

MANAGEMENT OF UROLITHIASIS – A REVIEW

Lawrence A, Koya MP.

North Shore Hospital, Auckland, New Zealand

Urolithiasis is a common multi-factorial disease that has been recognized and documented in medical literature since the Greek and Roman physicians. Urolithiasis encompasses both renal and ureteric stones. It is estimated to affect 102 per 100,000 per year New Zealanders¹, 15% of American men, 6% of American women² and 3 - 9% of Australians³ in their life-time. Stone disease varies with age, gender, ethnicity and season⁴. Fifty percent of patients will have recurrent stone disease within 5 years, so it can be considered a disease for life⁵.

Its commonality means that it is not only managed by urologists but more frequently in the acute setting by family practitioners and emergency specialists. Due to this it is important to have a working understanding of etiology and management of stone disease. This article attempts to introduce the complex etiology of stone disease, highlight the natural history of stone disease left untreated and cover important points of contemporary management.

Etiology

Stone formation is usually multi-factorial with more than one element increasing a patient's risk for stone formation. A detailed clinical history, examination and appropriate metabolic work up are important to identify possible risk factors for targeted prevention and treatment strategies. Some of these etiological agents are listed in table one.

Stones are made up of multiple constituents but

the first step in any stone formation is supersaturation of the urine⁶. This results in crystallization of constituents and a nucleus for further stone growth/aggregation. This step is usually inhibited by compounds in our urine but in some patients these are absent or defective.

Calculi are of different constituents including calcium, oxalate, uric acid, magnesium, phosphate, ammonium and cystine. Eight five percent of stones are calcium-based stones such as calcium oxalate and calcium phosphate. Ten to 15% are infection/struvite calculi, approximately 5% to 10% are uric acid calculi, and rarely cystine calculi. Over 90% of calculi are radio-opaque, and only uric acid, indinavir calculi are truly radio-lucent.

A smaller group of patients have metabolic disorders that result in more prevalent stone formation. As demonstrated in table two, this results in an increase incidence of calcium stones or non-calcium stones. Further details regarding metabolic disorders and related stone formation are beyond the scope of this article.

Natural History

The natural history of untreated stones is dependent on size and location. Staghorn or partial staghorn stones should never be left untreated as <5% of these stones remain asymptomatic. Greater than 75% of patients have significant complications including renal deterioration, sepsis due to xanthogranulomatous, pyelonephritis and/or perinephric abscess. These stones still have a mortality rate of up to 50%, largely due to renal failure⁷. Smaller renal stones have been shown to both grow in size and cause symptoms, or remain the same size and asymptomatic. This means that their management remains controversial, as there is no universal standard of care⁸.

Ureteric stones cause obstruction of the kidney. Within hours of obstruction occurring there are changes in renal blood flow and pressure within the obstructed kidney. These changes are reversible depending on the duration and extent of obstruction and if stones are treated and the collect-

Urine	Anatomy	Metabolic Abnormalities	Disease States	Diet
Urinary composition	Urinary stasis	Hypercalciuria	Metabolic acidosis	High protein diet
Urinary volume	Urinary obstruction	Hypocitraturia	Cystinuria	High sodium diet
Presence of stone inhibitors	e.g. horse shoe kidney	Hyperoxaluria	Inflammatory bowel disease	High oxalate diet
		Hyperuricosuria	Medications	Low fluid intake/ dehydration

Table 1: Some etiological agents for stone diseases

Calcium based stone abnormalities	Non calcium based stone abnormalities
Hypercalciuria	Hyperuricosuria with urine pH <5.5
Hypocitraturia	Cystinuria
Hyperuricosuria with urine pH >6	Struvite/ infection stones
Hyperoxaluria	
Hypercalcaemia	

Table 2: Calcium and non-calcium stone abnormalities

ing system becomes unobstructed. If stones are left untreated then these changes in renal blood flow and renal pressure are permanent. Long-term nephron damage can therefore occur.

