

## A Retrospective Study to Determine the Type and Fate of Urinary Calculi Incidence among Urinary Patients

**Saad Muhmood Hussain**

College of Medicine Diyala University

Correspondence: Assistant Professor Saad Muhmood Hussain (MRCS, FICMS)

[dr\\_saad70@yahoo.com](mailto:dr_saad70@yahoo.com)

### Abstract

**Background:** Kidney stones do not have single, well defined cause, but are the result of a combination of factors. A stone is created when the urine does not have the correct balance of fluid and a combination of minerals and acids .when the urine contains more crystal-forming substances than the fluid can dilute, crystals can form .Normally the urine contains components that prevent these crystals from attaching to each other. However, when these substances fall below their normal proportions, the stones can form out of an accumulation of crystals. **Objective:** The study was aimed to determine the type and fate of urinary calculi in our province and assessment the types of crystaluria among the patients with urinary calculi. **Patients and methods:** This is a retrospective study that held in diyala province (Baquba teaching hospital and private clinics) in the period from 1/1/2004 to 31/12/2011 in which 2100 patients with urinary calculi included 1500 patients were males and 600 patients were females. The ages were ranged from (6 months to 75 years). These patients were submitted to clinical examination, urinalysis and serum uric acid estimation and imaging Kidneys, Ureters, Bladder x-ray (KUB), ultra sonography, Intravenous urography (IVU) and Computed tomography (CT) scanning with contrast.100 stones were analysed. **Result:** The sex ratio (male/female) was 2.5/1. 1650 patients (78.57%) with single stone, while 450 patients (21.42%) with multiple stones. 200 patients (28.57%) showed hyperuricaemia. The types of crtstaluria using urinalysis showed 900 with oxalate, 490 with uric acid, 180 with phosphate, 120 with carbonate and 390 with no crystal. 1710 patients had past their stones spontaneously, 300 patients needed Extracorporeal shockwave lithotripsy (ESWL) and 90 patients were in need for surgical intervention. Also the stones analysis showed 60 stones composed of mixed crystal determine the type, fate, 20 stones composed of uric acid only determine the type, fate, 10 stones composed of uric acid and oxalate, 10 stones composed of phosphate only. In conclusion, there was high incidence of uric acid stones, and most urinary stones can passed spontaneously.

**Keywords:** Crtstaluria, carbonate, urinalysis, oxalate, phosphate uric acid, urinary calculi

{**Citation:** Saad Muhmood Hussain. A retrospective study to determine the type and fate of urinary calculi incidence among urinary patients. American Journal of Research Communication, 2013, 1(2): 56-65} [www.usa-journals.com](http://www.usa-journals.com), ISSN: 2325-4076.

## Introduction

Kidney stones do not have single, well-defined cause, but are the result of a combination of factors (1) Deficiency of vitamin A cause desquamation of epithelium, the cells form a nidus around which stone is deposited. (2) Dehydration leads to an increased concentration of urinary solutes and tends to cause them to precipitate (3) The presence of citrate in urine, 300-900 mg /24 hr (1.6-4.7 mmol/24 hr) as citric acid tends to keep otherwise relatively insoluble calcium phosphate and citrate in solution, the urinary excretion of citrate is under hormonal control and decreases during menstruation so decreased urinary citrate will lead to stone formation. (4) Renal infection. (5) Inadequate urinary drainage and urinary stasis. (6) Prolonged immobilization. (7) Hyperparathyroidism [1] Kidney stone or calcium oxalate crystals in the kidney can be due to underlying metabolic conditions, such as renal tubular acidosis [2], Dent's disease [3], Primary hyperoxaluria [4], and medullary sponge kidney [5].

Kidney stones are also more common in patients with Crohn's disease [6]. Patients with recurrent kidney stones should be screened for these disorders. This is typically done with a 24 hour urine collection that is chemically analyzed for deficiencies and excesses that promote stone formation [7]. There has been some evidence that water fluoridation may increase the risk of kidney stone formation. In one study, patients with symptoms of skeletal fluorosis were 4.6 times as likely to develop kidney stones [8].

