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THE INFLUENCE OF DIETARY AND LIFESTYLE FACTORS ON PSORIASIS

Wpływ czynników dietetycznych i stylu życia na przebieg łuszczycy

Streszczenie
Łuszczyca to przewlekła, zapalna choroba skóry. Obejmuje wiele postaci, które różnią się obrazem klinicznym od siebie. Istotne znaczenie w patogenezie łuszczycy i w prowokowaniu zaostrzeń objawów, przypisuje się czynnikom środowiskowym. Istnieją pojedyncze doniesienia, że objawy łuszczycy może nasilać podaż glutenu w diecie pacjenta. Dane dotyczące współistnienia tej dermatozy z nadwrażliwością na gluten mimo, iż są wciąż niejednoznaczne, to sugerują, że ten czynnik może mieć wpływ na przebieg choroby. Badania wskazują również na związek łuszczycy z występowaniem otyłości. Zwraca się również uwagę na fakt, iż odpowiednio skomponowana dieta umożliwia regulację mechanizmów zapalnych, mających ogromne znaczenie w tej jednostce chorobowej. Liczne badania potwierdzają też częstsze występowanie nałogu tytoniowego i alkoholowego u pacjentów z łuszczycą.
Słowa kluczowe: łuszczyca, dieta, otyłość, gluten, styl życia

Abstract
Psoriasis is a chronic, inflammatory skin disease. It includes many forms that differ from each other in the clinical picture. In the pathogenesis of psoriasis and in exacerbations of symptoms, environmental factors are important. There are individual reports that the symptoms of psoriasis may increaseas a result of increased gluten supply in the patient's diet. Data concerning the coexistence of this dermatosis with hypersensitivity to gluten (although it is stillinc onclusive) suggests that this factor may affect the course of the disease. Researches also indicate a relationship between psoriasis and the occurrence of obesity. Furthermore, attention is drawn to the fact that a properly composed diet enables regulation of inflammatory mechanisms that are extremely important in this disease. Numerous studies also confirm more frequent occurrence of tobacco and alcohol addiction among patients with psoriasis.
Keywords: psoriasis, diet, obesity,gluten, lifestyle

Psoriasis is a chronic, inflammatory skin disease. It was described for the first time more than 200 years ago [Stawczyk et al., 2011]. This is a recurrent disease that affects the skin and the osteoarticular system [Wasiluk, Ostrowska and Stefańska, 2012]. It is observed in many forms that differ from each other. Common psoriasis is the most common variety [Jabłońska and Majewski, 2008]. About 2-3% of the world's population and about 1 million people in Poland suffer from this disease [Neneman and Adamski, 2009]. Taking into account the size, location and nature of lesions on the skin, several forms of psoriasis are distinguished: psoriasis gutatta, psoriasis vulgaris palmo-plantaris, chronic plaque psoriasis, psoriasis inversa,

psoriasis capitis, psoriasis unguium, psoriasis pustulosa, erythrodermia psoriatica or psoriasis arthropatica [Wolska and Langner, 2006].

Psoriasis is a common dermatosis, the occurrence of which depends on geographical, ethnic and racial factors. It is rarely observed among people of black race, more often among people of oriental origin, and most often in the Caucasian race (2% of the population). The reasons of these differences in the incidence has not been fully understood. They may depend on genetic and environmental factors. The illness occurs with similar frequency in both sexes [Łuczkowska and Żaba, 2005]. The first signs of psoriasis can appear at any age. About 85% of patients are diagnosed before the age of 30 [Stawczyk et al., 2011]. It occasionally appears in the neonatal of infant age [Wasiluk, Ostrowska and Stefańska, 2012].

Psoriasis has been classified as one of complex diseases with a multi-genetic and multifactorial inheritance model. Its essence is excessive epidermal proliferation. It is considered that the disease process is mediated by T lymphocytes. Activation of auxiliary T lymphocytes (Th1) plays a key role in the formation and maintenance of psoriatic lesions. They dominate on both early and fully developed skin lesions. These cells support cellular immunity through the activation of cytotoxic T lymphocytes and macrophages. Auxiliary T lymphocytes produce numerous pro-inflammatory cytokines. They include: interleukin 2 (IL-2), 3 (IL-3), 12 (IL-12), tumor necrotic factor alpha and beta (TNF-alpha and TNF-beta) and interferon gamma (IFN-gamma) [Stawczyk et al., 2011]. Despite the fact that the role of T lymphocytes in the pathogenesis of psoriasis raises no doubts, it is still unknown what causes their activation and against which antigens the inflammatory response is directed. In the literature, an initiating role is attributed to streptococcal superantigens. They can stimulate polyclonal proliferation of T lymphocytes through direct attachment to particles of the class II major histocompatibility complex (MHC) on antigen-presenting cells. This leads to the release of pro-inflammatory factors, which causes stimulation of keratocytes and psoriatic epidermal proliferation [Nakajima, 2012; Zheng et al., 2007].

