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Impact & Causes of Urinary Stone: A Review

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Abstract

The process of stone formation in the kidneys, bladder and/or urethra (urinary tract) is called urolithiasis. Men are twice as likely to produce stones as women. Stone obstruction of the ureters or renal pelvis is characterized by excruciating, intermittent pain radiating from the flank to the groin or genital area and the inner thigh. Rock types are named according to their mineral composition. The most common minerals are struvite (magnesium ammonium phosphate), calcium oxalate, urate, cystine and silica. The most abundant rock on Earth contains calcium. Protection measures depend on the type of stone.

Keywords: Urethra, Struvite, Calcium Oxalate, Urate, Silicate, Cystine

Introduction

Stones that form in the urine (i.e., in the kidneys, ureters, bladder, or urethra) are called urolithiasis. "Urolithiasis" = pebon (urine) and lithos (stone). Urinary stones are one of the most important causes of urinary tract infections and a significant source of morbidity. Stone formation is a urinary tract disorder that affects approximately 12% of the world's population and has a recurrence rate of 70-81% in men and 47-60% in women.[1] It is estimated that stone formation occurs in at least 10% of stone patients. People in industrialized parts of the world suffer from problems with urinary stone formation. The incidence of kidney stones is lower in the south than in other regions.[2] Kidney stones are three times more common in men than in women, due to the stimulation of testosterone and the stone-inhibiting power of estrogen.[3] Urinary stones B.C. It has been found not only in the tombs of Egyptian mummies since 4000 B.C., but also in the tombs of Egyptian mummies. It has also been found in North American Indian graves from 1500 to 1000 BC.[4] The creation of the stone is also recorded in early Sanskrit texts between 3000 BC and 2000 BC.[5] The problem of stone formation is considered a medical problem due to its multifactorial etiology and high recurrence rate. Stone formation also results from

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incompatibility between promoters and inhibitors. Due to the power of testosterone and the suppressive effect of estrogen on stone formation, its incidence in men is three times higher than in women. [6]

Types of Urolithiasis

Urolithiasis Types Stone types are named according to their mineral composition. . The most common minerals are Struvite (magnesium ammonium phosphate), calcium oxalate, urate, cystine and silica.[7]

Name of stone	Approximate incidence	Constituents
Calcium oxalate	70 % of all stones	Calcium, oxalate
Calcium phosphate	10 % of all stones	Calcium, phosphate
Uric acid	5-10 % of all stones	Uric acid
Struvite	10 % of all stones	Calcium, ammonia, phosphate
Cystine	Less than 1% of all stones	Cystine
Medication- induced stones	Less than 1% of allstones	Composition depends on medication orherbal m product(examples include indinavir, ephedrine, guaifenesin, silica)

Calcium oxalate stones

The most common type of kidney stones worldwide contains calcium. For example, calcium-containing stones represent about 80% of all cases in the United States; thesetypically contain calcium oxalate either alone or in combination with calcium phosphate in the form of apatite orbrushite.[8, 9] Factors that promote the precipitation of oxalate crystals in the urine, such as primary hyperoxaluria, are associated with the development of calcium oxalate stones.[10] The formation of calcium phosphate stones is associated with conditions such as hyperparathyroidism[11] and renal tubular acidosis.[12]

Oxaluria is increased in patients with certain bowel diseases, including bowel diseases such as Crohn's disease, or in patients who have had a tubectomy. Small intestine or small bowel bypass surgery. Oxaluria may also be increased in patients with increased oxalate intake (found in vegetables and nuts). Primary hyperoxaluria is a rare autosomal recessive genetic disease that usually occurs in childhood.[13]

Calcium oxalate crystals in urine are the most common form of human kidney stones, and the formation of calcium oxalate crystals is one of the toxic ethylene glycols. Hydrated forms of this compound occur naturally as three minerals: calcite (monohydrate known from some coal seams), wisterite (dihydrate), and a rare trihydrate called Grass oxidation mineral. Most crystals resemble six-sided prisms and often resemble pickets on a wooden fence. More than 90% of the crystals in the urine sediment will have this form. These other shapes are less common than hexagonal prisms, but are important to know quickly in an emergency.[14]

