

## Dressing of Stones in the Kidneys-Nephrolithiasis

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**Annotation:** Pulmonary stone disease (urolithiasis) is a chronic, relapsing-prone substance exchange disease characterized by the presence of concretions in the respiratory tract, that is, in the kidney, bladder and urinary tract (stones). Stones are also collected in the kidney from not drinking more fluids, infectious inflammations in the urinary tract, a decrease in the volume of bladder secretions, an increase in the content of stone-forming substances.

**Keywords:** nephrolithiasis, Enzymopathy, Oxaluria, hyperparathyroidism, hypercalciuria.

Kidney stone (the causes of nephrolithiasis are different in everyone, which means that this disease has many causes. According to modern theories, congenital changes in the kidneys and urinary tract occupy a large place among the causes of kidney stone disease. These innate causes can be divided into three types. Congenital alterations of the proximal and distal ducts are enzymopathy (tubulopathy). Hereditary defects in the anatomical development of the urinary tract. Hereditary neurosis and nephrotic syndrome. Enzymopathy (tubulopathy) is understood to arise from deficiency of these enzymes or their complete absence, or to disrupt the work of the renal ducts and to make the metabolism trapped due to these. Tubulopathies that can arise as a result of genetic changes are understood to be an error of metabolism. The following tubulopathies can be distinguished, which are mainly common in the Middle Region and are more relevant to the presence of stones: oxalaturia, cystinuria, aminoaciduria, galactosemia and less common lactozemia and ricketsimonic diseases. Although uraturia is common, its mechanism has not yet been fully determined. In tubulopathies, substances that participate in the formation of stones in the kidneys are collected. In the kidneys and urinary tract of a person, mainly oxalate is made up of calcium, uric acid, magnesium, ammonium phosphate, Cysteine. It should be noted that oxalaturia, cystinuria, an advanced type of aminoaciduria, changes in sugar metabolism can be not only congenital, but also acquired after, for example: occur as a result of a violation of their function in diseases of the kidneys and liver (cholecystitis, hepatitis, pyelonephritis, glomerulonephritis, etc.). In such cases, the reciprocal relationship of congenital and then acquired tubulopathies occupies a special place. For example: when pyelonephritis interferes in addition to congenital oxaluria, there is a possibility of becoming uraturian. As a result, even in both kidneys of the same patient, which are different in the composition of the kidneys, stones can appear, the composition of which does not resemble each other. That is, Ca and are neither synergistic elements to each other nor the more consumed Ca increases. Excess Ca is excreted into the external environment through urine. Based on hypercalciuric acidosis, the following three causes: 1. Poor absorption of Ca in the intestine; 2. Detachment of Ca from bone due to pathological processes; 3. The kidney is considered to be unable to rank the amount of Ca. The listed factors are  $C_3(PO_4)_2$ ,  $CaC_2O_4$ ,  $CaCO_3$  containing rocks. The most common of these are  $CaC_2O_4$  type rocks. The stones are initially in small measurements (1mm to 3 cm), the sum of salts is the case, and in people who adhere to a healthy lifestyle, the formation of peshobalate Stones is due to the fact that the pH in the urine changes around 5.1 – 5.9. Among those who live in noxias with less magnesium content in water and food, oxalate stones are more likely to be formed. The stronger the renal inflammatory process, the higher the amount of oxaluria. Oxaluria and pyelonephritis occur in the Carib half of patients suffering from kidney stones. Oxaluria is one of the most common tubulopathies in the middle region. The fact that it is also found in the caraltosh seeds of the patient testifies to the fact that it is a hereditary disease. Taajublisi is that, despite oxaluria, only 1 part of the kidney stones will be composed of calcium oxalite, while the rest will be composed of phosphate mixed i.e. oxalate and phosphate salts. The appearance of phosphate stones is due to increased function of the thyroid glands, secondary

hyperparathyroidism. This is the result of disruption of phosphate reabsorption. This is caused by the head by the deposition of oxalate crystals in the kidney tissue or by disruption of phosphate reuptake caused by tissue inflammation. The formation of oxalate stones is due to a change in the pH in the urine around 5.1 – 5.9. Among those who live in noxias with less magnesium content in water and food, oxalate stones are more likely to be formed. The stronger the renal inflammatory process, the higher the amount of oxaluria. Uraturia occurs in 1/4 of patients with kidney stones, and sometimes in the relatives of patients, especially in their men. The disease is caused by a violation of the synthesis of purine nucleotides. The last product of purine metabolism is uric acid. It is secreted by the secretions of the ducts and the filtrations of the balls, and is also reabsorbed in the ducts. The excretion of uric acid does not exceed an average of 800 mg % per day. The appearance of uraturia occurs in two ways: from a violation of the synthesis of purines, which enhances the appearance of uric acid (in such cases, in addition to uraturia, the volume of uric acid in the blood also increases), as well as when the reabsorption of uric acid in the renal ducts decreases. The appearance of large amounts of uric acid occurs in cases of excessive breakdown of nucleotides as well as in pyelonephritis.

