Summer here in coastal Southern California is nothing like where I grew up on the east coast. In June and (even this) July in Newport Beach, we are lucky to have a few hours of sunlight in the late afternoon, barely warming the air into the 70’s.

When I become nostalgic about my summers gone by, I think of intense heat, sweating, the beach, transient relief by thunderstorms, and yes, trips to Fenway Park to watch hardball. With this in mind--and the MLB All-Star Game coming back to Anaheim this week, I thought I would share my thoughts on a “human” hardball, of sorts, that is, kidney stones. After all, summer and dehydration drive more patients to ER’s and urologists’ offices with these maladies, which probably affect 5-10% of Americans during their lifetime.

A prior MEEditorial (May 2009) addressed the issue of kidney stones (urolithiasis) more from a clinical perspective, including typical symptoms and recommended treatments. I want to focus on the cause of urinary stones. Fact is, as is typical in many avenues of medicine, there is more we don’t than we do know in this regard.

The demographics: men have 2-3 x the incidence of stones compared to women. Caucasians lead other racial groups in this infamous distinction. Hot/humid climates produce more stones. The incidence of kidney stone disease has been slowly increasing, but theories as to “why” vary.

In my judgment, there has to be a predisposition to getting urinary stones. It cannot be diet or low fluid intake alone. Although there are specific known genetic and hereditary causes for urolithiasis, they comprise a small minority of patients. These include disorders such as renal tubular acidosis, hyperoxaluria and cystinuria. Urologists suspect there may be some more powerful (as yet unidentified) single or poly-genetic mutation that places one at risk for urolithiasis-- and that proneness may be transferred into an actuality by dietary
and lifestyle choices. How the genetics yields causation is speculative-- it could have to do with hyperabsorption of certain nutrients in the diet, the loss of beneficial substances in excess via the kidney’s filtration system, lack of excretion of inhibitory chemicals into the urine, or some kind of damage to the macroscopic and microscopic parts of the kidney’s filtration or excretion systems. Unproven theories have included poor blood flow to the kidney’s papillae with deposition of small calcium deposits (forming tiny plaques @ and below the lining of the inside of the kidney) onto which stone crystals can gravitate like a magnate.

Two people can have the exact same diet; one never has urolithiasis, the other gets several stone attacks a year.

We can identify several chemical factors in patients who ARE prone to stones including excess amounts of calcium excreted into the urine, hyperabsorption of oxalates (found in certain foods like beets, spinach, rhubarb, chocolate, cola beverages) from the small intestine and low urinary volume. Much higher concentrations of calcium mated to oxalate can occur in human urine than, for example, in water.

Not knowing exactly what causes kidney stones makes it such that advice on dietary prevention is often aimed only at those who already have suffered. Most stones are made of calcium oxalate (in a monohydrate or dihydrate form). Less common calcium phosphate/carbonate or so-called triple phosphate (“infection”) stones; as well as non-calcified uric acid stones follow in prevalence.

I think the following advice is sound to those who have experienced urolithiasis. First, let’s assess by CT or other x-rays if you still have stones in the kidneys and whether they are worthy of treatment, e.g., if left alone, what is the worst that could happen? I worry more about the patient who has passed a stone or had one removed—and still has 4-5 more stones in each kidney. A high fluid intake helps. Water is great. [There is not a lot of evidence that stone formation is higher in municipalities have a “hard’ water supply.] Since citric acid is one of the best nonspecific inhibitors of the formation of stone crystal s in the urine, a
generous intake of fluids with a high citrate/oxalate ratio is quite important. Lemonade seems to fit the bill, although orange juice isn’t bad. Stone formers should drink three full glasses of, e.g., lemonade, daily. How much overall fluid you should drink depends on a lot of factors such as body size, metabolism, activity, and sweat. A good guideline is to drink enough fluid so that your urine ALWAYS appears more a clear than yellow color.

In terms of diet, we discussed low oxalates in the diet. The pendulum for limiting calcium in the diet swung away from calcium restriction for quite some time--but now, it appears academic urologic studies suggest that patients with calcium stones SHOULD be restricting not only oxalate but also calcium intake. There is also ample evidence that a low salt diet (low sodium) helps prevent urolithiasis, possibly because sodium drags calcium into the urine with it.

I also tell my stone patients to have a passed or removed stone analyzed—this information is probably better than blood tests or 24-hour collections of urine at assessing risk for stones and for giving advice as to further treatment and/or prevention.

I suspect urinary “hardballs" will always be a fact of life in humans (as well as other animals); but improved treatments as well as better understanding of causation will likely reduce suffering, as time progresses.

So let’s “play ball”!

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