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Diet in hyperuricemia and gout – myths and facts

Dieta w hiperurykemii i dnie moczanowej – mity i fakty

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Słowa kluczowe: dieta w dnie moczanowej, zalecenia żywieniowe, produkty ubogopurynowe, alkalizacja ustroju.

Summary

The influence of dietary habits on the gout development has been described for over a hundred years, but at present many opinions are being verified, which have not been so far supported by epidemiological or clinical studies. The promotion of healthy dietary behaviours, keeping proper body mass and adequate dietary pattern are the factors, which support lowering of serum uric acid. Diet in gout should be well-balanced and contain the proper amount of selected nutrients, not only low-purine but also alkalizing products, rich in antioxidants, and provide an adequate amount of fluids. The following manuscript presents the recent data of dietary recommendations in gout, which are supported by clinical or epidemiological studies.

Streszczenie

Wpływ zwyczajów żywieniowych na rozwój dny moczanowej jest opisywany od ponad stu lat, jednak obecnie weryfikuje się wiele opinii, które nie były do tej pory poparte badaniami epidemiologicznymi lub klinicznymi. Promowanie prozdrowotnych zachowań żywieniowych, utrzymanie prawidłowej masy ciała oraz odpowiedni sposób żywienia są czynnikami sprzyjającymi obniżeniu stężenia kwasu moczowego w surowicy. Dieta w dnie moczanowej powinna być prawidłowo zbilansowana pod względem ilości poszczególnych składników odżywczych, powinna zawierać nie tylko produkty ubogopurynowe, lecz także alkalizujące i bogate w antyoksydanty, a także dostarczać odpowiednią ilość płynów. W pracy przedstawiono najnowsze doniesienia dotyczące zaleceń żywieniowych w dnie moczanowej poparte badaniami klinicznymi lub epidemiologicznymi.

Introduction

Despite continual progress in diagnostic and therapeutic techniques for gout, novel and effective prevention and treatment methods are still needed. High uric acid levels (hyperuricemia) are not only a risk factor for developing gout but also predispose those affected, including patients with rheumatoid arthritis, to cardiovascular disease – the primary cause of morbidity and mortality in the world [1, 2]. Hyperuricemia, which is an independent risk factor for cardiovascular complications, may occur even in young people, including populations with a low incidence of cardiovascular disease [3, 4].

Gout, with an incidence of 1–2% in the general population, constitutes the most common form of arthritis in men aged between 40 and 60 years [5]. The disease has been associated with purine-rich foods, which are common in a normal diet (Fig. 1). At the same time, given the known side effects of drugs inhibiting uric acid synthesis or excretion, novel and alternative methods to ensure safe treatment of hyperuricemia are sought [6].

Hyperuricemia and diet – epidemiological and clinical data

The type of clinical studies, selection of control and treatment groups (e.g. with accurate diagnosis of gout or only hyperuricemia), duration of follow-up, as well as age, ethnicity, and genetic predisposition of the study population play an important role in determining the effect of diet on both uric acid levels and the development of gout. The sheer number of these factors may pose various challenges in accurately estimating the impact of diet on serum uric acid elevation. Interestingly,

