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THE ROLE OF THE NEPHROLOGIST IN THE TREATMENT AND PREVENTION OF NEPHROLITHIASIS

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Abstract

Nephrolithiasis is a disease with a high risk of recurrence: 50% within five years and up to 80–90 % within 10 years after the first episode. Often, with this diagnosis, there is only symptomatic treatment with the aim of expelling the kidney stone and removing the pain. In such patients, however, initial stratification of the risk of recurrence based on the risk factors present is essential. Every patient with nephrolithiasis, whether it is a single episode or recurrent, should follow general measures to prevent kidney stones. Depending on the results of the kidney stone analysis and the risk of recurrence, it is necessary to carry out a specialised nephrological metabolic examination which is performed by a nephrologist in Slovakia, with subsequent personalised recommendations depending on the cause of recurrent nephrolithiasis. These include dietary and regimen measures, as well as pharmacological treatment.

In the following article we summarize specific measures for individual types of nephrolithiasis, as well as basic preventive measures for the recurrence of kidney stones, with a focus on dietary risk and protective factors.
Keywords: recurrent nephrolithiasis, prophylaxis, nephrology, specific treatment

LIST OF ABBREVIATIONS

CaOX calcium-oxalate
RTA renal tubular acidosis
UTI urinary tract infection

INTRODUCTION

Nephrolithiasis is a kidney disease in which urinary stones form in the uropoietic system. The individual probability of developing nephrolithiasis during life is 5–10% (1, 2). The risk of recurrence of nephrolithiasis from the first episode is 50% within five years and up to 80–90% within 10 years (3). Compared to individuals with a single episode of nephrolithiasis, patients with recurrent nephrolithiasis are more susceptible to severe urinary metabolic abnormalities (4). Considering the above, the issue of recurrent nephrolithiasis is more complex than it might seem at first glance. In a practical sense, nephrolithiasis manifests itself as renal colic with subsequent spontaneous urination of the stone, or the need for a spasmo-analgesic treatment or the need for an urological intervention. From the point of view of the management of such a patient, the treatment is only symptomatic, not causal – aethiology of the kidney stones formation is not treated as well as we do not reduce the probability of recurrence of nephrolithiasis. In the following article we will focus on the

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basic examination of patients with nephrolithiasis, as well as universal preventive dietary measures for preventing recurrent nephrolithiasis.

THE RISK OF RECURRENT NEPHROLITHIASIS STRATIFICATION

To figure out who is most likely to get recurrent nephrolithiasis, the urinary stone must be looked at before the metabolic exam can be done (5). Patients with recurrent nephrolithiasis should perform a urinary calculus analysis at each episode due to a change in urinary stone composition (6, 7). If an analysis of a urinary stone is not possible, it is necessary to take a detailed sample of the patient's dietary habits, history of kidney stones, family history, and current medication used. With diagnostic methods, it is necessary to perform a sonographic examination of the abdomen or a spiral computed tomography. From a blood examination perspective, it is necessary to examine the plasma values of creatinine, total albumin, ionised calcium, and uric acid. It is also necessary to examine urine with a dipstick test for the presence of leukocytes, erythrocytes, nitrites, proteins, pH of urine, and specific gravity. Microbiological culture of urine, urine pH profile (urine pH measurement after each urination, at least four times a day), and microscopic examination of urine are also necessary. Every patient, following the passage of a urinary stone, must implement the same diagnostic algorithm, followed by a stratification of the risk of recurrence of nephrolithiasis. There is a high risk of recurrence of nephrolithiasis in the presence of:

1. General risk factors
   a. early primary manifestation of nephrolithiasis (renal colic in patients < 18 years old),
   b. positive family history of nephrolithiasis,
   c. infectious, brushite, urate kidney stones,
   d. solitary kidney (a solitary kidney is not a risk factor for the recurrence of kidney stones per se, but the prevention of recurrence is crucial in an episode of nephrolithiasis).
2. Diseases associated with a higher risk of nephrolithiasis:
   a. hyperparathyroidism,
   b. nephrocalcinosis,
   c. gastrointestinal diseases (jejuno-ileal bypass, intestinal resections, non-specific intestinal inflammations, malabsorption syndromes) and bariatric surgery
   d. sarcoidosis.
3. Genetically determined diseases:
   a. cystinuria (types A, B, and AB),
   b. renal tubular acidosis (type I),
   c. primary hyperoxaluria,
   d. xanthinuria,
   e. cystic fibrosis.
4. Anatomical abnormalities:
   a. tubular ectasia,
   b. obstruction of the ureteropelvic junction,
   c. stricture of the ureter,
   d. obstructive uropathies (urinary diversion – ileal conduit, prostate diseases, neurogenic dysfunction of the lower urinary tract)
   e. vesicoureteral reflux (8).