Astronauts seem to show a higher risk of developing kidney stones during or after long duration space flights [9]. The most common type of kidney stone is composed of calcium oxalate crystals, occurring in about 75 to 80% of cases, and the factors that promote the precipitation of crystals in the urine are associated with the development of these stones. Perhaps counter intuitively, current evidence suggests that the consumption of low calcium diet is associated with a highest overall risk for the development of kidney stones [10]. This is perhaps related to the role of calcium in binding ingested oxalate in the gastrointestinal tract. As the amount of calcium intake absorption, the amount of oxalate available for absorption into the blood stream increases this oxalate then excreted in greater amount into the urine by the kidneys. In the urine, the oxalate is very strong promoting the calcium-oxalate precipitation, and it is about 15 times stronger than calcium.

Other types of kidney stones are composed of struvite (magnesium, ammonium and phosphate), calcium phosphate and cystine. Struvite stones are also known as infection stones, urease or triple-phosphate stones. About 10-15% of urinary calculi consist of struvite stones [11]. The formation of struvite stones is associated with the presence of urea-splitting bacteria most commonly *Proteus mirabilis* also klebsiella, Serratia, Providencia Spp. These organisms are capable of splitting urea into ammonia, decreasing the acidity of the urine and resulting in favorable conditions for the formation of struvite stones. Struvite stones are always associated with urinary tract infections [12].

The formation of calcium phosphate stones is associated with conditions such as hyperparathyroidism and renal tubular acidosis. Formation of cystine stones is uniquely associated with people suffering from cystinuria, who accumulate cysteine in their urine. Cystinuria can be caused by Fanconi's syndrome. Urolithiasis has also been noted to occur in the setting of therapeutic drug use, with crystals of drug forming within the renal tract in some patients currently being treated with Indinavir, Sulfadiazine or Triamterene. Preventive strategies may include dietary modifications with the goal of reducing excretory load on the kidneys [13].

Hypocitraturia is a known risk factor for kidney stone formation. By forming soluble complexes with calcium, citrate prevents crystal nucleation, aggregation and growth. Therefore, the presence of citrate in the urine reduces the risk for calcium stone formation [14].

Nephrolithiasis is a result of formation and retention of crystals within the kidneys. The driving force behind crystal formation is urinary supersaturation with respect to the stone-forming salts, which means that crystals form when the concentrations of participating ions are higher than the thermodynamic solubility for that salt. Levels of supersaturation are kept low and under control by proper functioning of a variety of cells including those that line the renal tubules [15].

All stones share similar presenting symptoms, and urine supersaturation with respect to the mineral phase of the stone is essential for stone formation. However, recent studies using papillary biopsies of stone formers have provided a view of the histology of renal crystal deposition which suggests that the early sequence of events leading to stone formation differs greatly, depending on the type of stone and on the urine chemistry leading to supersaturation. Three general pathways for kidney stone formation are seen: (1) stones fixed to the surface of a

renal papilla at sites of interstitial apatite plaque (termed Randall's plaque), as seen in idiopathic calcium oxalate stone formers. (2) Stones attached to plugs protruding from the openings of ducts of Bellini, as seen in hyperoxaluria and distal tubular acidosis and (3) stones forming in free solution in the renal collection system, as in cystinuria. The presence of hydroxyapatite crystals in either the interstitial or tubule compartment and sometimes both of the renal medulla in stone formers is the rule and has implications for the initial steps of stone formation and the potential for renal injury [16]. The study was aimed to determine the type, fate and assess the types of crystaluria among the patients with urinary calculi