The natural history of ureteric stones is depended on their size and location in the ureter. Ueno et al established ureteric stone passage times can be highly variable, however passage is dependent on stone size, location, and side⁹. Stones that are 4mm or less have a 90% chance of passing independent of surgical intervention. Stones larger than this have a decreasing likelihood of passing with increasing size and increasing proximity to the kidney.

Diagnosis

Stone disease can present either acutely as renal colic or as a chronic disease process, such as microscopic hematuria, recurrent urinary tract infections or renal failure. Stone disease may also be an incidental finding.

Renal colic should always be considered in the differential of acute abdominal pain. It is classically unilateral loin pain severe in nature that radiates to the groin, which waxes and wanes in nature. It may be associated with nausea and vomiting and is not relieved by postural changes.

Confirmation of the presence of stones is vital. Included in the work up to confirm the diagnosis should be a focused clinical history looking for possible risk factors for stone formation. These include:

- previous episodes of stone disease and family history of stone disease
- diet (high salt and oxalate)
- inadequate fluid consumption
- medications
- disease processes (such as gout, diabetes, bariatric surgeries, inflammatory bowel disease)
- immobilization
- urinary/bowel diversion surgery .

Imaging is important to:

1. establish the clinical diagnosis is correct

2. locate the stone, and establish stone burden/ size
3. direct further management with this information¹⁰.

CT scanning without contrast is the most accurate imaging available. However it is expensive and not readily accessible in some areas. Alternatively an ultrasound scan with Kidney Ureter Bladder (KUB) X-Ray should be considered¹¹.

It is crucial to recognize early, patients who require immediate urological intervention for their stone disease. This includes patients with:

- obstructed infected system/sepsis
- obstructed solitary kidney
- bilateral obstruction
- renal impairment/failure due to obstruction
- those in whom adequate analgesia is not controlling their pain.

Management

Management of any stone is dictated by stone location, composition, size and patient factors e.g. co-morbidities, solitary kidney and occupation.

All management of stones can be thought of as acute, definitive and preventative. Preventative management involves education of patients to decrease their risk of stone disease by modifying diet and hydration. Hydration is the most vital step in prevention as chronic dehydration has been identified as a cause of urolithiasis. Increasing fluid intake has been shown to decrease stone incidence. Borghi et al showed in calcium oxalate calculus formers that increasing the urine output to greater than 2 liters a day, results in a 12% recurrence in stone formation, compared to those with no specific fluid recommendations who had a 27% recurrence of calculi¹².

Any further diet changes should be dictated by common sense, and patients should be encouraged to eat a balanced diet and maintain a healthy BMI¹³. Any large modifications of diet should be dictated by biochemical analysis. However general guidelines for decreasing calculus recurrence are increased fluid intake, decreased sodium intake, decrease oxalate foods, reduce

animal protein to 2 servings a day and maintenance of a normal calcium intake.

Some stones are partially amenable to dissolution therapy; these include uric acid and cystine stones. Dissolution therapy is based on the solubility characteristics of the calculus in urine (pKa) and the manipulation of this such that more of the uric acid or cystine is soluble¹⁴. Citrate based medications; such as potassium citrate; are used as dissolution therapy and can be used to successfully manipulate the pH of the urine and pKa of the solutes with resultant dissolution of calculi. If implemented, the pH of the urine has to be monitored as if altered dramatically (increased to an alkaline state), this can cause the precipitation of calcium based stones.

Ureteric stones greater than or equal to 5mm should be referred for a urological opinion¹⁵. They have a decreased likelihood of passing spontaneously and hence to prevent a long term sequelae should be managed surgically. Ureteric stones 4mm or less can be considered for medical expulsion therapy (MET)¹⁶. MET decreases the need for opioid analgesia, decreases the time to expulsion and decreases the need for surgery. Alpha blockers and Calcium channel blockers can both be used for MET. A trial of MET can be considered unsuccessful if the stone is still present at four weeks post commencement. If MET does fail, then the patient should be referred on for a urological opinion.