Vitamin D: biochemistry, metabolic pathways, physiological role and regulatory mechanisms vitamin D belongs to the fat-soluble group. However, it is not a vitamin in the classical sense of the term, since it has various biological effects through its interaction with specific receptors located in the cell nuclei of many tissues and organs. In this context, the active metabolite of vitamin D acts as real hormone, so it received the latter name "D-hormone". But in keeping with historical traditions, in the scientific literature it is called vitamin D. Vitamin D is naturally only available in a very limited amount of food. In the human body, it is produced only under certain conditions, when ultraviolet (UV) rays of the sun fall on the skin. Biological inert vitamin D, formed by exposure to The Sun, obtained as food and food additives, the body must undergo two processes to activate hydroxylation. The first occurs in the liver and converts vitamin D to 25-hydroxyvitamine d – 25 (OH) d, also known as calcidiol – less active (Depot) the second hydroxylation occurs mainly in the kidneys and its result is the synthesis of physiologically active 1,25-dihydroxyvitamine d – 1,25 (OH)<sub>2</sub> D or calcitriol. Therefore, in addition to external causes in the development of the maxillary foci of urinary stone disease, the state of the human body, pathogenetic internal factors also undoubtedly occupy a large place. One of the edge factors that cause phosphorus calcium metabolism at the origin of kidney stone, the increased function of the thyroid gland hyperparathyroidism occupies the main place. Hyperparathyroidism is not a congenital disease. The separation of phosphates is known to be under the control of shields or malaria. The hormone of pre-Kalkan malaria (paratgarmon) plays two different roles in calcium metabolism, on the one hand it enhances the excretion of phosphorus and reduces its reabsorption in the wings, and on the other it enhances the excretion of calcium salts from bone tissue. The increase in the excretion of phosphates is mutannosive to an increase in paratgarmon in the blood. The infestation of phosphates causes phosphate compounds to precipitate from the bones. Since it is also made up of phosphatbiricmari calcium salts, the calcium substance secretes a lot and its amount in the blood and urine increases. The result is phosphaturia. Hyperparathyroidism is primary as well as secondary. Primary hyperparathyroidism (adenoma of the calf glands) is characterized by an increase in the level of calcium in the blood and urine, increased activity of phosphaturia, shelochnaya phosphatase, a decrease in the process of thickening in the kidney. Primary hyperparathyroidism is not particularly high (1-2%) in patients with kidney stones. Secondary or restored hyperparathyroidism is a consequence of the process in the kidney. It can also be observed in tubulopathies that go without an inflammatory process, even in the case of undamaged kidney stone disease. Such damage to the kidney leads to a violation of the reabsorption of phosphates and calcium in the renal ducts, which increases the function of the shield or glands, and the clotting of the phosphate and calcium of the bone of its ventricles. Secondary hyperparathyroidism is differentiated from monolayer by hypercalcemia. Kidney colic caused by a stone often begins in emergency during and after hard work, when a lot of fluid is consumed during long walks. In the lumbar area, sharp pains appear under the bladder, which gradually occupy the abdomen. The patient is overexcited, crumpled – crumpled, Ox-woe pulls, even yells incessantly. Observing this condition of the patient can determine the disease even at a distance from him. The pain stops from time to time, it can last for several hours or even a few days. After

