Introduction

Contemporary surgical management of urinary stone disease has progressed tremendously due to improvements in extracorporeal shock wave lithotripsy (ESWL) technology and endourological instrumentation and ingenuity. The availability, ease, and high success rates of these procedures often overshadow the value of the metabolic component of stone disease but it is this facet that plays an important role in prevention of stone growth and recurrence.

This article will focus on a practical series of metabolic investigations and interventions that form the basis of a program for the medical evaluation and prevention of upper urinary calculi (Table 1). Issues specifically related to the older adult will be highlighted.

Strategic Aspects of Metabolic Evaluation

Important concerns in the assessment of older adults with urinary stone disease include why they are important, whom they affect, when to assess, where to test, and what investigations to do.

Why?
The lifetime risk of developing a kidney stone in North America is 10–15% with a recurrence rate of ~50% within 5–10 years. Nephrolithiasis is more common among men than among women throughout most of adult life except in the sixth decade, where the incidence falls for men but rises for women. Those affected are not keen to suffer through future recurrences of renal colic and will often request follow-up and advice to reduce the likelihood of recurrence.

Whom?
The issue of evaluating individuals with first time kidney stones remains controversial. Given that the 3–5 year recurrence or stone growth rate is 19–50%, one can make a strong argument for assessing all patients who have urinary stone disease. Also, keep in mind that a kidney stone may be the first manifestation of an unexpected underlying disease.

When?
Since the objective is to measure representative urinary risk factors, it is important to wait at least 3 months following a symptomatic stone episode to allow patients to return to typical fluid and dietary intakes, physical activity, and renal function.

Where?
An office-based physician can be the ideal investigator if he/she is prepared to take an interest in organizing the tests, reviewing results with the patient, making specific recommendations, and arranging follow-up visits to assess progress and compliance. Access to an interested outpatient dietician is valuable.
Table 1: Step-wise Approach to the Valuation and Prevention of Kidney Stones

<table>
<thead>
<tr>
<th>Step</th>
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<tr>
<td>Determine type of stone</td>
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<tr>
<td>Measure urinary risk factors</td>
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<tr>
<td>Manipulate fluid and diet</td>
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<tr>
<td>Arrange follow-up</td>
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<td>Consider drugs</td>
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What?

Typical causes of renal stones are calcium oxalate and/or calcium phosphate (although pure calcium phosphate stones are uncommon), 5–10% uric acid, 5–10% struvite, 1–2% cystine, and the remainder a variety of rare and unusual compositions (Figure 1).

The initial evaluation of an individual with urinary tract stones includes a complete urological history and focused physical examination with particular emphasis on the points in the history that will elucidate pre-urinary risk factors and establish the stone history. It should include a detailed description of previous stone events, that is, when they occurred, how they resolved (spontaneous or urological intervention and, if the latter, then what intervention was performed), what tests were done, and what the results were. Previous stone analyses should be sought. About 80–85% will be calcium oxalate and/or calcium phosphate (although pure calcium phosphate stones are uncommon), 5–10% uric acid, 5–10% struvite, 1–2% cystine, and the remainder a variety of rare and unusual compositions (Figure 1).

All patients should have up-to-date imaging. Noncontrast helical CT scan is the most accurate in diagnosing urinary stones as well as providing information regarding anatomy and obstruction; it may also help diagnose other unexpected causes of abdominal pain. Its downside includes limited availability, highest radiation exposure (i.e., about ten times more radiation than a plain x-ray of the abdomen), lack of information regarding function and expense; sometimes it is too sensitive in identifying inconsequential submillimetric stones. One should look for possible anatomical abnormalities, such as areas of infundibular/ureteric obstruction, medullary sponge kidney, and calyceal diverticula.

Renal ultrasonography can be useful to diagnose hydronephrosis and stones in the kidney and upper and lower ureter when CT is unavailable or radiation should be avoided for example, during pregnancy; it is poor in identifying mid-ureteric stones.

