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ОГЛЯДИ

ROLE OF INDIVIDUAL MICROELEMENTS AND VITAMINS IN HASHIMOTO'S DISEASE*

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Hashimoto's disease is a chronic autoimmune inflammation of the thyroid gland. It is associated with lymphocyte infiltration within the thyroid parenchyma and the presence of autoantibodies, most commonly to thyroperoxidase (anti-TPO) and to thyroglobulin (anti-Tg) [1]. The disease is more common in women, most commonly presenting between the ages of 50 and 55, and the incidence in the population continues to rise. It can progress initially with euthyroidism and inflammatory infiltration, followed by gradual destruction of follicular cells, decreased thyroid hormone production and hypothyroidism, which is the most common cause in countries without iodine deficiency problems [2, 3]. Over time, fibrosis of the thyroid parenchyma and a decrease in thyroid volume occur.

Patients with Hashimoto's disease have an increased risk of developing other autoimmune diseases such as celiac disease, malignant anemia, Sjögren's syndrome [4], myasthenia gravis [5] and systemic scleroderma [6].

The pathogenesis of Hashimoto's disease is extremely complex and the development of this disease is influenced by a number of factors, the interaction of which increases the likelihood of an autoimmune response. Genetic, environmental and hormonal factors often interact to induce an impaired immune response against normal thyroid cells. Environmental factors can increase the risk of disease, particularly in patients with genetic risk factors. These include bacterial and viral infections, exposure to harmful chemical products such as phthalates, and smoking [7].

An important role in the pathogenesis is played by Th17 helper T lymphocytes, which significantly increase the production of cytokines such as IL-21, IL-22 or IL-23 [8]. The ra-

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tio of regulatory T cells to Th17 cells also plays an important role, as regulatory T cells control the body's autoimmune response, whereas Th17 cells induce and amplify this process [9]. Identification of these cells may be a future target for causal treatment of Hashimoto's disease.

Changes in the intestinal microflora are also relevant in the context of Hashimoto's disease. The presence of Bifidobacterium and Lactobacillus strains significantly reduced the risk of autoimmunity, whereas the presence of Bacteroides fragilis strains increased the rate of disease [10].

The **aim** of this study is to review the literature to fully assess the impact of micronutrients on symptoms, prognosis and dynamics of change in the context of Hashimoto's disease. Articles were searched by entering keywords in PubMed databases. Scientific articles covering the period from 2016 to 2024 represent 76% of the items included in the literature. Studies from specialized research centers including meta-analyses, double-blinded randomized trials, large cross-sectional studies were considered. Studies from inexperienced centers with methodological flaws were discarded.

DESCRIPTION OF THE STATE OF KNOWLEDGE

Iodine

Iodine is undoubtedly one of the most important elements for the proper functioning of thyroid hormone metabolism. Iodine is a necessary element for the synthesis of thyroxine and triiodothyronine. The WHO recommends a daily intake of 150 μg of iodine per day, while pregnant women have an increased requirement and should take 250 μg of iodine per day [11]. One of the main sources of iodine in Poland is salt, but in the case of cardiovascular diseases, especially hypertension, it is recommended to limit its intake. Other dietary sources of iodine include seafood, fish, mineral waters with high iodide content, and dairy products [12], (Table 1).

There is an association between excess iodine and an increased risk of Hashimoto's disease. However, there are no uncontroversial and confirmed reasons for these associations. It is thought that excess iodine increases the production of cytokines, which stimulate inflammation of the thyroid. Another possible process that may play an important role in the pathogenesis of Hashimoto's disease is excess iodine, the processing of which in the thyroid cells increases unfavorable lipid metabolism, leading to the induction of oxidative stress and an increase in free radicals, resulting in thyroid damage [13]. Patients with excessive iodine intake also experience increased apoptotic processes and suppression of autophagocytic metabolism [14]. It is increasingly recommended that iodine levels be controlled to eliminate the negative effects of iodine excess and deficiency [15].

Selenium

Selenium is another very important element that has many key functions in thyroid hormone management. Sources of selenium, in addition to supplementation in the form of selenomethionine, include: marine fish, eggs, seafood, innards, Brazil nuts, onions, garlic and mushrooms [16], (see Table 1). The optimal concentration of selenium in the body is in the range of 60-140 μg/L. Excess selenium increas-

Table 1

Micronutrient rich dietary elements

Microelements and vitamins	Products
Iodine	Salt, seafood, fish, mineral waters, dairy products
Selenium	Marine fish, eggs, seafood, innards, Brazil nuts, onion, garlic, mushrooms
Zinc	Red meat, cereal products, beans, seafood
Magnesium	Spinach, peas, beans, bananas
Iron	Red meat, poultry, eggs, spinach
Vitamin D	Oily fish, fish liver oils, eggs, meat products
Vitamin B12	Red meat, milk, eggs, dairy products

es the risk of cardiovascular disease, adversely affects the lipid profile and its metabolism, and interferes with carbohydrate metabolism, increasing the risk of type 2 diabetes. However, it is selenium deficiency that is a significant risk factor for autoimmune diseases, including Hashimoto's disease [17].