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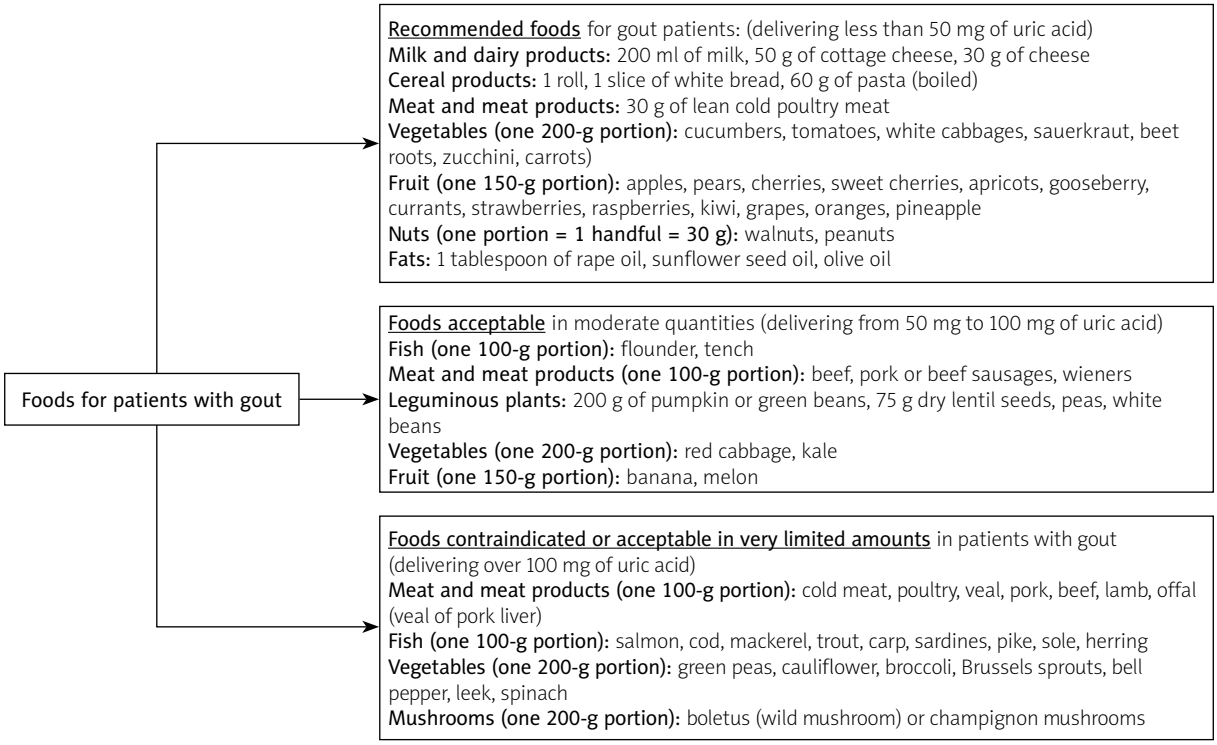

Fig. 1. Purine-containing foods for patients with gout and the amount of uric acid delivered (daily uric acid intake should not exceed 500 mg) – figure modified by authors [40].

the surveyed respondents themselves often mistakenly assume high uric acid levels to be synonymous with the diagnosis of gout. Moreover, for many patients, the treatment with xanthine oxidase inhibitors (e.g. in metabolic syndrome) is indicative of a gout diagnosis, and any joint pain concomitant with hyperuricemia is subjectively construed as an indicator of this disease.

Although there have been a number of papers emphasizing a cumulative effect of individual foods (especially purine-rich foods) on serum uric acid elevation, the reports published so far have been inconsistent, and sometimes even contradictory [7–9]. Therefore, many investigators claim that despite the unquestionable value of information obtained from studies on the effect of individual foods or nutrients on the development of hyperuricemia, it is more important to assess dietary habits as a whole and their effects on uric acid levels [8]. According to the American Dietetic Association, dietary purine content should be evaluated based on all the foods consumed during an extended period of time (e.g. one week) to include both the days when purine-rich foods are consumed and the days with low purine intake [7].

An association between diet and the development of gout was first suggested as early as in 1876 [10]. However, the relevance of diet was largely minimized with the advent of new pharmacological treatments for this disease (in both acute gout attacks and chronic gout) [11, 12]. Thus, nutritional education and life style modification are not always encouraged and used alongside medical treatment. Australian data show that only a very small proportion of gout patients receive education on health- and prophylaxis-oriented eating habits [13]. This may be a result of challenges associated with maintaining a low-purine diet, as it requires following certain rules and putting a greater effort into daily planning of daily food rations. Nonetheless, each patient greatly benefits from low-purine diet, and its effects in some patients are comparable to those achieved via medical treatment. Moreover, altering eating habits is more cost-effective and safer than pharmacotherapy, due to elimination of any drug-related side effects, which is especially important in the elderly [14]. This does not mean that medical therapy should be relinquished altogether, yet rational planning of the daily menu increases the chance of dose reduction (including complete discontinuation) and eliminates a number of risk factors for the development of gout (e.g. obesity or metabolic syndrome) [7].