Patients with a high risk of nephrolithiasis recurrence should undergo a specific metabolic examination, which involves collecting 24-hour urine twice in a row (9, 10). The timing of a specific metabolic examination is also important. It should be relayed in a period without the occurrence of renal colic, with a minimum 3-week interval between the expulsion of

the kidney stone and the 24-hour urine collection (11). A control metabolic examination should be carried out within a time horizon of 8–12 weeks from the first examination, with follow-up examinations at 12-month intervals in cases of normalisation of urinary parameters (12).

GENERAL MEASURES TO PREVENT THE RECURRENCE OF NEPHROLITHIASIS

Fluid intake

Adequate daily drinking is one of the basic preventive measures of recurrent nephrolithiasis; it recommends at least 2500 to 3000 ml of fluids per day, with a regulation according to current diuresis (optimal daily diuresis of 2000–2500 ml per day), requirements, and extrarenal fluid losses. (13). In practice, this means that the patient needs to take fluids regularly, not abruptly, and should increase their standard fluid intake during periods of sweating, increased physical activity, vomiting, or diarrhea. We can practically verify fluid intake by the value of the specific gravity of urine, the adequate value of which should be lower than 1.010 (13).

In the context of dietary measures, it is important to consider not only the quantity of fluids consumed, but also the quality or type of drinks consumed.

From the point of view of drinking coffee and other caffeinated drinks, the idea that plant-based foods like coffee and tea have a higher risk of calcium oxalate nephroliths was shown to be false. A meta-analysis by Ferraro and colleagues (2014) confirmed reciprocal results: coffee intake reduced the risk of kidney stones (14). The Mendelian randomization study by Yuan and Larsson (2022) also confirmed this, concluding that caffeine and coffee intake reduce the incidence of kidney stones (15). For healthy individuals, except for pregnant women, the European Food Safety Authority considers a daily intake of caffeine up to 400 mg, or around four cups of brewed coffee, to be safe (16). Other possible causes of tea consumption's protective effect include increased overall fluid intake and the antioxidant activity of phytochemicals such as polyphenols (17, 18). In the study by Ferraro et al. (2013), the authors confirmed an increased risk of kidney stones with an increased consumption of cola and non-cola-sweetened beverages. On the other hand, drinking beer reduced the risk of developing nephroliths by up to 41%, wine by 31–33%, and orange juice by up to 13% (19). Interesting results were obtained by a study by Finnish authors (1999) who concluded that each bottle of beer drunk daily reduced the risk of kidney stones by 40% (RR = 0.60, 95% CI 0.47–0.76). Caution is in order, especially in the case of urate kidney stones, as alcohol increases the risk of urinary excretion of urates (20).

The water's hardness is another indicator of the quality of the fluids received. Studies indicate that a calcium ion concentration exceeding 120 mg/l, a sign of water hardness, enhances the overall daily calcium intake. Consuming bicarbonate is a powerful alkalizing agent and enhances the body's ability to function as a buffer. By raising urine pH and citrate excretion, bicarbonate-rich mineral water can assist alkalinization therapy and enhance urine's inhibitory potential (21). In one study the likelihood of calcium oxalate and uric acid stones in the urine was examined in healthy individuals who were randomly assigned to receive either potassium citrate or an equal amount of bicarbonate-rich mineral water (22). Urine pH and citrate excretion were raised, significantly, and oxalate excretion was reduced when 2 litres per day of mineral water containing 1715 mg/l bicarbonate, or 2.55 g/day potassium citrate were consumed. The relative supersaturation of uric acid and calcium oxalate dropped dramatically in both groups. A study of healthy people under controlled dietary settings revealed that bicarbonate, calcium, and magnesium-rich mineral water raised urine pH and enhanced the excretion of citrate, magnesium, and the urinary inhibitors of calcium oxalate stone formation (23).

