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Kidney Stones with Composition and Structure of Stones in Children with Hyperparathyroidism

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ABSTRACT
Calcium-apatite and mixed stones were formed due to renal tubular acidosis and primary hyperparathyroidism. In patients with calcium nephrolithiasis, hypercalciuria is observed in 40-50% of cases, which develops as a result of increased absorption of alimentary calcium and disturbance of tubular reabsorption. The study was carried out using the method of X-ray diffraction analysis (X-ray diffraction), which was performed on a DRON-4 diffractometer. An analysis of the mineral composition of kidney stones was carried out in 54 children with nephrolithiasis (a comparison group) and 47 children with kidneys with primary hyperparathyroidism. (70.1%) children, of which 13 (31.1%) had single-sided single stones and 26 (62.2%) had multiple bilateral stones. In children with nephrolithiasis in primary hyperparathyroidism, 90 (kidneys) kidneys were affected by calculus. Among them, in 48 (53.3%) kidneys, coral stones were noted. A study showed that the mineral composition of kidney stones can be used to judge the damage to the skeletal system in primary hyperparathyroidism.

INTRODUCTION

Urine samples from healthy individuals may contain crystals of oxalate, phosphate, uric acid, cystine, etc. Their detection in the urine does not indicate urolithiasis. Calcium oxalate and calcium phosphate (41%) mixed stones, calcium oxalate and calcium phosphate (27%), calcium phosphate stones (7%), magnesium-ammonium phosphate (15%), uric acid stones (7%) and cystine (2%) are the different types of urinary stones[1-2]. The majority of kidney stones (n = 1041) were formed of calcium oxalate, while mixed calcium-oxalate and calcium-apatite stones were found in 485 (34.8%) and 146 (10.1%) of patients with calcium-apatite stones, according to experts who examined the composition of kidney stones in 1392 patients[3].

The formation of mixed and calcium-apatite stones was attributed to primary hyperparathyroidism and renal tubular acidosis[4]. Oxalates (acid or alkaline urine reaction): these are calcium salts of oxalic acid that are often thick, nearly black in color and have a prickly surface. The most prevalent and excessively saturated component that forms stones is calcium oxalate, which is 4-5 times more soluble in normal urine and has a nephrotoxic effect on kidney epithelial cells[5]. 40-50% of patients with calcium nephrolithiasis experience hypercalciuria, which is brought on by increased intestinal calcium absorption and tubular reabsorption disruption. Of these patients, idiopathic hypercalciuria is observed in 40% of cases[6].

Mineral concretions in the renal calyces and pelvis that are either free or attached to the renal papillae are known as kidney stones, or calculi. In contrast, nephrocalcinosis is the term used to describe diffuse renal parenchymal calcification[7]. Nephrolithiasis, also called urolithiasis, is the term for stones that form in the urinary tract when urine gets overly supersaturated with a certain mineral. This causes crystals to form, grow, aggregate and remain in the kidneys[8]. Worldwide, calcium oxalate (CaOx) combined with calcium phosphate (CaP) accounts for over 80% of kidney stones. Uric acid, struvite and cystine-containing stones are also prevalent and make up roughly 9%, 10% and 1% of all stones, respectively[9].

Certain comparatively insoluble medicines or their metabolites can also cause urine to become supersaturated, which can result in crystallisation in the renal collecting ducts (iatrogenic stones). Nephrolithiasis, for instance, can occur in HIV patients receiving treatment with protease inhibitors such indinavir and atazanavir[10] Both indinavir and atazanavir are processed by the liver and a significant amount of the medication is eliminated unaltered in the urine. This causes the drugs to crystallise and cause kidney stones[11]. Even when used in combination with other medications, atazanavir has the potential to crystallise in the urine and cause kidney stones[12].

MATERIALS AND METHODS

This study was conducted in Department of Gastroenterology with the collaboration of Nephrology at National Institute of Medical Sciences and Research, Jaipur. In our study, an analysis was made of the mineral composition of kidney stones in the examined children. The study was carried out using the method of X-ray diffraction analysis (X-ray diffraction), which was performed on a DRON-4 diffractometer. An analysis of the mineral composition of kidney stones was carried out in 54 children with nephrolithiasis (a comparison group) and 47 children with kidneys with primary hyperparathyroidism.

RESULTS

In patients with primary hyperparathyroidism, 18 (17.2%) children had unilateral kidney injury with calculus, 6 (28%) of these children had numerous stones. 39 (70.1%) children had bilateral renal lesions with calculus, 13 (31.1%) of them had single-sided stones and 26 (62.2%) had numerous bilateral stones. Ninety (kidneys) of the children with primary hyperparathyroidism and nephrolithiasis had kidney calculus. Coral stones were found in 48 (53.3%) of the kidneys out of them.