Increased body weight is a known risk factor for hyperuricemia. Laboratory and clinical studies show a consistent association between serum uric acid levels on the one hand and insulin resistance, metabolic syndrome, and obesity on the other. Hyperinsulinemia inhibits uric acid excretion, whereas weight reduction leads to reduced uric acid production and the subse-

quent decrease in uricosuria [15, 16]. These phenomena are due not only to a higher insulin sensitivity in peripheral tissues, but also to beneficial effects of a low-energy diet which is typically associated with a reduction in purine-rich food intake [17].

Diagnostic and interpretive challenges

Given the current body of knowledge, nutritional study results should be interpreted very carefully, as wrong conclusions may lead to unsuitable dietary recommendations (Table I). This is especially well illustrated by studies in Taiwanese populations – Nutrition and Health Surveys in Taiwan (i.e. the NAHSIT 2005–2008 study) that demonstrated reduced uric acid levels and a lower risk of hyperuricemia (at the same time showing an increased proportion of patients diagnosed with gout) in comparison to earlier findings (the NAHSIT 1993–1996 study) [8].

Diets containing vegetables, eggs, lean meat, soy products, seaweed, and caffeine as well as very limited amounts of bamboo shoots, offal, and non-alcoholic beverages were shown to have a beneficial effect on normalizing uric acid levels. Although most of these findings were rational from the clinical point of view, the suggested limitation in the consumption of bamboo shoots – a high-fibre product – has raised many doubts. Dietary fibre has hypolipemic properties and is beneficial to health [18]. A later, more detailed analysis of 24-hour dietary recall revealed that bamboo shoots constituted an ingredient in high-energy meat dishes and fatty meals; consequently, their occurrence in daily food rations showed a positive correlation with increased uric acid levels. Hence, the observed hyperuricemia was not associated with this high-fibre food itself, but rather with the quality and quantity of consumed fats that led to increased ketone body synthesis and the subsequent inhibition of renal uric acid excretion.

Epidemiological studies indicate an association between dietary habits of a given population with the particular geographic region – i.e. inhabitants of seaside or mountain areas demonstrate disparate dietary habits due to the local availability of certain foods (e.g. salt-water fish) [19]. Thus, the incidence of gout may vary from region to region. Studies to evaluate dietary habits should focus on individual geographic regions, and the findings should not be extrapolated to include the population of the whole country.

Table I. Gout – myths and facts (original table)

Analyzed factor	Myths	Facts
Purines	All purine-rich foods determine serum uric acid levels to the same extent	Adenine and hypoxanthine are considered to be the most uricogenic of all purines, in comparison to guanine and xanthine
Low-purine diet	A strict low-purine diet is crucial in the treatment of hyperuricemia and gout	It is not only a low-purine diet but also an alkaline diet that significantly affect serum uric acid levels due to more rapid and easier elimination of this compound with urine
Risk factors for gout	Purine-rich diet affects uric acid levels to the same extent in all patients	Obese hyperinsulinemic patients and the elderly are affected by a high-purine diet to a greater extent than patients with normal weight and those of younger age
Protein intake	In every case of gout, protein should be eliminated from the diet, due to the fact that protein-rich foods contain large amounts of purines	Proteins exhibit uricosuric properties and should be neither totally eliminated nor even significantly limited in the diet; moreover, milk and dairy products facilitate uric acid excretion due to their alkalizing effects on systemic fluids
Fat intake	The quality of fats (especially animal fats) is the main factor determining the amount of uric acid produced	In the case of gout, it is the quality rather than quantity of fats that is of greater importance, because uric acid excretion depends mostly on the levels of ketone compounds which are formed irrespective of the quality of fats if there is too much fat in the diet
Alcohol	The quality of alcohol determines the development of gout	Alcohol increases the risk of gout and should be eliminated from the diet in all cases; however, the most potent factor responsible for hyperuricemia and gout is occasional consumption of large quantities of alcohol, especially beer
Epidemiological studies	Most of the published population studies emphasize the role of diet in the development of gout	The quality of studies, group selection and methodology significantly affect the final conclusions; respondents to the present study's survey (especially those, interviewed by telephone) often – wrongly – took high uric acid levels or the fact of receiving medical treatment for indicators of gout diagnosis