sharp pains, nausea, notes, sometimes urination is accelerated. In some patients, reflector paralysis of the intestines, Hojat interruption, tension of the muscles of the abdominal walls may occur. It can occur during renal colic as a result of disruption of the functioning of the stomach, intestinal tract, tickling of the posterior lining of the abdomen that covers the kidney, as well as the inner abdominal organs, the neck part to the nerve fibers that connect the kidneys to the nerve nodes. Sometimes, during renal colic, it can be observed that the stone is porous to the urine, as well as a decrease in urine excretion due to the large amount of fluid excretion in the patient. There may also be anesthesia during kidney colic, dry mouth, headaches, elevated temperature, and other common signs. During renal colic, it can be determined that there is severe pain when the patient's rib is palpated below, the anterior wall is tense, there is a Pasternatsky sign. When identifying a Pasternatsky sign during a kidney colic, it is not suitable to coarsely dice the kidney area so as not to aggravate the pain. If the stone is located in the urethra pain in the lower abdomen it is precisely opposite the place where the stone is located that is determined. The increase in the patient's temperature during renal colic, and the increase in leukocytes in the blood, is due to the appearance of reflexes. As soon as the immature stones come out with urine, the kidney colic also relaxes. If the Stone does not separate, the renal colic can be repeated. Preparations with bacteriostatic, spasmolytic and sedative properties (cystinal, artemisole, enatin, etc.) are used in order to ensure that the stones that have given themselves the opportunity to dissociate are released faster. Cystinal, Ursosan 4-5 drops into a small piece of sugar 3 times a day before meals, in the case of avisan 0.05 tablets with enatin 1 gr capsule, consumed 3-4 times a night. Extract of the Marena kravilnoy Sukhoi plant also has the properties of urinating, spasmolytic, makes urine bitter, 2-3 tablets (0.25 gr) are consumed in a glass of warm water, In order to make urine bitter, 10-15 drops of chloristovodorod(solyannaya) acid are added to 1 glass of water, often taken with meals 3-4 times, from benzoic acid 0.05 gr 2 times a day, 0.5 gr ammonium chloride, 5-6 times a day. Recently, various rock solvents have been in heavy use. Patients with urate stones are known to shrink the size of the stones when they consume these substances, sometimes completely melting away, vitamin B6 0.02 g from Medicamentoses 3 per day, drunk for 1 month or 1 ml of 5% solution per day ora m/o is done, one course consists of 15 injections and is carried out several times a year, magnesium oxide 0.3 g 3 per day, drunk for 1 month. In hypercalcuria, thiazides (hydrochlorothiazide) are prescribed – the course of treatment is 1 month, diphosphonates the course of treatment is 1 month. When the metabolism of Shavel acid is disrupted, and in calcium-oxalate stones, the diet is aimed at limiting the introduction of shavel acid into the body: salad, spinat, shavel is prohibited, the consumption of potatoes, carrots, milk, cheese, chocolate, black currants, strawberries, bitter tea, cocoa is limited. Good results can be achieved if drugs such as histone ,phytolysin, urolesan, potassium citrate, lespenefril, cystenal, olimethine, avisan, dinefro,nephrazone are used.For example histone combined herbal preparation. It has diuretic, antispasmodic, litholytic, antimicrobial and anti-inflammatory effects. The drug regulates the Crystal-colloidal balance in dysmetabolic nephropathy, reduces the concentration of elements in the urine that contribute to the formation of stones (oxalic acid, calcium, hydroxyproline). Increases the level of elements that inhibit the process of formation of stone (sodium, magnesium, potassium). Acting on Musin, the drug helps to break down Stones, leads to their demineralization. It prevents the accumulation of particles around the core of the stone, which prevents its further growth. By stimulating urination and relaxing the smooth muscles of the urinary tract, histone helps remove oxalate and phosphate salts, uric acid and small stones from the urinary tract. Histone has bacteriostatic and bactericidal effects, especially associated with Klebsiella spp., Pseudomonas aeruginosa, Escherichia coli, and other gram-negative bacteria. Potassium citrate is a remedy that compensates for potassium deficiency in the body. Helps maintain the required internal and extracellular potassium levels. Potassium is the main intracellular ion and plays an important role in regulating various functions of the body. It is involved in the maintenance of osmotic pressure within the cell, in the processes of transfer and transmission of nerve impulse to innervated organs, in the contraction of skeletal muscles and in a number of biochemical processes. Reduces the excitability and permeability of the myocardium, inhibits in high doses - automatism. Potassium citrate hydroxides urine. As an additional remedy: it is used in infectious diseases of the urinary tract, in the early stages of uricosuric therapy with gout, to prevent the appearance of stones in the urinary tract and renal

tubules. Citrate is recognized as one of the inhibitors of urinary stone formation. If urine citrate and pH with potassium citrate could be elevated, recurrence of stone formation could be prevented. Urocit-K is a slow-release drug of potassium citrate. In conclusion, Urocit-K is an excellent preparation of potassium citrate and a good choice to prevent stone recurrence. Stone Drive with medicinal preparations is distinguished by high efficiency in the therapy complex. In order to increase the effectiveness of treatment, it is necessary to increase the fluid to 2 liters per day. Consuming 2 or more liters of liquid a day can help the natural washing of salts as well as the movement of small stones along the urethra. Regular physical activity: shaking the body while walking, jogging or jumping contributes to the release of small stones, "sand". Phytotherapy: small stones, a layer of oxalate plaque on the surface of cups can be regularly tried to remove by using one of the following methods - Birch buds and leaves, kidney tea, black elderberry flowers, fragrant purple roots, pear, decoction of beech bark. Every 2 weeks, it is better to change the herbal remedy so that tolerance does not appear. All these plants are capable of dissolving oxalates to some extent at the initial stage.

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