Selenium is often found in combination with proteins as antioxidant enzymes. An example of such an enzyme is glutathione peroxidase, which protects cells from oxidative processes and whose activity depends on selenium availability [18]. Excess dietary iodine can inhibit glutathione peroxidase activity and thus increase the intensity of oxidative processes leading to thyroid cell destruction [19]. However, selenium supplementation may help to reverse the pathological effects of excess iodine by increasing the number of regulatory T cells. These cells can modulate the body's autoimmune response by reducing the production of interleukin 2, which induces the production of antibodies, mainly anti-TPO and anti-Tg, by B lymphocytes [20].

Another important finding was a study showing that in patients with Hashimoto's disease, the use of both levothyroxine and selenomethionine produced a greater anti-inflammatory effect than levothyroxine monotherapy. This is because levothyroxine acts mainly on impaired monocyte function and selenomethionine acts by inhibiting cytokine production by lymphocytes and reducing the stimulation of B lymphocytes to produce autoantibodies. However, the mechanisms why this happens are not known and further studies are needed to clarify the mechanisms of action of these two substances [21].

In the most recent European Thyroid Association (ETA) guidelines on Hashimoto's disease, there are no standards for selenium supplementation. A large majority of ETA members stated that the current scientific evidence was insufficient to include selenium supplementation as a gold standard. However, a larger proportion of members were in favor of including selenium supplementation in patients with Hashimoto's disease who were not receiving levothyroxine [22].

In one study, women with Hashimoto's disease were significantly more likely to have low selenium levels than healthy women. Selenium-deficient women had higher levels of both anti-TPO and anti-Tg antibodies and TSH. This confirms the negative effect of oxidative processes on the thyroid and the stimulation of inflammatory processes. The authors recommend extending the study under conditions of dietary modification in order to standardize the management of selenium-deficient patients [23].

In one study, selenium-deficient patients with Hashimoto's disease who were euthyroid were given 200 μg of selenium per day for 6 months. This resulted in an increase in selenium levels and a decrease in anti-TPO and anti-Tg antibodies in the patients [24]. In another study, selenium supplementation had a similar effect, reducing anti-TPO antibodies through selenium's antioxidant properties [25]. In the next study, Hashimoto's disease patients with euthyroid or subclinical hypothyroidism were given 100 μg of selenium for 6 months. This reduced anti-TPO and anti-Tg antibodies and inhibited and slowed the development of full-blown hypothyroidism [26].

Selenium supplementation at a dose of 83 μg per day for 4 months without levothyroxine also helped normalize TSH parameters by maintaining a euthyroid state in selenium-supplemented patients [27]. Selenium supplementation has also been shown to increase glutathione peroxidase and selenoproteins [28].

There are also studies in which selenium supplementation did not have the expected effects. One of these is the study by Esposito et al. in which one group of patients received 166 μg per day for 6 months and the other group received a placebo. Both TSH and antibody results showed that supplementation was ineffective, which only confirms the fact that selenium needs to be studied more extensively in the context of standards of practice for Hashimoto's disease [29].

Zinc

Zinc is another element that plays an important role in the production of thyroid hormones. In the diet, the highest amounts of zinc are found in red meat, cereal products and seafood. However, eating large amounts of legumes can reduce the bioavailability of zinc [30]. It

regulates the activity of deiodinases, TRH and TSH. A correct zinc concentration determines the correct course of the transcription process of the structures necessary for thyroglobulin production [31]. Zinc, like selenium, has antioxidant properties and reduces the production of free radicals [32]. Zinc deficiency induces inflammatory activity of the immune system through dysfunction of regulatory lymphocytes and increased synthesis of pro-inflammatory cytokines. Zinc is also involved in thymic involution, which contributes to an increased risk of autoimmune disease with age [33]. The results of a study in a group of obese patients comparing the effects of selenium and zinc supplementation were inconclusive. Blood concentrations of both selenium and zinc did not change significantly. In the group that supplemented with both selenium and zinc, FT4 and FT3 parameters increased significantly and TSH decreased. In the group that took selenium supplements only, only FT3 levels increased significantly [34]. Further research on zinc supplementation in patients with Hashimoto's disease will be able to verify the real effect of the therapy.