The effects of diet on uric acid levels – myths and facts

Epidemiological and clinical studies have shown an effect of purine-rich food consumption on uric acid levels [3, 4, 9]. However, there are noteworthy differences in the effect of individual purine bases and their metabolites on endogenous purine synthesis and the resulting uric acid levels. Adenine and hypoxanthine are considered to have the highest uricogenic potential out of all purines (especially in comparison with that of guanine and xanthine) [20]. Moreover, the correlation of selected food intake and the development of gout is more complex and is not only due to the purine content of a given diet, but also to the intake of proteins, fats, and alkalizing products [21]. Moderate limitations on high-protein foods are recommended in the case of foods with very high animal (i.e. purine-rich) protein content [11]. Nonetheless, proteins have been shown to have uricosuric properties and to facilitate uric acid excretion. Serum uric acid levels are determined by both the quality and quantity of protein intake [22].

Foods rich in plant proteins include compounds found in leguminous plants and soya. Leguminous plants are a rich source of purines, however, their effect on uric acid levels depends on the make-up and size of dietary portions (Fig. 2).

Protein-rich low-fat dairy products have been shown to reduce uric acid levels and the risk of gout [9, 23, 24]. Therefore, the recommended proportion of fats in diet should not exceed 30% of the total daily energy intake. Apart from sea food and alcohol, fatty meat is one of the best predictors of uric acid levels and largely determines the risk of gout [9, 23, 24]. Conversely, lean meat intake showed an inverse correlation with uric acid levels [8]. Moreover, when dealing with gout, it is the quantity rather than quality of fats in the diet that is more important. Nonetheless, a reduction in animal fat intake with a compensatory increase in consumption of plant-derived fats (e.g. olive oil or vegetable oils) is recommended. This recommendation is a result of beneficial effects of mono- and polyunsaturated fatty acids on cardiovascular risk associated with hyperuricemia. The risk of cardiovascular disease tends to be lower with consumption of soybeans and soy products, which have been extensively studied over the last decade and more and more is known on their role in low-purine diets. Initially, soy – a leguminous plant – was believed to induce hyperuricemia and, as such, considered to be an entirely superfluous diary component. However, later studies indicated that soy products may have a beneficial effect on uric acid levels [8]. Tofu consumption was shown not to increase uric acid levels, even in patients with gout, whose serum uric acid levels exceeded 6 mg/dl [25]. Moreover, soy protein may be more effective than casein in reducing uric acid levels [26] and can help in the treatment of obesity due to its relatively high calcium content (calcium plays a role in adipocyte enzyme activation and reduces the amount of adipose tissue). These findings were confirmed by prospective randomized studies that showed that soy milk and soy creamer are effective in weight reduction [27]. Thus, in comparison to other purine-rich foods, soy products seem to have a limited impact on uric acid levels [23, 24] and therefore can be consumed in moderate amounts (foods of moderate purine content: between 50