Citrates

Dietary citrate converts to bicarbonate, and may elevate urine pH and citrate excretion (24). Citrus juices from oranges, lemons, and grapefruits contain large amounts of citrates. These juices may be consumed instead of medication that contains alkalizing agents. There are conflicting results from research on how drinking orange juice affects urinary risk factors for kidney stones. Three cohort studies have linked orange juice consumption to a lower chance of the development of kidney stones (19). Interventional studies done under a controlled eating regimen found that orange juice increased the pH and citrate levels in urine (25–27). The nutritional advice is to choose the whole fruit over the juice, to limit daily consumption of fruit juice to one serving, and to dilute the juice with water due to concerns over the high sugar and energy content as well as the lack of dietary fibre in orange juice (28). A study conducted on healthy individuals revealed that drinking nearly 2 litres of coconut water daily greatly increased the excretion of potassium, chloride, and citrate in the urine while having no effect on the pH (29).

Soft drinks

From the perspective of soft drinks, one study demonstrated a considerable increase in the risk of recurring stone development associated with the consumption of soft drinks, particularly those acidified by phosphoric acid. Consequently, cohort studies also showed a highly favourable correlation between risk of developing gout and soft beverages with added sugar (30, 31). The fructose content of sugar-sweetened soft drinks has been linked to an elevated incidence of incident kidney stone formation, which could account for at least some of these results (32, 33).

Protein intake

To prevent recurring nephrolithiasis, another general preventative approach is to reduce protein intake, with a daily limit of 0.8 to 1.0 g/kg of animal protein. Research suggests that a high-protein diet could negatively affect the urinary risk factors for the development of kidney stones. A high-protein diet can cause an acid load that can lower urine pH and citrate excretion, as well as raise urinary calcium (34, 35). A systematic review linked high-protein diets to increased urinary calcium excretion, a risk factor for the development of calcium stones (36).

Fruit and vegetable consumption

Increasing fruit and vegetable consumption is another dietary suggestion. Extensive observational studies linked an increased risk of stone formation to a larger dietary net acid load (37). These findings suggest that, rather than total protein consumption, the ratio of fruits and vegetables consumed to ingested protein may be a more accurate predictor of urinary stone development. Vegetables and fruits improved urine pH and citrate excretion while decreasing the relative supersaturation of uric acid and calcium oxalate in hypocitraturic stone patients (38). High nutritional proton load or high dietary acidity can lead to a reduced urine pH and citrate excretion, which can increase the risk of numerous types of urinary stones, especially the most frequent ones, which are calcium oxalate and uric acid. The capacity to bind calcium and excrete citrate, which inhibits the formation of stones, increases with urine pH, while the excretion of calcium in the urine decreases (39).

Carbohydrates

The relationship between carbohydrates and the risk of kidney stone production is not conclusive. Many studies have identified a positive correlation between the consumption of sucrose and the likelihood of stone formation in women but not in men (40–43). The increase in calcium excretion following the consumption of glucose orally has been attri-

buted to an augmented absorption of calcium in the intestines and a decrease in the reabsorption of calcium in the renal tubules (44). In recent decades, fructose consumption has significantly increased due to its use as a sweetener in beverages and food, often replacing sucrose or glucose. A comprehensive analysis found a direct correlation between the consumption of fructose and the likelihood of developing kidney stones (32). A study on a group of males found a direct correlation between fructose consumption and the risk of developing gout (31).

Fat intake

There is a lack of data on the relationship between dietary fat consumption and the risk of recurrent nephrolithiasis. The composition of fatty acids in one's diet, particularly the ratio of n-6 to n-3 polyunsaturated fatty acids, can affect the likelihood of developing calcium oxalate stones. Researchers have shown that idiopathic calcium oxalate stone patients have an unusually high level of arachidonic acid (C20:4n-6) in their plasma and erythrocyte membrane phospholipids compared to healthy individuals (45, 46). An increased prostaglandin E2 synthesis is believed to cause hypercalciuria by enhancing calcium absorption in the intestines and promoting bone tissue breakdown (47). The elevated amounts of arachidonic acid in phospholipids can lead to hyperoxaluria, which in turn enhances the transport of oxalate in the intestines and kidneys (48, 49). Urinary oxalate is considered a crucial risk factor for the development of calcium-oxalate stones. Modifications in the concentration of oxalate in urine can greatly enhance the degree of urinary supersaturation of calcium oxalate (50). It originates from the synthesis of oxalate inside the body and the consumption of oxalate through diet (51). Precursors like ascorbic acid and hydroxyproline primarily influence the liver's endogenous oxalate metabolism (52). Plant-based foods predominantly serve as the primary source of dietary oxalate. Dietary oxalate intake estimates vary significantly based on the consumption of foods that are high in oxalate. A precise understanding of the oxalate levels in foods is crucial for the dietary treatment of individuals with calcium oxalate stones. Spinach, rhubarb, white beans, sesame, and liquorice are examples of foods that have a high oxalate content. Food processing and preparation methods have a crucial role in determining the oxalate level due to the potential loss of oxalate into the cooking water during boiling (53). For instance, researchers discovered that the oxalate concentration in raw spinach is over five times greater than that in cooked spinach, with raw spinach containing 1959 mg/100 g and cooked spinach containing 364 mg/100 g (54, 55). Intestinal colonisation by the Gram-negative anaerobic bacterium Oxalobacter formigenes, which degrades oxalate, can be inversely linked to the production of calcium oxalate stones (56).