Fasting blood tests include calcium, uric acid, and creatinine. This list can be expanded, according to individual circumstances, but is usually sufficient to exclude significant abnormalities in renal function and calcium/uric acid metabolism. Serum phosphate does not appear to be an independent risk factor for urinary tract stone recurrence or complications, or a reliable early predictor of occult disease.

A fasting urine sample from each visit is analyzed as a routine urinalysis, for culture when indicated, and for calcium, creatinine, and pH. Fasting calcium/creatinine (Ca/Cr) ratios together with the serum calcium level allow a simple means of separating most individuals with hypercalciuria into absorptive, resorptive, and renal subgroups; the Ca/Cr should be less than 0.4 in the absorptive group but higher in the other two. Persistent elevations of fasting urinary pH suggest a degree of renal tubular acidosis.

Twenty-four urine collections are easy to perform and give important information on the risk of recurrent stone formation. Some older adults may benefit from written instructions reminding them how to perform them. Repetitive collections allow evaluation of patient compliance and risk. I prefer to obtain two 24-hour collections before each visit and try to have one done on a weekday and the other on a weekend. People eat, drink, and have different daily routines on weekdays and weekends, and these variations are reflected in their urine collections.

Individuals are required to record their dietary and fluid intakes while collecting their 24-hour urine. Ideally, these data are analyzed by a computer-based nutritional program, which establishes daily intakes of specific nutrients. This analysis provides objective information to review when counseling patients on recommended dietary changes.

Management

While patients are in the clinic, we review their diagnostic images, stone analyses, 24-hour urinary risk factors, and dietary analyses with them, so they have an opportunity to learn about their disease. It is very important to have a knowledgeable and interested dietician involved. The patient’s spouse or partner is encouraged to participate in dietary discussions, especially if the spouse or partner does most of the cooking, so that specific suggestions may be reinforced at home. Older adults often face unique problems: they may be a surviving spouse and may not have home support or an optimally balanced diet. Furthermore, older adults often have a reduced digestive and absorptive capability and may eat less due to diminished senses of taste and smell.

The general principles of therapy—increased fluid intake and elimination of dietary excesses to correct specific abnormalities of urinary risk factors—are outlined in Table 2. The objective is not to restrict anything below normal recommended intakes; this applies particularly to dietary calcium. Pharmacological intervention is only added if patients fail to comply or continue to show evidence of active stone formation. Correction of underlying abnormalities, such as parathyroidectomy for hyperparathyroidism, remains a cornerstone of therapy. Pre-urinary or environmental risk factors should be adjusted whenever possible.
Urine Volume

Although the importance of high fluid intake and urine output is stressed to all patients because it decreases the concentration of all solutes and counteracts urinary stasis, many individuals are chronically low water drinkers who find it very difficult to change. Advice should be given to patients to drink 10–12 glasses of fluids per day with ≥50% as water to keep their urine colourless. A slice of lemon or a few drops of lemon per glass or portable filtration systems can improve the taste of the water and make it more potable. Amounts should increase on days with high temperatures, increased physical activity, or dietary splurges. All older adults who head south for retirement will need to increase water intake!

Increased fibre intake among older adults, which is usually recommended to aid with constipation and other health concerns, also increases the need for water.

Occasionally, high fluid intake may be a problem for older adults assigned to a water-restricted diet related to cardiac or kidney diseases. Those with significant lower urinary tract symptoms such as urinary frequency and urgency (for example, overactive bladder), which tend to increase in prevalence with aging, will be reluctant to drink more because of an aggravation of their voiding symptoms.