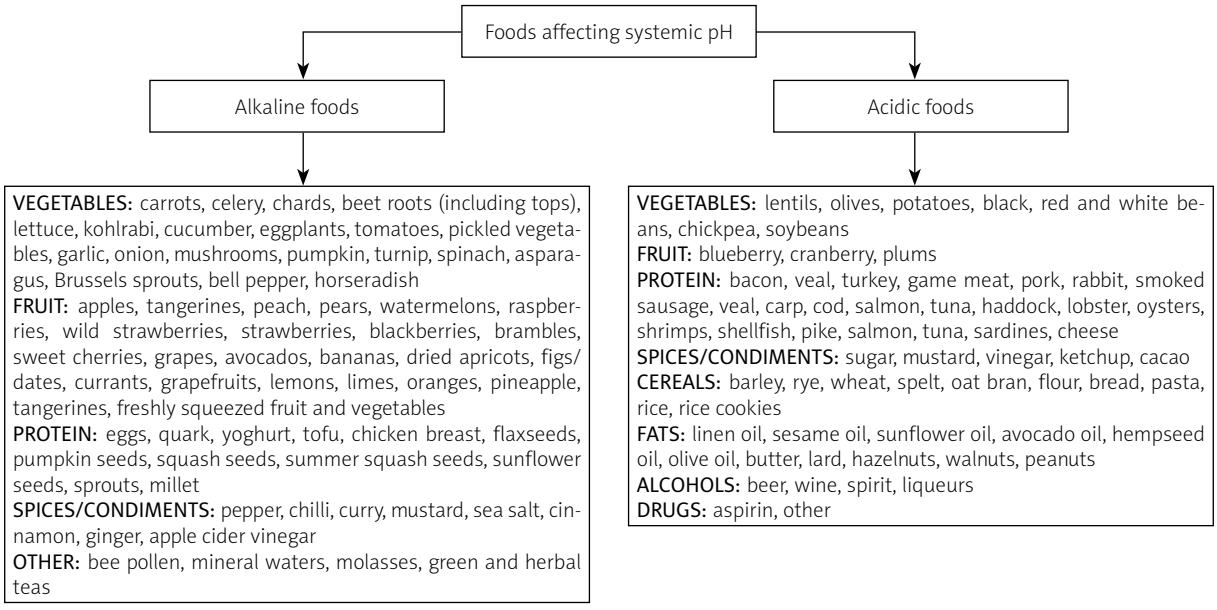

Fig. 2. Alkaline and acidic foods in the gout diet (figure modified by authors [6, 38, 39]).

and 100 mg/100 g of product) by patients with hyperuricemia.

As mentioned earlier, obesity (mainly visceral obesity) and alcohol consumption are risk factors for hyperuricemia and gout [28, 29]. Metabolic syndrome itself has been also associated with a higher incidence of gout attacks. Increased body weight is often due to a greater consumption of non-alcoholic beverages that tend to increase uric acid levels [3, 8, 15]. Studies in the American population, such as the Third National Health and Nutrition Examination Survey, demonstrated a direct correlation between uric acid levels and sweetened non-alcoholic beverage consumption, irrespective of any other risk factors for hyperuricemia [30]. Such beverages have high fructose content, which has been associated with hyperuricemia [14], elevated blood glucose and insulin levels, reduced uric acid excretion [30], and an increased proportion of patients with metabolic syndrome [15]. Although it has not been confirmed by all the studies [31], some authors believe excessive consumption of juices and carbonated beverages to be a risk factor for hyperuricemia. Alcohol, especially when consumed occasionally in large quantities, is one of the key risk factors for gout, especially gout attacks [32]. Alcohol metabolism results in lactic acid synthesis, which – by lowering the pH of bodily fluids – reduces uric acid solubility and causes its crystallization [19]. Additionally, there is an increase in hepatic metabolism of adenine nucleotide and endogenous uric acid synthesis. Moreover, low pH tends to inhibit renal uric acid excretion. Another important aspect of alcohol is its high purine content. Large quantities of purines can be found in beer – 500 ml of beer contains an equivalent of 170 mg of uric acid, which corresponds to 71 mg of purines (1 mg of purine compounds corresponds to an equivalent of 2.4 mg of uric acid). It is important to avoid simultaneous consumption of alcohol and fatty products due to their synergic action in inducing gout attacks [9, 19, 23, 24].

