Ideally this should be accomplished before antibiotic therapy is initiated [Ravel pg 227].

### **Diet & Nutrition**

Predisposing dietary factors for the development of kidney stones have long been noted. Diets high in refined sugar not only decrease an enzyme needed to inhibit stone formation, but also eliminate minerals needed to balance calcium concentrations. High oxalate foods such as chocolate, spinach, peanuts, black teas and cola drinks predispose to stone formation. Diets high in red meat consumption or high purine intake as in pork, contribute to the formation of uric acid stones. Generally, diets which are devoid of vitamins and minerals tend to create an imbalance of nutrients which are needed to

maintain the solubility of urinary waste products. Conversely, persons whom consume higher amounts of fruits and vegetables, especially green leafy vegetables, have lower incidences of nephrolithiasis.

Specific nutrients such as Vitamin B6 (Pyridoxine) has been found to be lower in stone formers. Vitamin B6 controls the endogenous production of oxalic acid, which, when coupled with excess calcium, predisposes the person to calcium oxalate stone formation. High glutamic acid levels in urine decreases the precipitation of calcium oxalate crystals. Decreases of glutamic acid levels are often associated with a deficiency of Vitamin B6. [Pizzorno, Murray] Vitamin K deficiency has been shown to affect growth of calcium oxalate stones via its interaction with glutamic acid. While not the first nutrient to consider as being deficient in chronic stone formers, it should be considered if the patient suffers from chronic steatorrhea or malabsorption syndrome. [ Dharmsathaphorn, K., et al.] Citrate has been shown to decrease formation of calcium oxalate stones and is often found to be deficient in stone formers. Additionally, Taurine has been shown to lower urinary oxalate and may be used as a preventive in chronic stone formers.

Contrary to common belief, high doses of Vitamin C have not been shown to be the cause of stones. While urinary oxalate levels have been shown to be elevated with increasing Vitamin C supplementation, the increase may be due to in vitro conversion following elimination [Wandzilak et al].

Magnesium is usually deficient in stone formers as low magnesium intake has been linked to an increase in kidney stone formation by increasing the solubility of calcium oxalate stones. With an acute onset of kidney stones increasing the magnesium intake to 2 gm/d for 5 days followed by 500 mg twice a day will help to eliminate further stone formation and may act to decrease the size of an existing one. Magnesium should be given in higher doses on a continual basis for those patients who have had more than one episode of nephrolithiasis.

Glycosaminoglycans and other semi-synthetic sulfated polysaccharides have been shown to impede urolithiasis by preventing crystal adherence, correction of abnormal oxalate flux, inhibition of crystal growth and agglomeration and prevention of renal tubule damage. [Boeve, E., et al.]

Acidifying or alkalizing the urine is also of benefit, depending upon the type of stone and urinary pH. Urinary pH levels can be manipulated by the use of juices and minerals. In particular prunes, cranberry juice or plums can be used to acidify the urine while sodium bicarbonate may be utilized to alkalize it. Sodium bicarbonate hydration of approximately 400 ml/m<sup>2</sup> every 4 hours has been recommended to maintain a constant urine alkalinity [Kempke pg 600]. Orange juice has been shown to reduce the incidences of uric acid and increased the inhibition of stone formation similar to potassium citrate. Orange juice however was shown to increase urinary oxalate while not altering calcium excretion; in contrast to potassium citrate, which decreased urinary calcium without altering urinary oxalate. As mentioned previously, the elevation in urinary oxalate due to Vitamin C may be an aberration. Overall, orange juice was felt by the authors to be beneficial in the control of calcareous and uric acid nephrolithiasis. [Wabner, C.L., Pak, C.Y.]

For what ever reasons, the human body's responds to the presence of a kidney stone by increasing the thirst mechanism, as well as urinary out put. Therefore high fluid intake is the only nutritional modification that has been universally found to be useful in all forms of nephrolithiasis. Some centers have found that the use of oligomineral waters seems to be important as they can obtain a high urine volume with a low electrolytes concentration. [Di Silverio, F., D'Angelo, A.R.] It is recommended that patients drink at least

1500 ml of water in order to achieve the beneficial effects of water therapy.

While cystine stones are relatively rare in occurrence, dietary manipulation is also useful. Avoiding foods which are high in methionine such as wheat, soy, dairy products, fish, lima and garbanzo beans, mushrooms and nuts helps to diminish cysteine concentration. A table of foods with the levels of methionine can be consulted.

### **Herbal Medicines**

Herbal medicines have long played a role in the treatment and relief of nephrolithiasis. Used singly or in combination they act to increase diuresis, relieve pain and relax muscle spasm which often accompanies passage. While many herbal medicines affect the formation of kidney stones in some way, only a few exhibit a primary action for the condition. The beneficial effects caused by many herbal infusions on urolithiasis can be attributed to a disinfectant action as well as to the presence of saponins. Some solvent

action with respect to the disrupting the formation of uric stones is primarily due to their capacity to alkalinize the urine. [Grases, F. et al.]

Prescribing herbal medicines based on specific indications rather than generalized symptoms enhances their effectiveness. Some of the more commonly used herbal medicines during an acute episode of renal stone passage are listed. It is recommended that the guidelines for administering specific medications be followed in order to achieve optimal results.

### **Homeopathic Medicines**

Homeopathic medicines have achieved a stellar record of performance in the treatment of pain and spasm accompanying an acute attack of kidney stones. When administered during the attack it is frequently found that the stone is passed soon after with a cessation of pain and spasm. It is important when considering the use of any homeopathic medicine to note the size of the stone as it may be too large to pass through the vesicle-ureter junction. If this occurs, surgical intervention may be needed in order to dislodge it. Certainly, the administration of the correct medicine will soon be followed by changes in the clinical picture over the next few hours and will probably require an additional prescription to conclude the case.

Homeopathic medicines work well with other therapies such as hot packs, herbal medicines and vitamin therapy. Some of the more common homeopathic medicines and their indications are listed.

### **Hydrotherapy**

Certainly and increased oral fluid administration is in order unless there is a blockage of the ureter or kidney. Then, surgical, lazer or shock wave intervention is in order to dislodge the stone. For the passage of smaller stones, hot packs over the affected flank helps to relax tense muscles form pain and spasm, allowing easier passage of the stone. These can be left in place for considerable periods as long as they are wet packs and are not extracting moisture from the skin. Caution should be taken in the elderly and diabetics as they are less sensitive to heat and burning of the skin may ensue.

### **Follow Up and Monitoring Outcomes**

Without intervention, follow up and preventive measures being taken, stone recurrence can reach 50% within 5 years.[pg 276 smith] With known stone formers, (i.e. greater than 2 episodes within the past year and/or a positive family history and 1 episode), periodic monitoring is in order. A routine urinalysis noting specific gravity, pH and the presence of hematuria as well as monitoring the patients diet and fluid and mineral intake should be done every 3 months. If the patient has not had a recurrence within the first year, then follow up every 6 months is sufficient.

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