Hypercalciuria

Elevated urinary excretion of calcium is a risk factor for recurrent calcium oxalate and phosphate stone disease. It is the best-studied, most talked about, and oft-treated urinary risk factor. Interestingly, it is the least important from a physicochemical point of view. Elevated urinary excretion of calcium should be managed as one urinary risk factor to manipulate, when necessary; however, I do not overly emphasize its correction by decreasing dietary calcium. Many individuals have already reduced their dietary calcium intakes based upon previous beliefs, despite the growing evidence to suggest that higher levels of calcium intake reduce the likelihood of calcium stone formation probably by binding to oxalate in the gut and reducing its absorption. This is particularly important in the older population who may have an element of osteoporosis and highlights the deleterious effect of reducing dietary calcium. A baseline bone densitometry study is useful for older adults, especially women, where there is evidence of hypercalciuria. One should aim to at least maintain the daily recommend allowances. Higher intakes, which may include supplementation with calcium citrate at meal times, will likely benefit the bone deficiency and risk of urinary tract stone disease. Renal hypercalciuria can be improved with thiazides, however, I use them only when dietary attempts at controlling all abnormal risk factors have been exhausted and renal hypercalciuria remains the major abnormality.

Hyperoxaluria

Even 10–15% elevations of urinary oxalate may have a significant impact on the saturation of calcium oxalate. Although the dietary contribution of oxalate to its urinary excretion is small, avoiding foods high in oxalate content can reduce this component. Enteric hyperoxaluria is uncommon but can lead to much higher levels of oxalate excretion. The principles of therapy are the same but may need to be expanded to include fat reduction, calcium and citrate supplements, and cholestyramine. Dietary fat is not important for individuals with kidney stones who don’t have gastrointestinal abnormalities.

### Table 2: Identification and Manipulation of Urinary Risk Factors for Specific Types of Urinary Stones

<table>
<thead>
<tr>
<th>Urinary risk factor</th>
<th>Type of stone</th>
<th>Dietary manipulation</th>
<th>Pharmacological manipulation</th>
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<tbody>
<tr>
<td>Low urine volume</td>
<td>a,b,c,d</td>
<td>Increased fluid intake</td>
<td>—</td>
</tr>
<tr>
<td>Hypercalciuria</td>
<td>a, b</td>
<td>Decreased excessive calcium, sodium, animal protein; increased fibre</td>
<td>Thiazides, potassium citrate</td>
</tr>
<tr>
<td>Hyperoxaluria</td>
<td>a</td>
<td>Increased calcium; decreased oxalate</td>
<td>Vitamin B6</td>
</tr>
<tr>
<td>Hyperuricosuria</td>
<td>a, c</td>
<td>Decreased purine</td>
<td>Allopurinol</td>
</tr>
<tr>
<td>Persistently low pH</td>
<td>a, c, d</td>
<td>Decreased animal protein</td>
<td>Potassium citrate, sodium bicarbonate, acetazolamide</td>
</tr>
<tr>
<td>Persistently high pH</td>
<td>a,b</td>
<td>—</td>
<td>Ammonium chloride (?)</td>
</tr>
<tr>
<td>Hypocitraturia</td>
<td>a</td>
<td>—</td>
<td>Potassium citrate</td>
</tr>
<tr>
<td>Cystinuria</td>
<td>d</td>
<td>Decreased sodium and animal protein (methionine)</td>
<td>Captopril, D-penicillamine, Thiola</td>
</tr>
</tbody>
</table>

(a = calcium oxalate, b = calcium phosphate, c = uric acid, d = cystine)
Low urine volume can cause all four types of urinary stones: calcium oxalate, calcium phosphate, uric acid, and cystine type stones. Other factors that cause urinary stones include hypercalciuria, hyperoxaluria, hyperuricosuria, persistently low or high pH, hypoci-traturia, and cystinuria.