No clear correlation has been shown between coffee consumption and serum uric acid levels, yet most guidelines suggest entirely eliminating coffee from daily diet. However, there are reports indicating that coffee consumption inversely correlates with uric acid levels [8, 24, 33]. The Third National Health and Nutrition Examination Survey revealed a moderate correlation between decaffeinated coffee consumption and uric acid levels, which suggests that it is not caffeine but rather other ingredients that may improve uricemia. This effect may result from the presence of xanthines that lower uric acid levels. Xanthines are compounds that naturally occur in foods and are structurally similar to allopurinol – a structural isomer of hypoxanthine and xanthine oxidase inhibitor [24, 33].

Recently, more and more emphasis has been placed on the role of antioxidants in gout diet, as they have been associated with the treatment of gout. Of particular importance seems to be the consumption of cherries that can reduce the risk of gout due to their significant anthocyanin, catechin, chlorogenic acid, flavone glycoside, and melatonin content. Anthocyanins and hydroxycinnamate isolated from cherries inhibit oxidation of LDL-cholesterol and have a beneficial effect on liposomes similar to that of vitamin E [34]. Isolated from cherries, anthocyanins also have anti-inflammatory properties via cyclooxygenase inhibition and scavenging free nitric oxide (NO) radicals [34, 35]. Moreover, anthocyanins and other phenols inhibit NO synthase and regulate the secretion of tumour necrosis factor α (TNF-α) by activated macrophages [34–36]. Cherry consumption is believed to reduce joint pain severity and the risk of gout [37]. The consumption of 227 g of cherries or cherry preserves for a period of 3 days to 3 months normalizes serum uric acid levels and reduces the number of gout attacks [34, 37]. In order to definitively endorse the inclusion of these products into dietary recommendations for gout patients, further clinical studies involving a large number of patients diagnosed with gout are necessary. This does not change the fact that cherries are rich in vitamins, minerals, dietary fibre, and phytocompounds, which – apart from their beneficial anti-gout effects – also reduce the risk of cancer, cardiovascular diseases and other chronic diseases that often accompany gout.

Alkaline diet in gout

Gout diet should not be based on a simple calculation of the quantity of purines in the consumed foods, as uric acid levels are also determined by acidic or alkaline properties of food [6]. Prehistoric *Homo sapiens* excreted more alkaline urine, which was due to the intake of large quantities of plant carbohydrates. As a result, uric acid was excreted considerably more rapidly than in the present-day populations. Unfortunately, the current Western diet includes high-energy density foods (rich in saturated fatty acids and refined sugars) that are low on nutrients and buffers, which leads to an increased acidity of urine, making uric acid excretion more difficult [38]. Therefore, the last decade saw an increasing emphasis on the role of alkalizing foods that facilitate uric acid elimination from the body. These processes involve human organic anion transporter 4 (hOAT4), which plays a role in urate re-absorption in proximal tubules [39]. Understanding the mechanism of action of this transporter in uric acid excretion has opened new possibilities of dietary treatment of hyperuricemia, as increased urine pH due to limited transport of hydrogen (H^+) ions derived from metabolic processes was shown to facilitate uric acid excretion [6].

Sulphur-containing amino acids, which determine urine acidity, are main components of animal proteins and therefore a high-meat diet facilitates the production of urine with the pH lower than that achieved on low-protein diet rich in vegetables and fruit. Patients who prefer meat and meat products are more prone to developing gout than vegetarians. Thus, properly balanced meals containing alkaline foods are more effective in uric acid elimination than a low-purine diet alone [6, 38, 39].