Struvite stones

About 10–15% of urinary calculi are composed of struvite (ammonium magnesium phosphate, $NH4MgPO4•6H2O$).[15] Struvite stones (also known as “bacterial stones,” urease or triphosphate stones) are produced by bacteria, usually in the presence of ureolytic disease. These bacteria metabolize urea into ammonia and carbon dioxide using the urease enzyme. This alkalizes the urine, creating favorable conditions for struvite formation. Proteus mirabilis, Proteus vulgaris and Morganella morganella are the most frequently isolated bacteria; Less common pathogens include Ureaplasma urealyticum and Providencia, Klebsiella, Serratia, and some Enterobacteriaceae species. Stones with this disease usually occur in people with spinal cord and other bladder diseases, urinary tract infections such as ileal conduit urinary

diversion, vesicoureteral reflux, and obstructive urinary disease. They also occur in people with metabolic diseases such as idiopathic hypercalciuria, hyperparathyroidism, and gout. Infected stones can grow rapidly and form large calyx horn (horn-shaped) stones, and complete treatment requires surgery such as percutaneous nephrolithotomy.[16] Struvite stones (triple phosphate/magnesium ammoniumphosphate) have a'coffin lid' morphology by microscopy.[17] Magnesium, ammonium and phosphorus are the building blocks for the formation of struvite crystals in urine. In addition, urine pH and its influence on the concentration of trivalent ionic phosphate (PO4-3) play a key role in struvite crystallization. As urine pH increases, H3PO4, H2PO4-1and HPO4-2are rapidly deprotonated (i.e., removal of hydrogen ions) increasing the concentration of PO4-3, a principal component and driving force for struvite crystal formation.

Uric acid stones

Approximately 5-10% of stones consist of uric acid. [18] People with certain metabolic abnormalities; It can form [19] uric acid stones, including obesity. Their occurrence is also associated with conditions that cause hyperuricosuria (too much uric acid in the urine) with or without hyperuricemia (too much uric acid in the blood). Their formation can also be associated with problems with acid/base metabolism in the urine (low pH), causing uric acid crystals to precipitate. The diagnosis of uric acid urolithiasis is supported by the presence of radioactive stones in the place of regular urine and the presence of uric acid crystals in fresh urine.[20] As mentioned above (calcium oxalate stones), patients with intestinal diseases (Crohn's disease, ulcerative colitis) are prone to hyperoxaluria and oxalate cyst stone formation. These patients also have a tendency to form urate stones. Urate stones are especially common after colectomy. Urate stones usually form as rhombic-shaped polymorphic crystals. They can also appear as polarizable squares or bars. [21] Patients with hyperuricosuria can be treated with allopurinol, which reduces urate production. Alkalinization of the urine can also help with this. Patients with hyperuricosuria can be treated with allopurinol, which reduces urate production. Urine alkalinization may also be helpful in this situation.

Cystine stones

Cystine kidney stones are caused by cystinuria, a disorder in the (genetic) transport of amino acids (the building blocks of amino acids). The protein called cystine) causes excess cystine in the urine (cystinuria) and the formation of cystine stones. Cystinuria is an abnormality in amino acid transport. Although cystine is not the only amino acid oversecreted in cystinuria, it is the least soluble of all natural amino acids. Cystine tends to pass through the urine and form stones in the urine. Small stones are excreted from the body with urine. However, large stones in the kidneys (nephrolithiasis) block the urine, and when the stones pass from the kidneys to the ureters and enter there, the urinary tract obstruction continues. The pressure in the ureters and kidneys increases. It causes dilatation (widening) of the ureters and compression of the kidney. Blockages can also cause urine to stagnate (not move), which can lead to urinary tract infections. Kidney stress and urinary tract infections can cause kidney damage. The damage can progress to renal insufficiency and end-stage kidney disease, requiring renal dialysis or a transplant.[22]

The stone are responsible for all the signs and symptomsof cystinuria, including:

- Hematuria -- blood in the urine
- Flank pain -- pain in the side, due to kidney pain
- Renal colic - intense, cramping pain due to stones in theurinary tract
- Obstructive uropathy -- urinary tract disease due toobstruction
- Urinary tract infections

Silicate stones or drug induced stones

Very rarely, stones can form as a result of taking certain medications or herbal products and the subsequent build- up of chemicals from those products in the urine. Some of these are Loop diuretics, Acetazolamide, Topiramate, Zonisamide, Laxatives (when abused), Ciprofloxacin, Sulfa medications, Triamterene,