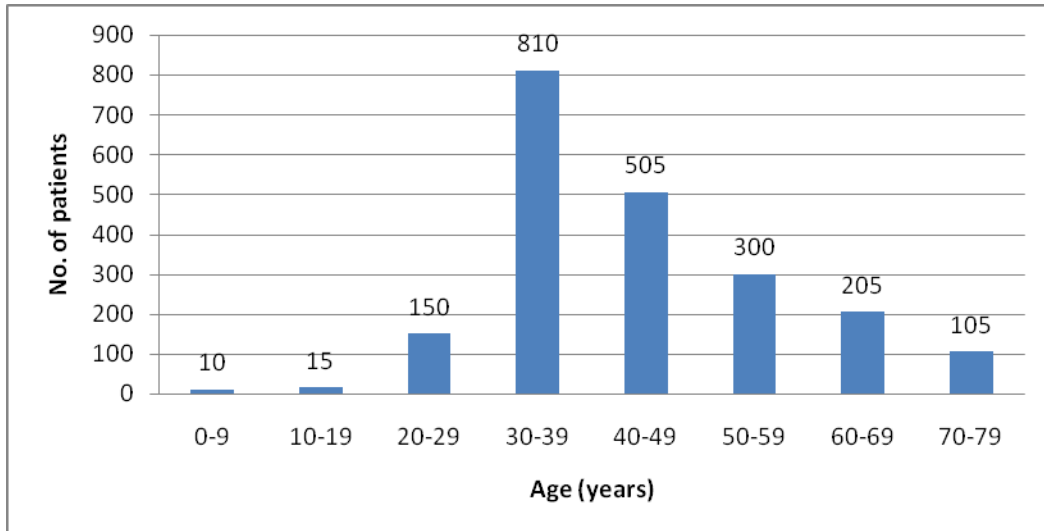
### **Patients and Methods**

This is a retrospective study was held in Baquba teaching hospital and private clinic/ Diyala province in the period from 1/1/2004 to 31/12/2011. Total of 2100 patients with urinary calculi were included 1500 males and 600 females. The ages are ranging from 6 months to 75 years those patients admitted with urinary problem were submitted to clinical examination. The general urine examination and serum uric acid estimated then sent for imaging of Kidneys, Ureters, Bladder x-ray (KUB), ultra sonographic examination some patients for Intravenous urography (IVU) and/or Computed tomography (CT) scanning with contrast were arranged for them when it is necessary according to ultra sonographic findings, follow up of the patients had been performed with ultra sonographic and KUB to assess the fate of stones.

It was about 100 stones collected from 70 patients were able to collect their stones after spontaneous passage and other 30 patients who had been operated upon with different modalities of surgery. Patients with recurrent and multiple stones were assessed for hyperparathyroidism by estimation of parathyroid hormone (PTH).

**Results**

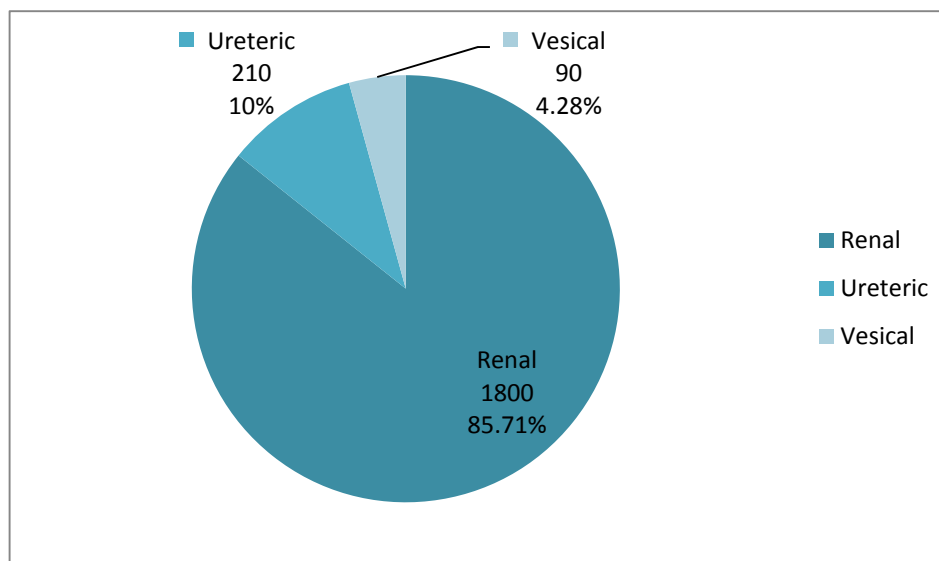
The results of this study showed the sex ratio (male /female ) was 2.5/1 and the major number of the patients was in age group 30-39 years old (Graph 1)



Graph 1: Showing the number of patients with renal stones to age group

Majority of patients 1650 (78.57%) had got single urinary stone, while 450 (21.42%) had got multiple urinary stones. On another hand, the majority of patients 1800 (85.71%) out of the total had got renal stone only, while other got urinary stones in different location (Fig I).

