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REVIEW PAPER

THE IMPACT OF CHEMICAL COMPOUNDS CONTAINED IN A DIET ON THE DEVELOPMENT AND PREVENTION OF UROLITHIASIS*

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Abstract

Urolithiasis is one of the most common diseases of the genitourinary system. The conditions associated with the development of urolithiasis show a wide variety, among which dietary behaviour is of great importance and is nowadays becoming increasingly important in the development of kidney stones. The aim of this study was to review the literature about the impact of chemical compounds contained in a diet on the development and prevention of urolithiasis. Factors impeding the proper absorption of calcium include phosphates, fatty acids, sulphates, citrates, oxalates, while the factor that stimulates the absorption of calcium in the intestines is the active form of vitamin D_3. In this regard, one should consider the amount of calcium, sodium, potassium, animal protein and simple carbohydrates consumed in a diet, as well as dietary habits regarding caffeine and alcohol. Also significant is the fact that a poorly balanced diet may condition hypercalciuria. In the case of excessive consumption of other compounds, such as simple carbohydrates and ethyl alcohol, increased urinary calcium excretion is observed, caused by disturbances in the tubular reabsorption of calcium and magnesium in nephrons. Nowadays, prophylaxis is gaining importance in the light of the growing number of cases of urolithiasis worldwide. A well-designed diet is a crucial element, affecting both the formation of deposits and treatment, hence sound knowledge of the principles of proper nutrition can significantly reduce the risk of the recurrence of renal colic. The most common cause of urolithiasis are nutritional errors, and the onset of treatment is associated with a change in dietary habits. The general nutritional recommendations for urolithiasis should be adapted to the type of urinary stones deposit and an individual patient's comorbidities. A tailored approach to combating the disease is required.

Keywords: urolithiasis, chemical factors, nutrition, diet.

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INTRODUCTION

Urolithiasis is one of the most common diseases of the genitourinary system. According to the literature, the prevalence of urolithiasis in different countries of the world ranges from 1 to 20% (Zawadzki, Imiela 2000, Bartoletti et al. 2007, López, Hoppe 2010, Cook et al. 2016). In Europe, urolithiasis ranges between 5% and 9%, and in Asia the prevalence varies from 1% to 19.1% (Liu et al. 2018, Tzelves et al. 2020). Considering the different dietary conditions encountered in different regions of the world, it should be noted that this disease most often affects societies in which the basic component of the diet is large amounts of meat, while it is rarely found in vegetarians.

The conditions associated with the development of urolithiasis show a wide variety, among which dietary behaviour is of great importance; nowadays this is becoming more and more important in the development of kidney stones (Basavaraj et al. 2007, Worcester, Coe 2010, Jayaraman, Gurusamy 2018, Jobs et al. 2018, Leslie, Sajjad 2019,). Initially, it was thought that a low-calcium diet was appropriate for preventing the formation and recurrence of urinary stones (Goldfarb 1990, Sorensen 2014). Curhan et al. (1993) indicated that low calcium intake potentially increases the risk of stones. This was confirmed by Sromicki and Hess (2020), who demonstrated the ability to reduce oxalate levels in humans by increasing calcium intake (with 20-fold oxalate intake – 2220 mg = 24.7 mmol/day) from 1200 mg to 3859 mg/day (96.5 mmol/day). This equates to a dietary calcium/ /oxalate molar ratio of 3.9 / 1. HPFS, NHS I and NHS II studies confirmed the protective role of increased calcium intake in urolithiasis, emphasizing that this effect has been proved only for dietary calcium (Arumuham et al. 2019). The opposite conclusion about the reduction of the recurrence frequency of urolithiasis in patients after limiting calcium intake was presented at the 5th Meeting of the EAU Section of Urolithiasis (Terzoni et al. 2019).

The aim of this study is to review the literature about the impact of chemical compounds contained in a diet on the development and prevention of urolithiasis.