Indinavir, Ephedrine, Guaifenesin, and products containing silica.[23]

Causes of urolithiasis

Dietary factors that increase the risk of stone formation include low fluid intake and high dietary intake of animal protein, sodium, refined sugars, fructose and high fructose corn syrup[24], oxalate[19], grapefruit juice, apple juice, and cola drinks. Stone formation commonly occur due to inadequate urinary drainage, foreign bodies in urinary tract, microbial infections, diet with excess oxalates and calcium, vitamin abnormalities like vitamin A deficiencies, excess vitamin D, and metabolic diseases like hyperthyroidism, cystinuria, gout, intestinal dysfunction etc.[25]

Calcium

Calcium is one component of the most common type of human kidney stones, calcium oxalate. Unlike supplemental calcium, high intakes of dietary calcium do not appear to cause kidney stones and may actually protect against their development.[19, 26] This is perhaps related to the role of calcium in binding ingested oxalate in the gastrointestinal tract. As the amount of calcium intake decreases, the amount of oxalate available for absorption into the bloodstream increases; this oxalate is then excreted in greater amounts into the urine by the kidneys. Urinary oxalate is a very potent catalyst of calcium oxalate precipitation, approximately 15 times more abundant than calcium. Other Electrolyte Besides calcium, other electrolytes also appear to affect kidney stone formation. For example, a high-sodium diet may increase the risk of stone formation by increasing urinary calcium excretion. [19] Fluoridation of drinking water may increase the risk of kidney stone formation through a similar process, but more research is needed to determine whether the water contains fluoride. Drinking is associated with kidney stones.[27] On the other hand, dietary potassium appears to reduce the risk of stone formation because potassium promotes the excretion of citrate, an inhibitor of urinary crystal formation. Dietary magnesium intake also appears to reduce the risk of stone formation to some extent because, like citrate, magnesium is an inhibitor of crystal formation in the urine.[19]

Vitamins

Although there is a belief in the medical community that taking vitamin C supplements is associated with an increased incidence of kidney stones[28].There is no evidence of a relationship between vitamin C and vitamin C and kidney stones. Results. Although taking too much vitamin C increases the risk of calcium oxalate stone formation, this rarely occurs in practice. The link between vitamin D intake and kidney stones is also strong. Taking too much vitamin D may increase the risk of stone formation by increasing calcium absorption from the gut, but there is no evidence that treating vitamin D deficiency increases the risk of stones.[19]

Other

There is no confirmed data proving the relationship between alcohol consumption and kidney stones. However, some believe that certain habits associated with consuming too much water and alcohol can lead to dehydration, which can lead to kidney stones.[29] The American Urological Association predicted that global warming will increase the incidence of kidney stones. Reducing the number of kidney stones in the United States by expanding “kidney stone use” in the southern United States.[30]

Supersaturation of urine

When urine is supersaturated with one or more of the calcigenic (crystal-forming) substances (the urine may be in solution when it is heavier than the amount of solvent in the urine) the substances come together through the nucleation process. forms crystals. Heterogeneous nucleation (which has a solid surface on which the crystal can grow) is faster than homogeneous nucleation (where the crystal must grow in a liquid medium without a surface) because it must be less strong. Cysts form cells of the renal papilla and may grow and collect in clusters. The stone formation process may be accelerated when the urine pH is abnormally high or low, depending on the chemical composition of the crystals. [21]The degree of

supersaturation of calcium-producing compounds in urine is related to pH. For example, at pH 7.0, the solubility of uric acid in urine is 158 mg/100 ml. Lowering the pH to 5.0 reduces the solubility of uric acid to less than 8 mg/100 ml. The formation of uric acid stones requires a combination of hyperuricosuria (high uric acid levels) and low urine pH; If the urine pH is alkaline, hyperuricosuria itself is not associated with uric acid stone formation. Supersaturated urine is necessary but not sufficient for the formation of urinary stones. Supersaturation may be the primary cause of uric acid and cystine stones, but calcium-based stones (especially calcium oxalate stones) may have a complex etiology. [32]