As mentioned earlier staghorn calculi are associated with a high morbidity. All patients with staghorn stones should be referred for urological input¹⁷. Smaller renal stones that are symptomatic should also be referred for consideration of ureteroscopy or Extra Corporeal Shockwave Lithotripsy (ESWL). However asymptomatic smaller renal stones may be treated conservatively with observation. This does require ongoing imaging to ensure no growth of the stone. If the stone was to grow or cause symptoms then the patient should be referred for more definitive therapy.

Stone disease is a significant burden on the health care budget in a country like Samoa. Patient education, healthy lifestyle practice and prevention with early diagnosis will help in improving the health of the nation and reduce spending of the precious health dollar.

References

1. Du J, Johnston R, Rice M. Temporal trends of acute Nephrolithiasis in Auckland, New Zealand. *NZ Med J.* 2009; 122: 1299
2. Stamatelou KK, Francis ME, Jones CA, et al. Time trends in reported prevalence of kidney stones in the United States: 1976–1994. *Kidney Internat.* 2003; 63(5): 1817–

- 23.
3. Hughes P. *Kidney Stone Epidemiology. Nephrol.* 2007; 12: 26– 30.
4. Lo S, Johnston R, Al Sameraai A et al. Seasonal Variation in the Acute Presentation of urinary Calculi over 8 years in Auckland, New Zealand. *Brit J Urol Internat.* 2009; 106: 96- 101.
5. Trinchieri A, Ostini F, Nespoli R et al. A prospective study of recurrence rate and risk factors for recurrence after a first renal stone. *J Urol.* 1999; 162: 27–30.
6. Coe FL, Evan A, Worcester E. Kidney stone disease. *J Clin Invest.* 2005; 115(10): 2598 –608.
7. Singh M, Chapman R, Tresidder C, Blandy J. The fate of the unoperated Staghorn Calculus. *Br J Urol.* 1973; 45: 581- 585.
8. Keoghane S, Walmsley B, Hodgson D. The natural history of untreated renal calculi. *Br J Urol Internat.* 2010; 105(12): 1627-9
9. Ueno A, Kawamura T, Ogawa A, Takayasu H. Relation of spontaneous passage of ureteral calculi to size. *Urol.* 1977; 10(6): 544- 546.
10. Miller O, Kane C. Time to stone passage for observed Ureteral Calculi: A Guide for Patient Education. *J Urol.* 1999; 162(3): 690- 691
11. Niemann T, Kollmann T, Bongartz G. Diagnostic Performance of Low-Dose CT for the Detection of Urolithiasis: A Meta-Analysis. *American journal of Roentgenology.* 2008; 191:396- 401.
12. Borghi L, Meschi T, Amato F, Briganti A, Novarini A, Giannini A. Urinary Volume, Water and Recurrences in Idiopathic Calcium Nephrolithiasis: A 5-year Randomized Prospective Study. *Journal of Urology.* 1996; 155(3):839-843.
13. Eisner B, Eisenber M, Stoller M. Relationship between Body Mass Index and Quantitative 24hour urine chemistries in patients with Nephrolithiasis. *Urology.* 2010;75(6): 1289 -93.
14. Moran M, Abrahams H, Burday D, Greene T. Utility of oral dissolution therapy in the management of referred patients with secondarily treated uric acid stones. *Urology.* 2002; 59: 206-210.
15. Andersson L, Sylven M.et.al. Small renal caliceal calculi as a cause of pain. *Journal of Urology.* 1983 Oct;130(4):752-3.
16. Seitz C, Liatsikos E, Porpiglia F, Tiselius HG, Zwergel U. Medical therapy to facilitate the passage of stones: what is the evidence? *European Urology.* 2009; 56(3):455 -71.
17. Blandy JP, Singh M. The case for a more aggressive approach to staghorn calculi. *J Urol* 1976; 115: 505±6