Fig. I: Showing the distributions of the stones in the urinary tract



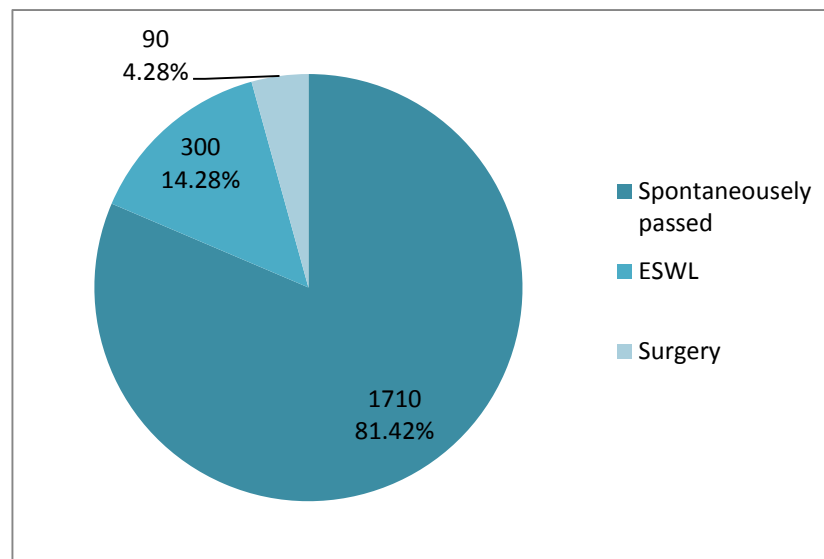
The blood analysis showed 200 (28.57%) patients out of total had got hyperuricaemia. The urine test showed the majority (900) of patients had oxalate crystal, 490 had uric acid, 180 with phosphate, 120 patients with carbonate and 20 patients showed mixed forms, while 390 patients did not show any crystalurea.

Table 1: Showing the types of crystalurea in patients with urinary calculi

Crystal	Oxalate	Uric acid	Phosphate	Carbonate	Other	Nil	Total
No. of Patients	900	490	180	120	20	390	2100

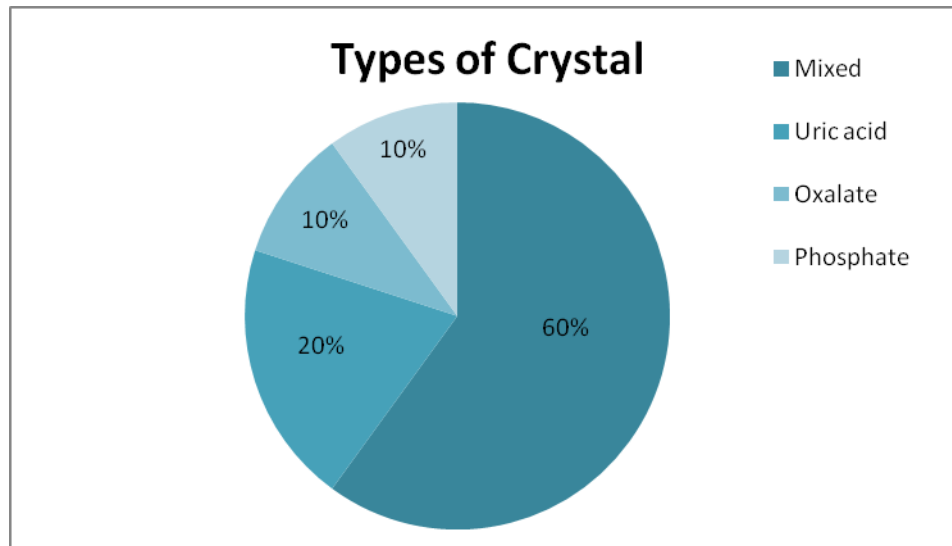
Regarding the treatment of the patients with urinary stones were showed that: 1710(81.42 %) out of total passed their stones spontaeously with aid of medical treatment and they did not need any surgical or ESWL treatment. However, 300(14.28%) patients were needed to ESWL due to persistent of their stones in spite of medical treatment while 90(4.28%) patients were in need for surgical intervention of different types of operations (nephrolithotomy, ureterolithotomy, dormiabasket extraction and vesicolithotomy (Figure II)