Inhibitors of stone formation

Most urine contains chelating substances such as citrate, which inhibit the nucleation, growth, and aggregation of calcium-containing crystals. Other endogenous inhibitors include calgranulin (an S-100 calcium binding protein), Tamm-Horsfall proteins, glycosaminoglycans, uropontin (a form of osteopontin), nephrocalcin (Acidic glycoprotein), prothrombin F1 peptide, and Bikunin (uronic acid-rich protein). . The biochemical mechanism of this drug has not been fully elucidated. However, when these particles fall below normal, the rocks coalesce to form rocks. Kidney stones are often caused by a combination rather than a single obvious one. People who eat a diet high in protein and low in water or calcium are more likely to form stones. They may result from metabolic diseases such as distal renal tubular acidosis, Dent disease, hyperparathyroidism, primary hyperoxaluria, or medullary sponge kidney. In fact, studies show that 3% to 20% of kidney stone patients have medullary sponge kidney. Kidney stones also occur more frequently in people with Crohn's disease. People with recurrent kidney stones are often screened for these conditions. This is usually done by collecting urine for 24 hours for chemical analysis to detect deficiencies and excesses that promote stone formation. [33]

Conclusion

In this review, the types and causes of urolithiasis are explained. Urolithiasis. Calcium oxalate deposition of all minerals is the main cause of urolithiasis. The result is three times more common in men than in women. Urolithiasis is less common in the southern region than in other regions. Urolithiasis usually occurs in people who eat a diet high in animal protein or who do not get enough water or calcium.

References

1. Soundararajan P, Mahesh R, Ramesh T, Hazeena Begum V. Prevention of chronic calcium oxalate urolithiasis in patients with Aerva Lanata. Indian Journal of Experimental Biology, 44, 2006, 981-986.
2. Rana Gopal Singh, Sanjeev Kumar Behura, Rakesh Kumar. Litholytic properties of Kulattha (Dolichous Biflorus) versus potassium citrate in kidney stone disease: a comparative study. JAPI, 58, 2010, 287.
3. Kalpana Devi V, Baskar R, Varalakshmi P. Biochemical effects in normal and rock-fed mice treated with ripe banana (Musa Paradisiaca) seed juice. Ancient Life Sciences, 3 & 4, 1993, 451 – 461.
4. Bahuguna YM, Rawat MSM, Juya V, Gnanarajan G. Antilytic effects of Eleusine Coracana granules. Saudi Arabian Journal of Pharmaceutical Sciences, 17, 2009, 182.
5. Surendra K. Pareta, Kartik Chandra Patra, Ranjit Havansh. Inhibition of calcium oxalate crystallization by Achyranthes bidentata in vitro. Hydroalcoholic Extract: A way to prevent stones. International Journal of Pharmacy and Biological Sciences, 432.
6. Kalpana Devi V, Baskar R, Varalakshmi P. Biochemical effects of treatment with ripe juice of plantain (Musa Paradisiaca) in normal rats and stone-forming rats. Ancient Life Sciences, 3 and 4, 1993, 451 – 461.
7. Türk, C. (Chairman), T. K. c. (2011). Recipe for urinary stones. Updated March 2011, 1-103.
8. Riley Jr. RF, Çekim 13: Pop Kuresi, s. 14. 192–207 Riley, Jr. . Thiab Perazzella (2005)