PATHOGENESIS OF UROLITHIASIS

The development of urolithiasis is a multi-stage process and depends on many factors, among which the micronutrients contained in the food play an important role, thus determining the chemical composition of urinary stones. The supersaturation of urine with a specific type of mineral salts beyond the capabilities of lithogenesis inhibitors (nefrocalcin, bikunin, Tamma-Horsfall protein, osteopontin, citrates, phytic acid residues, magne-

sium ions) or in the metabolic disorders associated with their deficiency result in a more intensive crystallization process (Duława 2009, Saxena, Sharma 2010). Epidemiological differences in the incidence of individual types of urinary stones depend on the specific diet for the population. Research (Wróbel et al. 2009) conducted on the Polish population showed a significant dominance of calcium oxalates and a small percentage of cases of stones composed of hydrocysapatite or struvite. The United Kingdom population data provided by Arumuham et al. (2019) showed the following frequency of particular types of urinary stones: calcium oxalate 80%, struvite (infection related) 2-20%, uric acid 5-10%, calcium phosphate calcium oxalate 10%, cystine 1%, pure calcium phosphate <1%. Different results were presented in the study (Durgawale et al. 2010) covering the Indian population. The most common types of stones were those composed of struvite and calcium oxalate followed by calcium carbonate, uric acid and cystine. The chemical composition of deposits is often heterogeneous, including for example stones formed from calcium oxalate as well as phosphates and uric acid (Durgawale et al. 2010). The process of excessive crystallization of stones formed from calcium oxalate $Ca(C_2O_4) \cdot H_2O$ depends primarily on hypercalcuria (M> 300 mg/d; K> 250 mg/d), and hyperoxaluria (>0.5 mmol/d/ 1.73 m^2) also from hyperuricosuria, hypomagnesemia (<0,7 mmol l^{-1}) and hypocytraturia (<2.5 mmol/d). At the favorable urine pH <5.8, the crystallization process of calcium oxalate and uric acid begins. The occurrence of the presented disorders is predisposed by intestinal absorption disorders (inflammatory bowel diseases, bariatric surgery, short bowel syndrome), primary hyperparathyroidism, pathological bone fractures causing calcium loss, sarcoidosis, multiple myeloma, urinary retention, gout, excessive calcium, vitamins D or C supplementation, a diet rich in oxalates, sodium and protein (Worcester, Coe 2010, Witalis, Filipek 2011, Skolarikos et al. 2015). The pathogenesis of phosphate urolithiasis, commonly called struvite, is associated with the physico-chemical process of mineral precipitation, which is responsible for the infection process caused by urease-positive bacteria: *Proteus, Pseudomonas, Klebsiella, Serratia or Ureaplasma*. Urease alkalizing urine to pH >7.2 (Różański 2010) starts a series of chemical reactions, from the hydrolysis of urea to CO_2 and ammonium groups, increasing the concentration of NH_4^+, PO_4^{3-}, CO_3^{2-}, to the production of struvite crystals $MgNH_4PO_4 \cdot 6H_2O$ carbonate apatite $Ca_5(PO_4)^3 \cdot CO_3$ and hydroxyl $Ca_5(PO_4)^3 \cdot OH$. Chronic persistent elevated urine pH prevents the dissolution of deposits, and the polysaccharide biofilm produced by *Proteus mirabilis* colonies increases protection and hinders the treatment of this type of urolithiasis, often requiring surgical intervention (Witalis, Filipek 2011, Türk et al. 2014). Stones from uric acid are formed in the acidic environment of urine, when its pH is lowered and the excretion of uric acid is increased. Daily uric acid excretion >800 mg in men and >750 mg in women justifies the diagnosis of overproduction. Hyperuricosuria ≥4 mmol/d and low pH <5.5 urine are most often associated with the consumption of large amounts of purines, gout, chronic diarrhea,

metabolic syndrome, insulin resistance, myeloproliferative disorders, tumor lysis syndrome, increased catabolism, less often the Lesch-Nuhan syndrome or the Henneman's defect (Peller 2012, Skolarikos et al. 2015). The pathogenesis of cystine urolithiasis includes poor solubility of cystine in urine, low urine pH, cystinuria (>400 mg/d), genetically conditioned disorder of dibasic amino acid transport in the renal tubules and small intestine. Urolithiasis has a recurrent tendency. Effective and chronic modification of a diet as part of primary and secondary prevention may prove to be crucial and certainly important in the prevention of the crystallization process in combination with appropriate treatment (Saxena, Sharma 2010, Skolarikos et al. 2015).