Most unprocessed foods have an alkaline pH (Fig. 2). Unfortunately, culinary treatment and food processing strip food not only of the most valuable nutrients, but also change its alkaline pH into acidic pH. Due to pH-dependent uric acid transport in the kidneys, uric acid excretion is limited in the presence of acidic urine, therefore it is important to maintain an adequate balance between alkaline and acidic foods, with a target ratio of 3 : 1. Foods with acidic properties include coffee (pH = 4), beer (pH = 2.5), Coca-Cola (pH = 2), and products grown with the use of pesticides or herbicides. Moreover, products containing aspartame, sodium glutamate, and food dyes also lower the pH of serum and urine. Conversely, such ions as potassium (pH = 14), sodium (pH = 14), calcium (pH = 12), magnesium (pH = 9) and caesium (pH = 14) have alkaline properties. Of note is the fact that acid- or alkaline-forming properties of foods are not associated with the pH of those foods prior to their consumption [6, 40]. One example is lemon, which has an acidic pH, however, becomes alkaline once digested and metabolized. Conversely, meat, which has an alkaline taste, turns acidic as a result of metabolic processes and lowers systemic pH [6, 38, 39].

Acidic and alkaline properties of the diet are determined not only by the type of foods, but also by the way they are prepared and combined into meals. Plants rich in chlorophyll and vegetable oils are considered to be highly nutritious. Alkaline mineral waters should be also included in the diet. Meals balanced in this way additionally facilitate uric acid excretion with urine, which increases the diet's effectiveness [6].

Dietary recommendations for gout

A gout diet should include normal intake of basic nutritional components, vitamins, and minerals. A number of products must be excluded, as they significantly affect uric acid levels: bone and meat stocks, broths, offal and cold meats, canned meat and fish, fish from the clupeidae family (sardines, sprats, herring), some cheeses (processed, blue, rennet cheeses, and high-fat creamy quarks), animal fats (suet, lard, bacon), corn, sorrel, spinach, rhubarb, chocolate, marinades, and certain spices (e.g. pepper, mustard, and root spices). A daily portion of meat or fish should not exceed 100 g. Purine-rich vegetables (such as leguminous plants, cabbage, Brussels sprouts, lentils, green peas, white beans) should be eliminated from the diet. Alcohol, coffee, black tea, cacao, and hot spices must be eliminated from the diet. Total daily intake of uric acid should not exceed the equivalent of 500 mg of uric acid, i.e. the amount of purine-containing products (substrate) [mg], which corresponds to the production of a specific amount of uric acid [in mg]. An important, unfortunately often ignored, recommendation is adequate fluid intake (especially alkaline-forming fluids, such as alkalizing mineral waters) in order to facilitate uric acid elimination [40].

Regular meal times are crucial, optimally with weighing the portions – especially in the case of purine-containing foods (see Fig. 1). Snacking between meals should be avoided, and the last meal should be eaten no later than 3 hours before going to bed to prevent accumulation of uric acid in the body [11, 40].

Carbohydrate-rich foods facilitate the excretion of urate from the body, whereas high-fat diet increases reuptake of these compounds in the renal tubules. The intake of milk and dairy products, as well as fruit and vegetables reduces the risk of hyperuricemia (no-purine foods include butter, cream, honey, and jam). Low-purine foods (apart from milk) include rice, fruit, and eggs; however, in the case of obesity or metabolic syndrome, one needs to bear in mind the need for simultaneous prevention of dyslipidemia and hyperglycaemia, as well as monitor the amount of no- or low-purine foods, which may interfere with the treatment of obesity due to their pro-atherogenic and pro-diabetic potential. The optimal culinary treatment method is cooking, stewing in a large amount of water (especially with respect to meat). Frying and baking are to be avoided [13, 40].

In summary, a gout diet must be adequately balanced in terms of individual nutrients, should include low-purine and alkalizing foods, and provide an adequate amount of fluids.

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