Figure II: Showing the modalities of treatment for stones.



The analysis of 100 stones showed the majority (60) of stones were mixed crystals in their composition of urate, phosphate, oxalate and oxalate, while 20 stones composed of uric acid only, 10 stones composed of oxalate and 10 stones composed of phosphate only (Figure III).

Figure III: showing the types of crystals composition



## Discussion

Crystalluria may occur in the presence or absence of hyperuricemia or hyperuricosuria. Low urine pH may cause crystals in the urine of normal subjects [17]. Uric acid stones form in association with conditions that cause hyperuricosuria with or without high blood serum uric acid levels (hyperuricemia) and with acid/base metabolism disorders where the urine might be excessively acidic (low pH) resulting in uric acid precipitation [18]. This supports our study findings since we found that only 200 patients had got hyperuricaemia but crystalluria of uric acid were found in 490 specimens. About 5-10% of all stones were formed from uric acid but in this study 20% of patients had got uric acid stones.

Urinary stone diseases are increasing in the Middle East, the mineralogical composition of the urinary stones were found as oxalate, cholesterol and uric acid with cysteine stones occurring more frequently than the others. Cholesterol and calcium oxalate stones are the most dominant

types of stones [19], but in this study found that the common types of stone were mixed type with none of cholesterol or cysteine stones.

Kidney stone occur in approximately 10 % of patients in their lifetimes, and > 10 crystal types have been reported in the literature [20], while this study detected that the stones contain only four types of crystals. Previous investigations done by Robertson and his colleagues on the influence of diet in particular of animal protein- rich diet on urinary stone formation [21] and this explain the reason of our patients' stones were uric acid types, which might be due to ingestion of high amount of meat by Iraqi people.

In the renal stones, the calcium oxalate and calcium phosphate were found in various crystal forms and states of hydration can be identified. Calcium oxalate monohydrate (COM) or whewellite and calcium oxalate dehydrate (COD) or weddellite were the commonest constituents of calcium stones. Calcium oxalate stones might be pure or mixed usually with calcium phosphate or sometimes with uric acid or ammonium urate [22] this is coincided with our study findings.

A reason for the low prevalence of pediatric urolithiasis is that pediatric urinary macromolecules have stronger inhibitory effects against oxalate induced renal cell injury and oxidative stress induced apoptosis. Furthermore, results suggest that osteopontin and calgranulin B expression is down-regulated in children due to this inhibitory effect and thus stone nidus formation is controlled [23]. This may explain the reason that the children had got low incidence of urinary stones which also coincide with our study finding in which that we found few cases of childhood had got urinary stones. In conclusion, there was high incidence of uric acid stones in our patients and most urinary stones can pass spontaneously, therefore, no need to be in hurry to decide other modalities of surgical treatment unless there is absolute indication for that.

### **Acknowledgment**

The author would like to thank the staff in the Baquba teaching hospital and private clinics in Diyala province for their assistances.