DIETARY FACTORS IN THE DEVELOPMENT OF UROLITHIASIS

In a high-protein diet, there is also a correlation with the occurrence of this disease, because excessive animal protein consumption contributes to increased calcium and uric acid excretion and reduced excretion of citrates. Moreover, protein is the main source of hydrogen ions, which leads to urine acidification and in turn increases the crystallization of cystine and urate salts (Borghi et al. 2002, Fink et al. 2009). As a result of the metabolic processes that absorbed proteins undergo, acidification occurs in the system, which determines the abnormalities of tubular calcium reabsorption in the kidneys. It is assumed that in healthy people, increased intake of protein in the diet increases urinary calcium excretion by about 0.04 mmol g^{-1} of protein consumed (Osther 2012, Leslie, Sajjad 2019). According to recommendations, the intake of protein of animal origin should be limited to 0.8-1.0 g kg^{-1} of body weight (Borghi et al. 2002, Fink et al. 2009).

Another type of diet, sodium-rich one, increases calcium excretion by reducing sodium reabsorption and secondary calcium reabsorption in the proximal tubule (Martin, Goldfarb 2007). According to Pfau and Knauf (2016), low liquid intake leads to high urine concentrations of lithogenic substances. This is why, the volume and type of fluid intake can be important in the development of urolithiasis. Ferraro et al. (2013) indicate that consumption of coffee, tea, beer, wine and orange juice is associated with a lower risk of nephrolithiasis. In another study, Rahman et al. (2017) indicate that citrus-based products, i.e. lemonade, orange and grapefruit juice, can increase urine citrate and urine pH, which precludes the formation of stones. Moreover, appropriate urine citrate concentrations support the inhibitory properties of Tamm-Horsfall protein. Among the beverages that increase the risk of stone formation, according to Saldana et al. (2007), are beverages acidified with phosphoric acid, which is an ingredient in non-alcoholic carbonated beverages, i.e. well-known cola drinks. Food products containing large amounts of oxalates, which correspond with a high risk of urolithiasis,

include black tea, coffee, chocolate, nuts, rhubarb, spinach, sorrel and strawberries (Duława 2009). Endogenous metabolism may also be a source of urinary oxalate due to glycine, vitamin C, hydroxyproline and glycolate transformation.

In studies by Taylor and Curhan (2008), attention was paid to the unfavourable effect of fructose on the pathogenesis of urinary stone formation. This sugar occurs naturally in fruit, while fructose-glucose syrup has become a very popular sugar substitute. Its presence can be noted in a number of products, e.g. juice, cola, sweetened beverages, ice cream, biscuits, jams, fish salads, bread, jar sauces etc. White et al. (2018) confirmed that after fructose consumption, serum uric acid concentration increases rapidly. Further research is required to assess the observed relationship of increased serum uric acid concentration in the health context. Perhaps a fructose--reducing diet should be used in urolithiasis, without excluding fruit intake, but significantly reducing the intake of low fibre juices and beverages with fructose-glucose syrup added.

DIETARY CALCIUM CONTENT

In the pathogenesis of urolithiasis, most noteworthy of all is the phenomenon of hypercalciuria, defined as urinary calcium excretion exceeding 300 mg per day in men and 250 mg per day in women or more than 4 mg kg^{-1} of body weight per day. Due to the significant relationship of hypercalciuria to the formation of urinary deposits, it is considered important to explore this issue. Calcium metabolism in the human body determines its homeostasis, which is why calcium is one of the macronutrients that should be consumed regularly to ensure the proper course of many pivotal physiological processes. The supply of this element in the diet should be adapted to the needs of individual age groups. The intestinal calcium absorption decreases inversely to the dietary intake of this microelement, and involves only its ionized form. In cases when calcium is bound by other intestinal substances, there is malabsorption of this nutrient. Factors impeding the proper absorption of calcium include phosphates, fatty acids, sulphates, citrates, oxalates, while the factor that stimulates the absorption of calcium in the intestines is the active form of vitamin D_3. Eating habits have a significant impact on the excretion of calcium in urine. In this regard, one should consider the amount of calcium, sodium, potassium, animal protein and simple carbohydrates consumed in the diet, as well as dietary habits regarding caffeine and alcohol. Also significant is the fact that a poorly balanced diet may condition hypercalciuria. Nevertheless, it has not been unequivocally determined whether the supply of calcium in the diet directly affects its excretion or the formation of calcium deposits in the urinary tract (Curhan et al. 1993, 1997, 2004).