DISCUSSION

The kidneys are the primary sites of manifestation for specific signs and symptoms of primary hyperparathyroidism. It was discovered that renal pathology resulting from recurrent nephrolithiasis or calcium accumulation in the renal parenchyma affected 60-70% of patients with primary hyperparathyroidism[13]. Most kidney stones are composed of calcium phosphate or oxalate. Large stones or recurrent stone development can impede renal function and cause infection and urinary tract blockage. Nephrolithiasis development is predisposed by increased urine calcium excretion. Phosphate retention and decreased renal function are other effects of nephrocalcinosis. Damage to the kidneys and skeletal system indicates increased parathyroid function. The direct effects of high calcium and parathyroid hormone cause the kidney tissue's mitochondrial activity to decline, which in turn leads to an excessive build-up of mucoproteins in the kidney tissue[14]. Mucoproteins cause the renal tubules to get clogged with protein molecules and cause urothelial necrosis. Protein components make up an organic base called the matrix, in which calcium crystals accumulate and form spherulites. These spherulites grow larger

Table 1 The composition of kidney stones consisting of mixed minerals in the examined children

Minerals	Urolithiasis (n = 39) (comparison group)	Renal form of primary hyperparathyroidism (n = 23)	Renal form of primary hyperparathyroidism with bone damage (n = 23)
Calcium oxalate monohydrate, Calcium oxalate dihydrate, ammonium urate	6 (12,1%)	2 (6,1%)	-
Calcium oxalate dihydrate, ammonium urate	11 (28,1%)	-	-
Calcium oxalate monohydrate, ammonium dihydrate	3 (6,1%)	2 (6,1%)	-
Calcium oxalate monohydrate, ammonium urate	8 (21,1%)	3 (13,3%)	-
Struvite, ammonium urate	7 (18,1%)	-	-
Calcium oxalate monohydrate, dollite	-	-	2 (11,1%)
Struvite, calcium oxalate monohydrate	8 (18,1%)	-	
Struvit, whitlockite	-	3 (6,1%)	2 (21,2%)
Struvit, calcium oxalate dihydrate, ammonium urate	-	3 (12,3%)	-
Hydroxylapatite, calcium oxalate monohydrate, ammonium urate	-	8 (39,2%)	5 (42,1%)
Brushite, whitlockite, calcium oxalate monohydrate	-	-	3 (10,1%)
Hydroxylapatite, calcium oxalate Monohydrate	-	2 (6,1%)	-
Struvite, brushite, sodium urate	-	2 (6,1%)	-
Hydroxylapatite, struvite	-	-	2 (11,2%)
Hydroxylapatite, ammonium urate	-	2 (6,1%)	-
Hydroxylapatite, calcium oxalate Monohydrate, calcium oxalate dihydrate	-	2(6,1%)	-
Total	39 (54,3%)	23 (70,1%)	23 (49,1%)

and become macrolites, which eventually constitute the nucleus of future calculi in the kidneys as a whole. Stone is created when mineral salts build up on macrolites. Urate stones are caused by high calcium and parathyroid hormone, which have an impact on purine metabolism. Nonetheless, there is ongoing debate on the frequency and type of stones that form in primary hyperparathyroidism and the makeup of stones that are found occasionally deviates from accepted theories.

Urinary stones containing calcium (mostly oxalates) are formed in part due to an increase in calcium ions. Urinary stones in the form of urates and phosphates can develop as a result of elevated uric acid and PO3-4 ions in the urine. Furthermore, an increase in uric acid concentration in urine causes oxalates to become less soluble[15]. For most patients, the onset of stone development is a rare occurrence. Consequently, regimens that are based on a highly customised study of risk variables and risk periods are the only ones that can be predicted to provide optimal compliance. The data gathered from 24-hour urine collections and other extended urine collections provides only an approximate picture of the period's particular risk variables. Precise urine pH values are, in fact, more common than uncommon. It is vital to give times that are thought to be particularly high risk more analytical attention. A thorough medical history and an examination of risk factors during times when low urine pH and CaOx supersaturation occur together can be used to gather this kind of information. A strategy like this could make it possible to create customised recurrence prevention[16].

These days, it is very appealing to remove kidney stones via flexible ureteroscopy[17] If that is not the best option, percutaneous removal of ever smaller stones can be accomplished using ever-tinier devices. Nonetheless, it would seem reasonable that every treatment choice strikes a balance between the intended course of action and the amount of work necessary to get there. Given the risks associated with the treatments and the likelihood of recurrence, active or pharmaceutical removal of leftover fragments should be taken into consideration. Without a doubt, recurrence preventions require additional development, improvement and customised application. Many people with idiopathic calcium stone disease may be prescribed some general medication once the mechanisms underlying stone formation are better understood. Nonetheless, it makes sense that urologists who treat patients with stones on a daily basis must take a sincere interest in and responsibility for their care if more advancements in the treatment of these patients are to be accomplished. The successful implementation of recurrence prevention methods in conjunction with stone removal is crucial. Both clinical and basic research must prioritise these two facets. In this sense the urologist has a rare opportunity to physically study and document the clinical characteristics of the pathology involved in stone development throughout various endoscopic

procedures. The need of providing patients with stone disease with both medical and surgical therapy in the broadest sense cannot be overstated.

CONCLUSION

A study showed that the mineral composition of kidney stones can be used to judge the damage to the skeletal system in primary hyperparathyroidism. They were characterized by phosphate stones, in the mineral composition of which apatites were found (81.1%) (hydroxylapatite, apatite, whitlockite, brushite, struvite-carbonate-apatite). Researchers who studied the composition and structure of urinary stones, including recurrent, noticed one characteristic feature: in relapses of urolithiasis the composition of the cameos was often phosphate. The stones, consisting of apatites, were coral and multiple. Kidney stones, consisting of calcium oxalate monohydrate, calcium oxalate dihydrate, ammonium urate, ammonium dihydrate, struvite, vitlocite, brushite, were characteristic of nephrolithiasis in primary hyperparathyroidism without bone damage and amounted to 87.5%.

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