From: john redden <redden@viriditasherbalproducts.com>
Subject: **Re: Herbalhall Digest, Vol 72, Issue 23**
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> Urolithiasis
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> Urinary calculi (syn. kidney stones, nephrolithiasis, urolithiasis) refers to the presence of calculi in the urinary tract, varying from microscopic crystalline granules to calculi several centimeters in diameter. Depending on their location in the UT, this condition is given different names: within the bladder, the condition can be termed vesical calculi, bladder calculi, vesical stones, or bladder stones; within the kidneys, the condition is termed nephrolithiasis, kidney calculi or kidneys stones.
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> In North America about 80% of the stones found in urolithiasis are comprised of calcium salts, as calcium oxalate or calcium phosphate. Between 5-10% are comprised of urates, another 5-10% are struvite (magnesium ammonium phosphate), and between 1-2% are comprised of cysteine. There are a variety of theories that describe the etiology and pathogenesis of urolithiasis, each depending upon the type of stone formed. (Berkow 1992)
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> The following is an overview of the different types of stones and their etiology:
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> -Urate stones are related to increased excretion of uric acid, often associated with the excessive consumption of purine-rich foods (e.g. animal proteins, legumes, coffee), gout, or from an inherited (x-linked) overactivity of phosphoribosylpyrophosphate synthetase, leading to increased uric acid production. (Smith 2005; Neiberger 2005; Berkow 1992)
> oOxalate stones are related to high oxalate intake (e.g. rhubarb, spinach, cocoa, pepper, tea), ethylene glycol poisoning, familial oxaluria, and ileojejunal diseases or surgery. (Smith 2005; Neiberger 2005; Berkow 1992)
> -Struvite stones are related to abnormal urinary alkalization from the activity of organisms that metabolize urea (e.g. Proteus, Pseudomonas, Staphylococcus, Candida), forming ammonia, which is then hydrolyzed to ammonium hydroxide, raising urine pH to 8 to 9, and causing the precipitation of magnesium ammonium phosphate. (Smith 2005; Neiberger 2005; Berkow 1992)
> -Cysteine stones are entirely related to hereditary cystinuria, characterized by the excessive excretion of cystine, which is relatively insoluble in urine, and thus precipitates in the UT. (Smith 2005; Neiberger 2005; Berkow 1992)
> -Calcium stones account for the vast majority of cases in urolithiasis, but the mechanism by which these stones is formed is unclear, and in most cases, is termed idiopathic hypercalciuria. The mechanism of action appears to be related to an increased intestinal absorption of calcium and decreased renal reabsorption. Some have speculated this may be related to excessive calcium ingestion, but this link has not been clearly established, and in some studies calcium supplementation appears to reduce the risk of calcium stones (Williams et al 2001). Excessive levels of circulating 1,25(OH)2D3 has also been suggested, but once again, the data on how this should affect vitamin D3 supplementation is unclear, except that excessive consumption could play a role. Other linkages between diet and idiopathic hypercalciuria include a low fiber diet (Ebisuno et al 1986), excessive sugar consumption (Lenmann et al 1969), and excessive salt consumption (Muldowney et al 1982). The excessive consumption of animal proteins has been suggested by preliminary trials (Hughes and Norman 1992), but contrary to expectation, one major clinical trial show that dietary protein restriction increased the risk of urolithiasis (Hiatt et al 1996). Omega 3 fatty acid deficiencies have been implicated in urolithiasis, with an increase in urinary PgE2 levels in hypercalciuric patients (Baggio et al 2000). In hyperparathyroidism there are increased levels of parathormone that acts to increase serum calcium levels, and is clearly linked to calcium stone formation, but accounts for relatively few cases.
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> Generally speaking, the underlying etiological factors for stone formation is related to an impairment in bladder emptying. This can be due to anatomical problems (e.g. prostatitis, tumors), a 'neurogenic' bladder (e.g. spinal cord injury, multiple sclerosis), the presence of foreign bodies in the UT that act as a nidus or “seed” that promotes stone formation (e.g. surgical staples, ureteral stents), or bladder inflammation (e.g. radiation, schistosomiasis). (Smith 2005; Neiberger 2005; Berkow 1992)
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> Although often asymptomatic, urinary calculi can cause pain, bleeding, urinary obstruction, and secondary infection. Renal colic frequently occurs when the calculi obstructs urination excretion into the bladder, and can be excruciating, originating in the lower back and radiating across the abdomen into the crotch and inner thigh. If calculi obstruct the bladder the pain is typically localized in the suprapubic region. Gastrointestinal symptoms such as nausea, vomiting, and abdominal distention are frequent, and as the stone is being passed additional symptoms such as chills, fever, hematuria, and frequent urination are very common. (Smith 2005; Neiberger 2005; Berkow 1992)

