Case Presentation

The Use of Lit-Control in Long Term Management of Recurrent Renal Stones; a Case Presentation.

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Key words: renal stones, recurrent, prevention, Lit-Control, crystallization

Abstract

Objective: To study the efficacy of Lit-control Balance in management of a patient with recurrent mixed renal stones.

Method: After multiple interventions for a patient with recurrent large renal stones, and stone formation laboratory investigations, the patient was started on treatment with Lit-control Balance and followed for 18 months.

Results: The patient is completely stone free after one year use of Lit-control balance.

Conclusion: The continuous use of Lit-control Balance help prevent the recurrence of renal stones in a well know stone former after one year of follow up.

Introduction

Clinical case description

Mr. M.R.I is a 48 years old (DOB 27/2/1976) Palestinian male patient from Bethlehem who works in trading. He is married and has no chronic illnesses. He gives a history of lumbosacral discectomy in 2012.

The patient gives a long history of recurrent stone formation since the age of 23 years. He gives a history of multiple episodes of renal colic and multiple stone formation some of which were passed spontaneously of needed management by ESWL or ureteroscopy.

He presented to my clinic for the first time on 21st November 2021 with recurrent right loin pain. Renal CT scan showed a large 3.3X2.8X1.5cm right renal pelvis staghorn stone causing mild back pressure associated with few tiny lower group calyceal stones. There were multiple middle and lower calyceal left kidney stones as well. (figure 1)

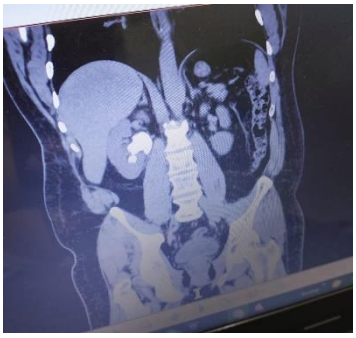

Figure 1:CT scan 21st Sep2021

On the 22nd Nov 2021 he underwent right pyelolithotomy and the stone was extracted in one piece and suturing was done over DJC (Figure 2). He was discharged home in good general condition. On the 6th December 2021 he underwent removal DJC and was free of stones.

Figure 2:stone extracted

On the 5th October 2023 he returned back with right loin pain and hematuria. Follow up CT scan showed a huge recurrent right renal pelvis staghorn stone 4X5 cm in size.

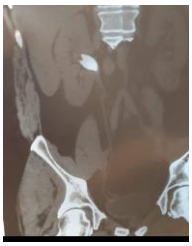

Figure 3: recurrent right renal stone 5th Oct 2023

On the 8th October 2023 he did redo- pyelolithotomy and complete extraction of stone.

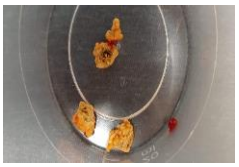

Figure 4: stone extracted 8th Oct 2023

Stone analysis revealed 50% uric acid and 50% calcium oxalate stone composition.

Figure 5: stone analysis

His blood and urine Lab workup revealed serum uric acid of 7.7 mg/dl and a normal parathyroid serum level with normal urine inhibitors and electrolyte composition.

On the 10th October 2023 he was started on Lit-Control Balance twice daily for 3 months then once daily life long.

He had regular follow up in the clinic with repeated renal CT scans showed no recurrent stones where the last CT was on the 23rd June 2024. The patient is still on regular follow up and continuous contact with me and is completely asymptomatic.

Discussion

Kidney stone is a major cause of morbidity and affects approximately 1–15% of the world's population (1). The types of kidney stone include calcium oxalate, calcium phosphate, uric acid, struvite, and mixed stones, among which calcium stones are the most common and include about 70 to 80% of the stones.

A big percentage of stone formers have no specific cause for formation of the stones. It is intriguing that despite marked abnormal urinary factors, most humans will not form stones. Alternatively, some patients develop stones despite normal urinary composition. So it is unclear exactly why many people form stones (2). A genetic component to recurrent stone formation has been recognized for decades. Studies have confirmed the heritability of patterns of urinary excretion of calcium, citrate, oxalate, and uric acid. Many genetic loci have been implicated in modifying disease risk but have not yet been translated into actionable therapeutics. Monogenic causes of nephrolithiasis do exist and include cystinuria, primary hyperoxaluria, adenine phosphoribosyl transferase deficiency, and Dent's disease.