The relationship between low calcium content in the diet and the risk of urolithiasis has been proven (Borghi et al. 2002, Martini, Heilberg 2002). The role of calcium in this respect is extremely important, because this element is responsible for the adhesion of oxalate in the intestinal lumen and thus inhibits its absorption, which in turn determines its excretion, i.e. the appearance in the urinary tract. In this respect, limiting the intake of calcium may result in the occurrence of food hyperoxaluria, which promotes the formation of deposits in the urinary tract. It is also worth pointing out that additional calcium supplementation (exceeding the daily requirement) in the diet may increase the risk of urinary calculus formation (Périmenis et al. 2005, Taylor, Curhan 2006, Fink et al. 2009, 2013).

It is generally accepted that every additional 100 mg of calcium supplied in the diet over recommended dietary allowances increases urinary excretion by 8 mg/day in healthy people, but in people with hypercalciuria this can be an increase of up to 20 mg/day. In the case of people with urolithiasis, the intake of large amounts of calcium (above 2000 mg/day) usually leads to hypercalciuria and/or hypercalcemia. The average diet provides 700-1000 mg of calcium per day. The recommended dietary allowances for both genders aged 19-51 are 1000 mg/day and increase up to 1200 mg/day for female aged >51 years. In order to maintain the intracorporeal homeostasis of calcium metabolism, the adult diet should provide at least 400 mg/day. Analysing the relationship between the amount of calcium supply in the diet and the intensity of hypercalciuria and observation of the tendency to precipitate deposits in the urinary system, one should take into account the current physiological state of calcium absorption mechanisms in the intestine, hormonal and metabolic changes induced by calcium already absorbed from the intestine, and interactions between calcium and other ingredients of ingested food (Srivastava, Alon 2007, Worcester, Coe 2008, Cioppi et al. 2009, Hall 2009, Leslie, Sajjad 2019).

Seeger et al. (2017) draw attention to the relationship of urinary calcium excretion with the amount of sodium in the diet. Literature data confirm that an increased amount of sodium intake is accompanied by increased urinary excretion of calcium, because these changes affect renal calcium transport, i.e. excess sodium inhibits the reabsorption of sodium and calcium in the proximal canal of the nephron. In addition, the effects of excess sodium include a reduced amount of citrate in the urine and an increased risk of sodium urate crystal formation. It is stated that the daily intake of sodium chloride (NaCl) should not exceed 3-5 g and, according to the 2019 EFSA recommendations, safe and adequate intake (AI) of sodium is 2.0 g/day for the general EU population of adults, which corresponds to 5 g salt/day (Srivastava, Alon 2007, Worcester, Coe 2008, Cioppi et al. 2009, Hall 2009, Leslie, Sajjad 2019, Turck et al. 2019). Consumption of an additional 5 g of table salt may increase urinary calcium by 40 mg/day and reduce urine citrate by 50 mg/day (Pak 2004, Saxena, Sharma 2010).

THE CONTENT OF SELECTED COMPOUNDS IN THE DIET

Potassium in foods is accompanied by organic ions, such as citrate, which is metabolized to bicarbonate. Therefore, it is beneficial for humans to consume potassium-containing products, such as fruits and vegetables, which increase urinary excretion of citrate (Straub, Hautmann 2005). The adequate intake of potassium according to WHO and EFSA guidelines, mainly in the form of fruit, vegetables and dairy products, should be at least 3500 mg/day for adults. NASEM guidelines for the United States population recommend an adequate potassium intake at the level of 2,6 g/day for women and 3.4 g/day for men (WHO 2009, Turck et al. 2016, NASEM 2019).

Another issue is the formation of uric acid crystals, which depends on the concentration of uric acid and the urine pH level (as the pH of the urine increases from 5.0 to 6.5, the solubility of uric acid increases). In the case of increased uric acid excretion, it would be reasonable to take action to neutralize the acidity of urine, e.g. by administering a xanthine oxidase inhibitor that inhibits the activity of this enzyme involved in purine metabolism and consequently reduces the production of uric acid. Prevention of the formation of uric acid stones consists of the administration of high doses of vitamins B_1 and B_2 along with preparations of magnesium and hydrochlorothiazide. Decreased consumption of meat, chicken and seafood will limit purine intake and thus the production of uric acid, while higher consumption of fruit and vegetables will contribute to an increase in urine pH level and thus reduce the risk of uric acid crystal formation (Saxena, Sharma 2010).