Magnesium

Magnesium is an important micronutrient required by numerous enzymes to catalyze many transformations in the human body, and is also important in the prevention of kidney stones and in the treatment of some cardiac arrhythmias. Magnesium deficiency is common in humans, but a balanced diet can ensure an adequate intake of this element. Foods rich in magnesium include spinach, peas, beans and bananas [35], (see Table 1). Magnesium has anti-inflammatory, immunomodulatory and antioxidant properties. While hypomagnesemia is a risk factor for many diseases, it is uncertain whether it has a direct effect on the risk of developing Hashimoto's disease. In one study, patients with reduced blood magnesium levels had increased anti-Tg antibodies and a higher incidence of hypothyroidism, while anti-TPO antibodies were comparable to normomagnesemic patients [36]. Another study looked at patients with Hashimoto's disease and antibodies to micronutrients and found significantly lower magnesium levels in these patients [37].

Iron

Iron is essential for normal thyroid peroxidase activity and thus has an important function in the production of thyroid hormones. Iron can be supplemented by both medication and a balanced diet. Most iron is found in red meat, poultry, eggs, spinach and pulses [38], (see Table 1). Iron deficiency in patients with Hashimoto's disease may result in decreased peroxidase activity, elevated TSH levels, and increased thyroid volume, which may be due to malabsorption caused by coexisting celiac disease or autoimmune gastritis, which is more common in this patient population [39]. Iron supplementation in patients with iron deficiency improves erythrocyte and hemoglobin parameters and allows for normal thyroid hormone synthesis [40]. A study comparing patients with current elevated anti-TPO and anti-Tg antibodies with healthy patients showed that patients with autoantibodies had a significantly higher incidence of anemia, suggesting that patients with autoimmune thyroiditis have an increased risk of iron deficiency anemia [41]. A recent meta-analysis has shown that iron deficiency increases anti-TPO antibodies, confirming the value of concurrent iron and thyroid hormone management [42].

Vitamin D

Vitamin D has many functions, both as a suppressor and an activator of certain genes. It has immunomodulatory and anti-inflammatory effects by influencing the differentiation of CD4+ T lymphocytes. Vitamin D can be produced endogenously by exposure to sunlight (the largest source of the active form of vitamin D, i.e. calcitriol), ingested in the diet (most abundant in oily fish, fish liver oils, eggs and meat products) and supplemented in the form of various pharmacological agents [43]. Vitamin D deficiency has been found to be significantly more common in patients with Hashimoto's disease and other autoimmune thyroid diseases. This may be due to diet as well as the disease itself. Vitamin D supplementation can improve thyroid hormone parameters and reduce anti-TPO and anti-Tg antibodies [44]. In one study, increased levels of autoantibodies and increased production of pro-inflammatory cytokines by Th1 and Th17 lymphocytes were found

in Hashimoto's disease patients with vitamin D deficiency compared with a group of healthy subjects [45]. Low vitamin D levels were associated with higher TSH and mild cognitive impairment in a group of patients diagnosed with Hashimoto's disease [46]. Other study showed reduced vitamin D levels, but only with the duration of the disease and not immediately from the time of diagnosis [47]. Vitamin D supplementation at a dose of 50,000 IU per week for 3 months significantly attenuated the autoimmune response by reducing the production of pro-inflammatory cytokines, having a beneficial effect on the T-lymphocyte system, inhibiting thyroid cell damage and reducing antithyroid antibodies [48]. Studies of the effect of vitamin D on the pathogenesis of Hashimoto's disease are inconclusive, but some show multiple benefits of supplementation. Further studies will show whether exogenous vitamin D supplementation has a real impact on the clinical picture of the disease, whether this intervention will become standard of care, and what the optimal doses of vitamin D are for patients to benefit most from such treatment.

Vitamin B12

Vitamin B12, or cobalamin, is an essential component in the formation of red blood cells and is needed for many enzymes that the body needs to function properly [49]. The main sources of vitamin B12 are foods such as red meat, milk, eggs and dairy products [50]. Patients with Hashimoto's disease have been shown to have a significantly higher prevalence of vitamin B12 deficiency than healthy people. Increased levels of anti-TPO antibodies have also been found in patients with autoimmune thyroiditis [51]. Malignant anemia is significantly more common in patients with Hashimoto's disease because of the increased risk of other autoimmune diseases. It is caused by vitamin B12 deficiency [52]. Because of the increased likelihood of vitamin B12 deficiency, it is recommended that patients have their vitamin B12 levels monitored. Further research is needed to determine whether vitamin B12 supplementation is worthwhile and at what dose [53].