Medical treatment
Acute renal colic is a severely painful condition, and is treated in an emergency care setting with narcotics such as intravenous morphine and codeine, and/or nonsteroidal anti-inflammatory drugs such as ketorolac tromethamine or oral ibuprofen. Antiemetic agents such as metoclopramide HCl and prochlorperazine may also be used. (Smith 2005; Neiberger 2005; Berkow 1992)

When an infection presents in combination with an obstruction of the urinary tract there is a significant risk of the leakage of urine or toxic urine by-products and as a result, death. Such conditions are treated aggressively with intravenous antibiotic therapy. Emergency surgical procedures consist of either a ureteral stent (a small tube placed endoscopically into the entire length of the ureter from the kidney to the bladder) or a percutaneous nephrostomy (a small tube placed through the skin of the flank directly into the kidney). (Smith 2005; Neiberger 2005; Berkow 1992)

Apart from acute care, the medical treatment of urinary lithiasis consists of a variety of therapies used to break down the calculi and prevent stone formation. The most common non-invasive surgical methods consist of extracorporeal shock wave lithotripsy (ESWL), in which a series of acoustic or ultrasonic shock waves are focused from outside the body onto the kidney or bladder stones. This process breaks up large stones into particles generally smaller than 1.5 cm in diameter, which then can be passed by the patient. Ureteral stents are often placed following these procedures to prevent obstruction from ureteral spasm and edema, and can cause considerable discomfort to the patient. Where very large stones cannot be broken down by ESWL, surgeons will perform a percutaneous nephrostolithotomy after lithotripsy, in which a sheath is surgically inserted into the kidney through which an endoscope is used to remove large stones. (Smith 2005; Neiberger 2005; Berkow 1992)

Non-surgical lithotriptic interventions include a variety of therapies depending on the nature of the stone and its chemical composition. Uric acid and cystine stones often respond well to the alkalization of the urine, using sodium bicarbonate to adjust the urine pH between 6.5-7.0. For pure uric acid stones the drug allopurinol helps to reduce uric acid excretion via the kidneys, whereas drugs such as penicillamine or 2-alpha-mercaptopropionyl-glycine (2alpha-MPG) are used to inhibit cysteine stone formation. Calcium-containing urinary calculi does not respond to urinary alkalization and often requires surgery if the stones cannot be passed, and thus prophylaxis is often recommended, including lowered salt and protein intake, and increased water consumption. (Smith 2005; Neiberger 2005; Berkow 1992)

Holistic treatment
Urolithiasis has long been described in the traditional medical literature, in Ayurvedic, Chinese and Unani medicine, and as a result, a variety of therapies have been used. Acute renal colic is not a condition that should be treated by holistic methods, and patients should be referred for emergency treatment. That said, a number of treatments can be undertaken to break stones down and prevent their formation in mild to moderate, or chronic complaints. Many of these herbs have traditional names or synonyms such as “stone-breaker” or “gravel root,” and are used in all types of lithiasis:
-in the West: Gravelroot (Eupatorium purpureum), Stoneroot (Collinsonia canadensis), Yellowdock (Rumex crispus), Parsley Piert (Aphanes arvensis), Pellitory-of-the-wall (Parietaria diffusa), Hydrangea (Hydrangea arborescens), Cornsilk (Zea mays), Couchgrass (Agropyrion repens)
-in India: Gokshura (Tribulus terrestris fruit), Agnimantha (Premna integrifolia root), Pashanabheda (Bergenia ligulata root), Kantakari (Solanum xanthocarpum fruit), Varuna (Crataeva nurvala bark), Shilajitu, Manjishta (Rubia cordifolia root), Ela (Eletarria cardamomum fruit), Guggulu (Commiphoa mukul resin), Manjishta (Rubia cordifolia root), Bibhitaki (Terminalia belerica fruit)
-in China: Jin Qian Cao (Desmodium styracifolium, Lysimachia christinae), Ze Xie (Alisma plantago-aquatica), Bian Xu (Polygonum aviculare), Jin Sha Ten (Lygodium japonicum), Qu Mai (Dianthus superbus), Shi Wei (Pyrrosia lingua), Yu Mi Xu (Zea mays), Ji Nei Jin (chicken gizzard)