Calcium intake

Hypercalciuria is a significant predisposing factor for calcium stone development. Evidence has shown that consuming an appropriate amount of calcium from both dairy and non-dairy sources can help reduce the formation of urinary stones (57). To prevent bone loss and excessive oxalate absorption and excretion, dietary calcium limitations must be avoided. Restricting dietary calcium decreases the amount of calcium in the intestines, which increases the absorption of unbound oxalate and thus leads to higher levels of oxalate in the urine (58).

Salt intake

The consumption of dietary sodium chloride increases the risk of stone formation because it tends to increase the excretion of calcium in urine (59). An increased consumption of sodium chloride can lead to the excretion of calcium by preventing the reabsorption of calcium in the renal tubules due to the expansion of extracellular fluid volume caused by sodium.

Interventional investigations conducted on healthy persons have demonstrated that for every 100 mmol (equivalent to 2300 mg) increase in daily sodium consumption there is an approximate rise of 1 mmol in daily calcium excretion (60).

General preventive measures to reduce the risk of the urolithiasis recurrence as well as pharmacological agents to prevent stone formation are summarized in Tables 1 and 2 (61).

Table 1 General preventive measures for kidney stone formation

Fluid intake	Fluid amount: 2500–3000 ml
	Water is the preferred fluid.
	Diuresis: 2000–2500 ml/24 hours
	Specific weight of urine: < 1,010 g/day
Nutritional advice for a balanced diet	Balanced diet – avoid excessive intake of vitamin supplements
	Increase intake of fibre and vegetables
	Optimal calcium intake 1000–1200 g a day
	Limited salt intake < 4–5 g/day
	Limited animal protein intake: 800–1000 mg/day
Lifestyle advice	Normal BMI
	Adequate physical activity
	Avoid excessive fluid loss
	Reduce intake of alcohol
	Reduce intake of calorie-containing fluids

Table 2 Pharmacological substances used for kidney stone prevention

Agent	Rationale	Stone type
Alkaline citrates	Alkalinisation Hypocitraturia Inhibition of calcium oxalate crystallisation	Calcium oxalate Uric acid Cystine
Allopurinol	Hyperuricosuria Hyperuricaemia	Calcium oxalate Uric acid Ammonium urate 2,8-Dihydroxyadenine
Calcium	Enteric hyperoxaluria	Calcium oxalate
Captopril	Cystinuria Active decrease of urinary cystine levels	Cystine
Febuxostad	Hyperuricosuria Hyperuricaemia	Calcium oxalate Uric acid
L-methionine	Urine acidification	Infection stones Ammonium urate Calcium phosphate
Magnesium	Isolated Hypomagnesuria Enteric hyperoxaluria	Calcium oxalate
Sodium bicarbonate	Alkalinisation Hypocitraturia	Uric acid Cystine
Pyridoxine	Primary hyperoxaluria	Calcium oxalate
Thiazide	Hypercalciuria	Calcium oxalate Calcium phosphate
Tiopronin	Cystinuria	Cystine

CONCLUSION

Nephrolithiasis is a diagnosis whose aetiology is often overlooked by either urologists or nephrologists. Due to the high risk of recurrence in patients with a primary attack of renal colic, it is necessary to analyse the presence of risk factors that can be especially removed with the initiation of general, specific pharmacological as well as non-pharmacological measures to reduce the risk of nephrolithiasis and thus improve the quality of life of patients.

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