1. 9 Coy, Florida; Evan , A ; Worcester , E (2005). Clinical Nephrolithiasis Umut, B; Longman , CB (2003). “United States Study of Diagnosis, Treatment, and Outcomes of Primary Hyperoxaluria.” Pediatric Nephrology 18(10): 986–91. Research 115(10): 2598- 2608
9. Umut, B; Longman, C. B. (2003). “United States Study of Diagnosis, Treatment, and Outcomes of Primary Hyperoxaluria.” Pediatric Nephrology 18(10): 986–91.
10. National Endocrine and Metabolic Disease Information Service (2006). “Hyperparathyroidism (NIH Publication No. 6-3425).”
11. National Endocrine and Metabolic Disease Information Service (2008). “Renal tubular acidosis (NIH Publication No. 09–4696)”
12. Demes, Daniel. ASCP Rapid Compendium of Clinical Pathology, 2nd ed. ASCP Press, Chicago, 2009.
13. Clinical pathology of ethylene glycol poisoning. Archived from the original on 2 May 2012. Access date: 2012-05
14. Carl A. Osborne, D.P. (n.d.). University of Minnesota Minnesota Urolithiasis Center. Monitor CaOx to prevent uroliths, 1-2.
15. Weiss, M.; Liapis, H; Tomaszewski, J.E.; Arend, L. J. (2007). "Chapter 22: Pyelonephritis and related diseases, reflux nephropathy, hydronephrosis and nephrolithiasis." Janet in JC; Olson, J.L.; Schwartz, M. M. et al. Heptinstall
16. Demes, Daniel. ASCP Rapid Compendium of Clinical Pathology, 2nd ed. ASCP Press, Chicago, 2009.
17. Mo, O.W. (2006). "Kidney stones: pathophysiology and clinical management." Lancet 367(9507): 333–44.
18. Chory, N; Cooper B, Robertson W, Choong S, Rickards D, Unwin R (2010). "The Least Practical Guide to Kidney Stone Management". Nephron Clin Appl 116(3):c159–71
19. Reilly Jr. RF, Tshooj 13: Public Environment, p. 107-1 192–207, Reilly Jr. Thiab Perazella (2005)
20. Demes, Daniel. ASCP Rapid Compendium of Clinical Pathology, 2nd ed. ASCP Press, Chicago, 2009.
21. Eric Fjerstedt; Hanevik Lotta; Jepsen Jan-Olov; Hans-Goran Tiselius; Peter Soderqvist; Toth Dondenenberg (2003). "Urinary excretion of total cystine and the diamines arginine, lysine, and ornithine is associated with genetic findings in cystinuria patients treated with sulfhydryl compounds." Urology Research 31(6):417–25.
22. Osborne CA: Analysis of 451,891 canine uroliths, feline uroliths, and feline urethral plugs, 1981-2007: Minnesota Urolithiasis Center Report: VCNA 2008; 391: 83.
23. Knight J, Assimos DG, Easter L, Holmes RP (2010). "Fructose is metabolized into oxalic acid and glycolic acid."
24. Suman Kumar Mekap, Satyaranjan Mishra, Sabuj Sahoo and Prasana Kumar Panda. Anti-urolytic activity of Crataeva magna Lour. tough. Indian Journal of Natural Products and Resources, 1(2), 2011, 28-33.
25. Dietary Committee Reference Intakes for Vitamin D and Calcium, Tolerable Upper Intake Levels: Calcium and Vitamin D, p. 403-56 Committee to Review Dietary Reference Intakes for Vitamin D and Calcium (2011)
26. National Academies Committee on Fluoride in Drinking Water (2006). "Chapter 9: Effects on the Kidneys". Fluoride in Drinking Water: A Scientific Review of EPA Standards. Washington, DC: National Academies Press. 236-48: I.
27. Goodwin, J.S.; Mangum, M. R. (1998). “Challenging Quackery: Attitudes Toward Micronutrient Supplements in American Academic Medicine.” Archives of Internal Medicine 158(20): 2187–91. doi:10.1001/archinte.158.20.2187.
28. Rodman, J.S.; Seidman, C. (1996). "Chapter 8: Eating Problems". Rodman, J.S.; Seidman, C.; Jones, R. No More Kidney Stones (1st ed.). New York: John Wiley &

Sons, Inc. s. 100-1 100–1 46–57. ISBN 978-0-471-12587-7.

29. Brawer, MK; Makarov, DV; Partin, AW; Roehrborn, CG; Nickel, JC; Lu, SH; Yoshimura, N; Chancellor, MB et al. (2008). "Best of the 2008 AUA Annual Meeting: Highlights from the 2008 Annual Meeting of the American Urological Association, May 17–22, 2008, Orlando, FL". Reviews in Urology 10 (2): 136–56. PMC 2483319. PMID 18660856.
30. Reilly Jr. RF, Chapter 13: Kidney stones, p. 192-207, Reilly Jr. and Perazella (2005)
31. Umut, B; Longman, C. B. (2003). "United States Study of Diagnosis, Treatment, and Outcomes of Primary Hyperoxaluria." Service of Pediatr Nephrology 18(10): 986–91. Access date: 27 July 2011.
32. National Kidney and Urological Diseases Clearinghouse (2008). "Medullary sponge kidney (NIH publication number 08-6235)." Kidney and urinary tract diseases: List of topics and names from A to Z. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Public Health Service, US Department of Health and Human Services. Tau txais Lub Xya Hli 27, 2011.

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