The general principles of therapy involve increased fluid intake and elimination of dietary excesses to correct specific abnormalities of urinary risk factors.
Hyperuricosuria
Supersaturation with uric acid is necessary for the formation of uric acid stones. Since the main dietary sources of urinary acid are purine and nucleic acids, the major emphasis is to moderate their intake. When this urinary risk factor remains a difficulty after correction of other abnormalities, further suppression can be achieved with allopurinol.

pH
Urinary pH normally fluctuates throughout the day, but some persons excrete urine with a fixed high or low urinary pH. This lack of variability can negatively influence their risk of stone formation. If this problem is suspected, ask patients to measure and chart their urinary pH at home for 2 to 3 days before their clinic visit.

Individuals with a fixed low urinary pH, for example those with an ileostomy, tend to produce large quantities of uric acid crystals because of the low solubility of uric acid in a concentrated acidic environment. Similar problems may occur for individuals susceptible to cystine crystal formation. For both groups, dietary moderation of animal protein is a cornerstone of medical management because it will lead to a modest increase in urinary pH. If this measure is insufficient the next step is pharmacological intervention. One of the best drugs to increase urinary pH is potassium citrate. Sodium bicarbonate can be used for individuals with hyperuricosuria—as long as they have no difficulties with congestive heart failure or hypertension—but it is contraindicated for those with cystinuria because of the risk of increasing cystine excretion with the increased sodium load. While taking either of these drugs, patients should frequently monitor their urinary pH and avoid achieving a persistent value above 7.2 to prevent calcium phosphate precipitation.

The most common causes for high fixed urinary pH are urinary tract infection with a urea-splitting organism, hyperparathyroidism, and incomplete renal tubular acidosis. Persistently elevated urinary pHs over 7.2 are associated with an increased proportion of phosphate in the divalent and trivalent forms and higher rates of precipitation of calcium phosphate crystals either alone or as calcium magnesium ammonium.

Hypocitraturia
Citric acid exists primarily as the anion, citrate, at physiological pHs and since it binds to and forms a more soluble complex with calcium than oxalate, a low urinary excretion of citrate increases calcium oxalate supersaturation. Oral administration of alkali increases citrate excretion and can be achieved by the administration of potassium citrate.

Anatomical Abnormalities
Anatomical abnormalities of the urinary tract may predispose individuals to stone formation when they are associated with obstruction or recurrent/persistent urinary tract infection.

Key Points
Kidney stones occur frequently among the North American population and tend to be recurrent.

The physician and dietician can counsel the patient on how to reduce the likelihood of further recurrences or stone growth by increasing fluid intake and dietary modification. If this is unsuccessful, a variety of medications are available depending upon which variable is abnormal.

It is important to establish the type of stone an individual is forming either by stone analysis or educated guess from clinical scenario or x-ray appearance.

The general principles of therapy are increased fluid intake and elimination of dietary excesses to correct specific abnormalities of urinary risk factors. Only when this approach fails and a high risk of stone recurrence is present should drugs be introduced into the treatment strategy.

Long-term success is determined by the degree of patient education, compliance, and reduction in stone recurrence and growth.

Older adults with significant comorbidities may not be candidates for the surgical procedures required to correct these problems. Optimization of urinary risk factors predisposing to stone formation and judicious use of long term suppressive antibiotics may be the best you can offer in some circumstances.

Conclusions
Urinary stones are a recurrent problem in the majority of affected individuals. Older adults present unique problems because of the likelihood of complicating comorbidities, social circumstances, and difficult-to-change lifelong habits. An approach that is organized, efficient, cost effective, and stabilizing is required. It is important to establish the type of stone an individual is forming either by stone analysis or educated guess from the clinical scenario or x-ray appearance. Several abnormalities of urinary risk factors are usually relevant to particular types of stones. These problems can usually be identified and corrected.

Initial interventions should be directed to specific fluid and dietary manipulations to improve abnormalities in individual patients. Only when this approach fails and a high risk of stone recurrence is present should drugs be introduced into the treatment strategy. Occasionally this may introduce problems for older adults who may already be assigned to a variety of medications. Long-term success is determined by the degree of patient education, compliance, and reduction in stone recurrence and growth.

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References