In the case of cystine stones, diet also plays an important role, similar to the formation of uric acid stones, because increasing urine pH also increases the solubility of cystine. Therefore, it is recommended to reduce the consumption of sodium and increase the intake of fruit and vegetables. Because cystine is a derivative of the amino acid methionine, it is also recommended to limit the intake of food containing methionine, including products of animal origin, tofu, mushrooms, potatoes, broccoli, cauliflower, spinach, green peas, beans, avocados, peanuts, pistachios, etc. (Saxena, Sharma 2010).

In the case of excessive consumption of other compounds, such as simple carbohydrates and ethyl alcohol, increased urinary calcium excretion is also observed, which is caused by disturbances in the tubular reabsorption of calcium and magnesium in nephrons. During excessive alcohol consumption, urinary calcium excretion can reach up to 200%. As regards nutritional habits, the consumption of coffee (caffeine) is not clinically significant, because a significant increase of calcium excretion requires the consumption of very large amounts. Regarding insoluble fibre, it has the ability to bind free calcium in the intestinal lumen, helping to inhibit its absorption, although soluble fibre increases the absorption of calcium. In the case

of increased water consumption, a positive effect on the reduction of urinary calcium should be noted, but it does not directly affect the amount of calcium excretion (Worcester, Coe 2008, Leslie, Sajjad 2019).

PREVENTION OF THE OCCURRENCE OF UROLITHIASIS

Nowadays, prophylaxis is very important due to the growing number of cases of urolithiasis in the world. A diet is a very important element affecting both the formation of deposits and to their treatment, hence sound knowledge of the principles of proper nutrition can significantly reduce the risk of the recurrence of renal colic. There is no doubt that the most common cause of urolithiasis are nutritional errors, while the onset of treatment is associated with a change in dietary habits. Adapting patients to new nutritional requirements is usually a challenge because for many the replacement of most meat with vegetables and the exclusion of many sweets is not always acceptable. It is commonly observed that patients usually abandon the recommended proper dietary habits as soon as they feel better.

The general dietary recommendations for urolithiasis should be adapted to the type of deposit and other individual disease characteristics. In general, one can recommend a diet rich in whole grain cereal products with a low glycaemic index and glycaemic load, raw fruit and vegetables, and vegetable oils. A tailored approach to combating the disease is required, thus it is important to remember the comprehensive nature of nutrition of a person suffering from urolithiasis, also taking into account any other conditions. In the case of a low-purine diet, it is necessary to limit the supply of purines by limiting the supply of protein to 40-50 g/day (3-4 meals a day), selecting low-purine products, thus limiting or excluding offal, mutton, pork, fish, cocoa, strong tea, meat and fish stocks, legumes and mushrooms. Due to the content of purine compounds, the best source of protein for people with urolithiasis are dairy products, preferably natural and unsweetened. In a lacto--vegetarian diet, it is recommended to consume milk, fruit and vegetables, which are characterized by a low purine content, but also to maintain low acidity of urine (pH <5.5), because urine pH >6.5 reduces the tendency toward precipitation of uric stones. When deposits occur in the human urinary tract, a very important element of treatment is an appropriate amount of fluid intake, preferably mineral water, so that the amount of liquids consumed should result in passing almost transparent urine. A specific amount of water intake cannot be estimated precisely, because this depends to a large extent on the temperature and humidity of the environment and physical activity, but it is usually three litres of water per day. Fluids should be taken throughout the day evenly; it is also recommended to drink a full glass of water overnight (just before going to bed) (Assimos, Holmes 2000, Agarwal et al. 2011, Gul, Monga 2014, Han et al. 2015).