Magnesium deficiencies have been linked to calcium stone formation, with a disruption in the normal Ca:Mg ratio, and is an independent risk factor for stone formation (Johansson et al 1982). Pyridoxine (vitamin B6) has been shown to inhibit the production and urinary excretion of oxalates (Gershoff and Prien 1967), and works synergistically with magnesium (Lyons et al 1966). Vitamin K, found most abundantly in leafy green vegetables, appears to be important inhibitor of calcium oxalate monohydrate formation (Nakagawa et al 1983), and is perhaps why a lower incidence of urinary calculi is found in vegetarians (Robertson et al 1982).

Calcium citrate supplements should be avoided in high doses and in patients with a history of urinary lithiasis due to an increase in urinary calcium levels. Other forms of citrate however may be beneficial (i.e. magnesium citrate), as citrate taken alone has been shown in clinical trials to reduce the urinary saturation of calcium oxalate and calcium phosphate, and inhibit stone formation (Whalley 1996).

The holistic treatment of urolithiasis is as follows:

1. Dietary changes.
-increase soluble and insoluble fibers in diet, e.g. leafy green vegetables, rice and oat bran
-eliminate sugar
-reduce salt, alcohol, tea, coffee and dairy consumption
-reduce excessive purine-containing foods (e.g. shellfish, shrimp, pork, beef, etc.)
-reduce oxalate-containing foods (e.g. rhubarb, black tea, spinach, parsley)
-increase total water intake
-increase magnesium-containing foods (e.g. barley, sesame, coconut, avocado)
-specific foods: red adzuki beans, chicken gizzard

2. Herbal therapies.
-lithotriptic botanicals: e.g. Eupatorium, Aphanes, Zea, Agropyrion, Tribulus, Bergenia, Crataeva, Rubia Terminalia belerica, Desmodium, Lysimachia, Alisma plantago-aquatic, Polygonum, Lygodium, Shilajit
-demulcents: Althaea, Ulmus, Plantago, Calendula, Symphytum, Bambusa (manna), talc
-antispasmodics: Dioscorea, Lobelia, Hyocyamus
-antimicrobials (in struvite stones and concurrent infection): Arctostaphylos, Chimaphila, Barosma, Echinacea, Hydrastis, Tribulus, Commiphora, Crataeva, Tinospora, Phellodendron, Artemisia, Piper methysticum, Rubia, Santalum, Azadirachta, Lysimachia, Polygonum aviculare, Coptis
-alkalizing diuretics: Urtica, Taraxacum folia, Galium, Apium

3. Supplements
-synbotics: 6-8 billion bacteria (e.g. a mixture of Lactobacillus acidophilus, Bacillus bifidum and Staphylococcus faecium), thrice daily, with meals
-EPA/DHA, 1000 mg each daily
-magnesium citrate (in hypercalciuria), 600 mg daily
-calcium (in hypercalciuria; not in citrate form), 300-600 mg daily
-vitamin B6 (in hypercalciuria), 50 mg daily (taken with a B-complex)
-vitamin K (in hypercalciuria), 2 mg daily
-folic acid (in hyperuricosuria), 5 mg daily
-sodium bicarbonate (in hyperuricosuria, monitor urine pH; avoid in struvite calculi), 1/2-1 tsp 3-4 times daily