## References

1. Russell R.C.G, Norman S. Williams, Christopher J.K. Bulstrode (2004). The Kidneys and Ureters, Short Practice of Surgery, 4<sup>th</sup> Edition, p1315-1317.
2. Labcd Moe, Orson W. (2006). Kidney stones: pathophysiology and medical management. *Lancet* 367 (9507):333-344.
3. Lloyd SE, Pearce SHS; Fisher SE; Steinmeyer K, Schwappach B, Scheinmam SJ, Harding B, Bolino A, Devoto M, Goodyer P, Rigden SPA, Wrong O, Jentsch TJ, Craig IW, Thakker RV (1996). A common molecular basis for three inherited kidney stone diseases. *Nature* 379 (6564):445-449.
4. Hope B, Langman CB (October 2003). A United State survey on diagnosis, Treatment and outcome of primary hyperoxaluria. *Pediatric Nephrology* 18 (10):986-991.
5. Ginalski JM, Portmann L, Jaeger P (1991). Does medullary sponge kidney cause nephrolithiasis? *American Journal of Roentgenology* 156 (4):872-3.
6. Bohles H, Beifuss OJ, Brandl U, Pichl J, Akcetin Z, Demling L (1988). Urinary factors of Kidney stone formation in patients with crohn's disease. *Klinische Wochenschr* 66 (3): 87-91.
7. Marshall Cavendish Corporation (2007). Diseases and Disorders, volume 2 .Marshall Cavendish. PP. 491. [ISBN 0761477721].
8. National Research Council (2002). Fluoride in Drinking Water: A Scientific Review of EPA's Standard. New York: National Academies Press. [ISBN 030910128X].
9. Ciftcioglu N, Haddad RS, Golden DC, Morrison DR, McKay DS (2005). A Potential cause for kidney stone formation during space flights: enhanced growth of nanobacteria in microgravity. *Kidney International* 67 (2):483-91.
10. Parmar, Malvinder S. (2004). Kidney Stones. *British Medical Journal* 328(7453): 1420-1424.
11. Jennette JC, Heptinstall RH (2007). Charles Jenntte, Jean L. Olson Schwartz, Melvin M.; Silva, Fred G. Heptinstall's pathology of the kidney. 1 (6th ed.). Lippincott Williams and Wilkins. PP. 1063. ISBN 0781747503].
12. Jennette JC, Heptinstall RH (2007). Charles Jenntte, Jean L. Olson Schwartz, Melvin M.; Silva, Fred G. Heptinstall's pathology of the kidney. 1 (6th ed.).Lippincott Williams & Wilkins. PP. 1077. [ISBN 0781747503].
13. Goldfarb, David S., Coe, Fredric L. (1999). Prevention of recurrent nephrolithiasis" *American Family physician* 60 (8):2269-
14. Zacchia M, Presig P. (2010). Low urinary citrate: an overview. *J Nephrol.* 3 supply 16:S49-56.

15. Khan SR, Canales BK (2009). Genetic basis of renal cellular dysfunction and the formation of kidney stones. *Urol Res*; 37(4): 169-80.
16. Evan AP. (2010). Physiopathology and etiology of stone formation in the kidney and the urinary tract. *Pediatr Nephrol*, .25 (5): 831-41.
17. Kurmar, Vinay, Fausto, Nelson, Fausto, Nelso, Robbins, Stanley L, Abbas, Abul K. Cotran, Ramzi S. (2005). Robbins and Cortran Pathologic Basis of Disease (7th ed.). Philadelphia, Pa. Elsevier Saunders. pp. 1012. ISBN 0-7216-0187-1.
18. Halabe A, Sperling O. (1994). Uric acid nephrolithiasis. *Mineral and electrolyte metabolism* 20 (6):424-31.
19. Abboud IA. (2008). Mineralogy and chemistry of urinary stones: patients form North Joradan. *Environ Geochem Health*. 30(5):445-63.
20. Park S. (2007). Medical management of urinary stone disease. *Expert Opin Pharmacother*. 8(8):1117-25.
21. Bichler KH. (2006). Thirty-eight years of stone meetings in Europe. *Urol Res*, .34(2):70-8.
22. Trinchieri A, Castelnuovo C, Lizzano R, Zanetti G. (2005). Calcium stone disease: a multiform reality. *Urol Res*;33(3): 194-8.
23. Momohara C, Tsujihata M, Yoshioka I, Tsujimura A, Nonomura N, Okuyama A. (2009). Mechanism underlying the low prevalence of pediatric calcium oxalate urolithiasis. *J Urol*.; 182 (3):1201-9.