Dietary recommendations for oxalate stones apply to cooking, stewing, baking in foil or parchment, and frying without fat. Recommended products include meat, fish, poultry and large amounts of liquids. In Table 1, a list of products is given, depending on the indications and contraindications in the presence of oxalate stones in the body. Dietary recommendations for kidney stones apply to a lacto-vegetarian diet alkalizing urine, and to limiting meat and processed meats to 100-150 g per day, unlike the recommendations contained in the nutrition pyramid for healthy people, which suggests the consumption of 2-3 meat servings per week 110 g each. The recommended products include vegetables, fruit, milk and its products, flour dishes, sugar and plenty of liquids. It is advisable to prepare soups or vegetable stews. In Table 1, a list of products is given depending on indications and contraindications in the presence of uric acid stones in the body. Dietary recommendations for phosphate stones concern the consumption of urine acidifying foods such as meat, fish, sausages, bread, cereals, pasta and butter. It is advisable to limit consumption of milk and its products, vegetables, fruits and potatoes. In addition, products containing high amounts of calcium and phosphorus, including drinking water with a low calcium content, are not recommended. In Table 1, a list of products is given depending on indications and contraindications in the presence of phosphate stones in the body (Assimos, Holmes 2000, Agarwal et al. 2011, Gul, Monga 2014, Han et al. 2015).

PROBIOTICS FOR PREVENTION OF URINARY STONES

The structure of the gastrointestinal microbiome plays an important role in the process of metabolism and then the absorption of macro- and microelements. A special role in preventing the formation of urinary stones is played by intestinal microbes enriched in the presence of obligate anaerobic bacteria from the species *Oxalobacter formigenes*. These bacteria in the natural environment colonize the gastrointestinal tract of vertebrates, regulating levels of oxalate, whose degradation provides them with an energy source. The presence of this species found in the patient's faeces is associated with a lower risk of oxalate urinary stone formation. In most cases, the microbiome of patients with urolithiasis is deprived of *Oxalobacter formigenes*. Fermented dairy products, in addition to the benefits associated with limiting the supply of purines, are a rich source of anaerobic probiotic bacteria like *Bifidobacterium* or *Lactobacillus spp*, whose enzymes effectively degrade oxalates present in the diet. The study by Mogna et al. (2014) showed oxalate-degrading efficacy for *O. formigenes, Lactobacillus* and *Bifidobacterium spp.* at 98%, 68.5% and 11% respectively. Although it is possible to colonize the intestines with the above probiotic strains to reduce the risk of developing calcium oxalate stones, additional studies are needed

to confirm the positive therapeutic effect, as the studies conducted so far show a variable effect (Goldfarb 2007, Ferraz et al. 2009, Lieske et al. 2010, 2017).

PRODUCTS OF PLANT ORIGIN IN THE PREVENTION OF UROLITHIASIS

The basic reason for the formation of urinary stones can be detected in nutrition. Research has repeatedly confirmed the protective effect of a diet rich in plant products. An important part of the diet, in addition to vegetables and fruit, are spices, infusions and plant extracts, as well as seeds. Dietary fibre contained in plant products mechanically contributes to the reduction of urinary calcium and oxalate excretion and thus limits the formation of urinary stones. Common features of plants with proven action in the prevention of urolithiasis are antispasmodic, diuretic, anti-inflammatory and crystallization inhibiting effects. They achieve their properties through the content of various chemicals, among which catechin, epicatechin, epigallocatechin 3-gallate, diosmin, rutin, quercetin, hyperoside and curcumin are studied most often. A significant role is attributed to such plants as green tea, pomegranate, raspberries, parsley, black cumin, oregano, bitter orange, nettle, and less popular as *Rubia cordifolia, Solanum xanthocarpum, Pistacia lentiscus, Dolichos biflorus*.

Green tea, not quite recommended for patients diagnosed with urolithiasis caused by calcium oxalate building as it is a source of oxalate, finds application in treatment of this disease owing to the content of catechins, whose oxalate-reducing properties counteract the crystallization process (Nirumand 2018).

The chemical compounds contained in raspberries inhibit the activity of aldosterone and epithelial sodium channels, which results in a strong diuretic effect and thus limitation of the formation of stones in the presence of calcium oxalate. The polyphenols found in raspberries, known for their antioxidant properties, appear to control the level of citrate, magnesium and glycosaminoglycans, which are inhibitors of the formation of kidney stones (Nirumand 2018).

Rubia cordifolia has a protective effect against the formation of urinary stones by preventing hyperoxaluria and hypercitaturia, regulating the resorption of citrates and reducing calcium excretion, which lowers the acidity of urine predisposing to urolithiasis (Nirumand 2018).