Stones are likely to recur, with at least 50% of individuals experiencing another stone within 10 years of the first occurrences and 75% in 20 years (1). The exact mechanism of renal stone formation is still not totally understood where there is a complex process involving crystal nucleation, aggregation and/or secondary nucleation, fixation within the kidney, and more aggregation and secondary nucleation. These steps are heavily modulated by the balance of amounts of stone constituents appearing in tubular fluid, their concentration as affected by water excretion, the pH of tubular fluid and/or urine, and the balance of promoters and inhibitors that are not major components themselves of the clinical stones (3). Thus, the role of crystallization inhibitors at different stages of stone development, the influence of preexisting solid particles and the effects of variations in urine composition is the target for prevention of stone formation., success strategies for diminishing stone recurrence rate have been based on manipulating these processes. The key element, therefore, appears to be inhibition of the steps in calculogenesis (nucleation, crystal growth, aggregation, and crystal/stone retention). Urolithiasis will not develop if any one of these steps is blocked (2).

To study the possible effects caused by the interaction of some urinary components on the inhibition of calcium oxalate crystallization. Such interactions are susceptible to importantly change the inhibitory behavior of some urinary components by producing either positive (synergistic) or negative effects on preventing crystallization (4).

The crystallization inhibitory capacity of target urine is explained by the combined effect of the compounds present in the complex urine matrix rather than the individual action of each compound. This kind of interactions is of key value in designing prophylactic treatments of urolithiasis based on

inhibitors intake. Known inhibitors include citrate, pyrophosphate, phytate, and magnesium, as well as proteins such as uromodulin, glycosaminoglycans, osteopontin, and calgranulin.

Urine pH modifies the solubility of different solutes. Calcium phosphate and struvite are less soluble at a higher pH; thus, in alkaline urine these components are predisposed to form stones. By contrast, uric acid and cystine are less soluble at a lower pH, making their stones more likely to form in acidic urine.

Urinary citrate appears to be an important factor in the crystallization process of calcium oxalate and calcium phosphate. The urinary excretion of citrate was found to be significantly lower in patients with calcium oxalate stone disease as compared with normal subjects, and about 30 per cent of the calcium stone formers can be considered as hypocitraturic. The lowest excretion of citrate was recorded in urine collected during the night. Citrate has significant effects on supersaturation with respect to both calcium oxalate and calcium phosphate, it also inhibits the growth of these crystals. In addition, citrate appears to be capable of inhibiting the aggregation of crystals composed of calcium oxalate, brushite, and hydroxyapatite. The heterogenous growth of calcium oxalate on calcium phosphate is also counteracted by citrate. As a consequence of the crucial role of citrate in these processes, stone prevention with alkaline citrate has become an attractive form of treatment in patients with recurrent stone formation. Single evening dose administration of sodium potassium citrate resulted in an of sodium potassium citrate resulted in an increased excretion of citrate, reduced levels of the calcium/citrate ratio as well as supersaturation with respect to calcium oxalate and a decreased rate of stone formation (5).

There is an extraordinary capacity of phytate (myo-inositol hexaphosphate), a substance present in blood, urine, interstitial and intracellular fluids, to inhibit crystallization of calcium salts (oxalate and phosphate). Clinical studies clearly demonstrated that phytate plays an important role as a crystallization inhibitor of calcium salts in biological fluids and becomes a clear alternative in the treatment of calcium oxalate urolithiasis (6).

The calcium antagonistic effect of magnesium is also important for reducing the risk of kidney stones. Magnesium decreases the possibility of stone formation by working through three proposed mechanisms: 1) binding oxalate in the gastrointestinal tract, preventing its absorption, 2) binding oxalate in urine to outcompete its interaction with calcium and 3) chelating with citrate, preventing its resorption to increases urinary citrate levels (7). Despite these benefits; the clinical evaluation of magnesium has demonstrated equivocal results. Some studies were able to demonstrate that an increased magnesium intake was associated with decreasing hyperoxaluria in patients on a retrospective review of 24-hour urinalysis. Yet other prospective, double-blind, randomized, controlled trial conducted to examine the effectiveness of magnesium hydroxide in the prevention of calcium oxalate stones failed to show a difference.

Many studies showed that the synergistic effect between magnesium and phytate on calcium oxalate crystallization suggests that a combination of these 2 compounds may be highly useful as antilithiasis therapy (8).

For patients at risk of recurrent renal stones, a detailed medical evaluation and an individualized approach to dietary and pharmacological prevention are important aspects of their care.

In my patient the use of Lit-Control Balance containing calcium magnesium phytate with allopurinol and increase fluid intake succeeded in prevention of recurrent combined stones (calcium oxalate and uric acid) for over a year now.

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