CONCLUSION

Hashimoto's disease is a disease for which there is still no causal treatment. Patients are treated with thyroid hormone replacement therapy. However, hormone treatment does not directly influence autoimmune, oxidative and inflammatory processes. In genetically predisposed patients, environmental factors over which the patient has control play an important role in the pathogenesis of the disease. The presence of these factors often determines the disease activity, the patient's condition and the severity of the autoimmune processes. Micronutrients such as iodine, selenium, iron, magnesium, zinc and vitamins B12 and D are an important part of the environmental factors. The antioxidant properties of selenium, zinc, iron and magnesium help to control disease activity, reduce inflammation that damages thyroid cells and reduce anti-thyroid antibody parameters. Selenium also increases the number of regulatory T lymphocytes, which inhibit the autoimmune process. In patients with Hashimoto's disease, it is recommended that iodine levels be monitored to minimize the adverse effects of both deficiency and excess. It is also advisable to check selenium, magnesium, zinc and vitamin levels regularly. Particular attention should be paid to patients with malabsorption and inflammatory bowel disease, in order to establish a balanced diet, in collaboration with endocrinologists and dieticians, to limit dietary deficiencies of these micronutrients. The use of prescribed pharmacotherapy and appropriate dietary management in patients with autoimmune thyroiditis improves quality of life, reduces complications and controls disease activity. Currently, micronutrient supplementation is not included in the guidelines of the endocrine societies for Hashimoto's disease, but promising results suggest that further research is needed to standardize and expand the treatment of this disease to include supplementation with these components.

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Introduction and objective. Hashimoto's disease is a chronic autoimmune thyroiditis. The chronic process of this disease leads to hypothyroidism. Various micronutrients and vitamins play an important role in the course of the disease and may have a significant impact on the development and treatment of Hashimoto's disease. The aim of this study is to review the literature to fully assess the impact of micronutrients on symptoms, prognosis and dynamics of change in the context of Hashimoto's disease. Articles were searched by entering keywords in PubMed databases. Scientific articles covering the period from 2016 to 2024 represent 76% of the items included in the literature. Studies from specialized research centers including meta-analyses, double-blinded randomized trials, large cross-sectional studies were considered. Studies from inexperienced centers with methodological flaws were discarded.

Abbreviated description of the state of knowledge. Current treatment of clinically overt hypothyroidism is based on the use of levothyroxine. The dose of substitution treatment is determined individually for the patient depending on the symptoms observed, TSH values and age. Currently, there are no recommendations for supplementation of microelements for the disease, which is due to the need to expand research and determine which micronutrients are worth taking to increase the quality of life of patients.

Conclusion. The number of cases of Hashimoto's disease is steadily increasing worldwide. Increasing attention is being paid to elements of the patient's diet and environment, in addition to the substitution treatment itself. The modification of a broadly defined lifestyle and the influence of environmental factors may in the near future find a place in the standard therapeutic management of patients with chronic autoimmune thyroiditis.

Keywords: Hashimoto's disease, micronutrients, vitamins, pathophysiology, supplementation, review.

РОЛЬ ОКРЕМИХ МІКРОЕЛЕМЕНТІВ ТА ВІТАМІНІВ ПРИ ХВОРОБІ ХАШИМОТО

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Хвороба Хашимото — хронічний аутоімунний тиреоїдит. Хронічний процес цього захворювання призводить до гіпотиреозу. Різноманітні мікроелементи та вітаміни відіграють важливу роль у перебігу захворювання та можуть мати значний вплив на розвиток та лікування хвороби Хашимото. **Метою** цього дослідження є огляд літератури для повної оцінки впливу мікроелементів на симптоми, прогноз і динаміку змін у контексті хвороби Хашимото. Пошук статей проводився за допомогою введення ключових слів у базах даних PubMed. Наукові статті, що охоплюють період з 2016 по 2024 рік, становлять 76% статей, включених до літератури. Були розглянуті дослідження спеціалізованих дослідницьких центрів, включаючи мета-аналізи, подвійні сліпі рандомізовані дослідження, великі перехресні дослідження. Дослідження недосвідчених центрів з методологічними недоліками були відкинуті.

Скорочений опис рівня знань. Сучасне лікування клінічно вираженого гіпотиреозу базується на застосуванні левотироксину. Доза замісної терапії визначається індивідуально для пацієнта залежно від виявлених симптомів, показників тиреотропного гормону і віку. Наразі немає рекомендацій щодо прийому мікроелементів при даному захворюванні, що пов'язано з необхідністю розширити дослідження та визначити, які мікроелементи варто приймати для підвищення якості життя пацієнтів.

Висновок. Кількість випадків хвороби Хашимото невпинно зростає у всьому світі. Крім самого замісного лікування, все більша увага приділяється елементам раціону та навколишнього середовища пацієнта. Модифікація широко визначеного способу життя та вплив факторів навколишнього середовища можуть найближчим часом знайти місце в стандартній терапії хворих на хронічний аутоімунний тиреоїдит.

Ключові слова: хвороба Хашимото, мікроелементи, вітаміни, патофізіологія, добавки, огляд.