Parsley is a plant rich in antioxidants, such as flavonoids, carotenoids, vitamin E and C. It also possesses a number of protective properties concerning the kidneys. The high content of parsley's magnesium and chlorophyll as well as the plant's ability to regulate urine pH prevent the precipitation

of calcium oxalates and reduces the loss of protein through the urinary tract (Nirumand 2018).

Pomegranate is used in many urinary tract diseases. Due to the richness of anti-inflammatory ingredients, such as polyphenols, anthocyanins, alkaloids, it is used in inflammatory diseases of the urinary tract with dysuric symptoms. Chemicals contained in pomegranate also affect the relaxation of the muscular urinary tract, which facilitates the passage and excretion of urinary stones. Studies have also shown the relationship between the high antioxidant potential of this plant and the protective effect against oxidative damage to the renal tubules (Nirumand 2018).

Equally effective is the protective effect of nettle, lemon juice and nigella against urolithiasis (Nirumand 2018).

Nephroprotective and anti-urinary stone formation effects have been demonstrated for plants such as *Angelica sinensis, Quercus gilva, Phyllanthus niruri* and coconut water. Studies on *Angelica sinensis* polysaccharide have shown that it has properties that inhibit the formation of calcium oxalate crystals and that its anti-crystallization effect is stronger than that of potassium citrate (Wang 2018). *Quercus gilva*, which is a source of antioxidant and anti-inflammatory ingredients such as phenolic compounds, prevents the development of urolithiasis (Youn 2017). Studies conducted on *Phyllanthus niruri* have shown that the basis of the anti-stone activity of this plant is the increased excretion of potassium and magnesium through the urinary tract, which, by affecting the concentration of sodium, increases urine alkalization and the presence of citrate in it, which inhibits the formation of calcium oxalate (Pucci 2018). One of the predisposing factors for the development of urinary stone formation is hypocytraturia. It is counteracted by the consumption of coconut water, the composition of which increases urine levels of citrate, potassium and chloride. Despite the strongly alkaline reaction, coconut water does not change urine pH or alkalinize urine (Jiang 2018). Supplementation with preparations of plant origin is beneficial in patients struggling with urolithiasis (Nirumand 2018).

CONCLUSIONS

The most common cause of urolithiasis are nutritional errors and the onset of treatment often involves a change in dietary habits. The general nutritional recommendations for urolithiasis should be adapted to the type of urinary stone deposit and an individual patient's comorbidities. When planning a nutrition program for a person suffering from urolithiasis, particular attention should be drawn to limiting the supply of protein from animal sources and to replace it with the consumption of dairy products and vegetable protein. Restrictions should be imposed on the supply of NaCl and fructose. It is suggested to limit the consumption of fruit and vegetables rich

in fructose or methionine and select those that are rich in fibre and potassium. Water is the drink of choice for patients with urolithiasis, and its supply should be increased, taking into account portions drunk at bedtime and at night. Current recommendations do not mention a significant reduction in calcium intake, the amount of which in the patient's diet should meet the daily demand consistent with the patient's sex and age. A tailored approach to treatment of the disease is required.

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Table 1

List of food products indicated and contraindicated in the presence of selected types stones in the body

Types of stones	Forbidden	Allowed	Recommended
Oxalate	spinach, sorrel rhubarb, beetroot leaves, lemons, dried figs, chocolate, cocoa, natural coffee, strong tea, spicy spices, peas, beans, soy, lentils	potatoes, carrots beetroot, green peas tomatoes, tomato concentrate, plums gooseberry, sugar milk, sweets	large amounts of liquids, meat, fish eggs, butter, bread cabbage, cucumbers lettuce, onion fruits (except those listed), grain products
Phosphate	legume seeds (peas, beans, soybeans, lentils) cheese, cream cheese, canned fish	milk, eggs, potatoes vegetables, fruits	large amounts of liquids, meat, low fat cheese, fish, bread, groats, pasta butter
Uric acid	liver, brain, blood sausage, mutton, pork, caviar, herring, sardines, chocolate, cocoa, natural coffee, strong tea, nuts, mushrooms	veal, beef, poultry fish, meat and fish stocks	large amounts of liquids, vegetables fruits, milk, low fat cheese, yoghurt, kefir, sugar butter in small amounts, potatoes grain products

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