A characteristic feature of psoriasis is phenotypic heterogeneity. A plaque form occurs in 90% of patients. It is characterized by the occurrence of disease outbreaks in the form of red plaques that are often covered with silver-gray scale. These lesions are usually located on elbow skin, knees skin and in the area of scalp. Other clinical forms are less common [Stawczyk, 2011].

In the formation of psoriasis and exacerbations of symptoms, environmental factors are important. They include: infections, stress, injuries, alcohol and nicotine [Antosik, Krzęcio-Nieczyporuk and Kurowska-Socha, 2017]. Moreover, it should be noticed that an inadequate unhealthy lifestyle, and thus an incorrect diet, can intensify inflammatory processes. This factor has an impact on the clinical course of the disease, treatment and occurrence of complications [Wasiluk, Ostrowska and Stefańska, 2012].

There are individual reports that symptoms of psoriasis, atopic dermatitis or rosacea may increase after intakeof gluten in the patient's diet. These diseases belong to the circle of autoimmune/inflammatory diseases, coexisting with other diseases of this group, including celiac disease. There are studies showing that atopic dermatitis is more common among patients diagnosed with celiac disease and among their relatives. Furthermore, the relationship between psoriasis and celiac diseases is the interesting topic for researchers. Despite the fact that available data about the coexistence of these diseases is still ambiguous, it suggests that psoriasis may coexist with unrecognized celiac disease because the presence of anti-gliadin antibodies in these patients is more common. Some authors suggest that a gluten-free diet for patients with psoriasis improves the course of the disease.

Therefore, it can be assumed that the confirmation of the hypothesis about the presence of elevated levels of antibodies observed in the case of celiac disease in patients with psoriasis, atopic dermatitis or systemic lupus may widen the panel of diagnostic tests that should be conducted and change their dietary recommendations. It should be assumed that the introduction of a gluten-free diet will improve their quality of life and/or reduce the symptoms of the disease. However, this has not been supported by any scientific research yet.

In recent years, the gluten-free diet has become very fashionable and the unreasonable elimination of gluten, without the support of diagnostic tests, happens very often. Such a gluten-free diet must be strictly observed by people with diagnosed celiac disease. Neglecting nutritional restrictions for such patients can

lead to the atrophy of intestinal villi, limiting the absorption of nutrients and consequently – malnutrition. On the other hand, a total gluten-free diet used throughout life enables patients to avoid these complications.

Gluten is a toxic factor in celiac disease, allergy (especially to wheat), as well as in gluten sensitivity, recently described by scientists and doctors. Gluten sensitivity is a special form of gluten intolerance that may affect up to 6% of population. Currently conducted scientific research indicates that an incorrect reaction to gluten may be a pathogenic factor in a number of diseases [Wasiluk, Ostrowska and Stefańska, 2012].

Gluten is defined differently. FAO/WHO defines it as a water insoluble protein fraction that occurs in wheat, barley, rye, oats and hybrid varieties, e.g. triticale [Badiu et al., 2014]. The definition of gluten presented by the European Commission is similar, and it refers to its intolerance [Rossel et al., 2014; Wojtasiak and Kunachowicz, 2014]. In medical sciences, the definition of gluten only refers to the ethanol soluble fraction of prolamins, including gliadin in wheat, hordein in barley and secalin in rye. Gluten is a mixture of glutenin, gliadin, albumin and globulin.

The pathogenesis of gluten-dependent enteropathy (celiac disease) is determined by genetic, environmental and immunological factors [Kruszewski, 2001; Andrych, Iwona and Kryszewski, 2006; Czerwionka-Szafarska, Szafarska-Popławska and Muller, 2006]. Approximately 90-95% of patients with celiac disease have the HLA-DQ2 gene, and the remaining group of patients have the HLA-DQ8 gene. Celiac disease is transmitted in an autosomal dominant mode [Jarosz and Dzieniszewski (ed.), 2005]. An environmental factor that induces the disease is the presence of specific fractions of cereal proteins belonging to prolamins in the diet. These proteins include: gliadin (wheat protein), secalin (barley protein), hordein (rye protein) [Karczewska, 2004; Szajewska, 2008]. As a result of digestion of gliadin by gastric and pancreatic enzymes, toxic gliadin peptides are formed in predisposed persons. They present high affinity for HLA-DQ2 and HLA-DQ8 molecules.

A fragment of gluten (polypeptide) is responsible for the activation of the immune system. It consists of 33 amino acids (including glutamine and proline residues) and is resistant to stomach acid and proteolytic enzymes. An important factor is the presence of the tissue transglutaminase (tTG), which is involved in gluten metabolism. The mechanism initiates contact of gluten with the intestinal mucosa. The above-mentioned gluten proteins (characterized by a high content of glutamine) are a substrate for tissue transglutaminase. This enzyme causes deamination of glutamine to glutamic acid. This leads to almost hundred-fold increase in affinity of toxic peptides to HLA molecules and activation of gliadin-specific T lymphocytes (responsible for the initiation of inflammatory processes in the intestine) [Jarosz (ed.), 2011].

Immunological theory assumes the existence of enzymatic disorders that cause abnormal digestion of gluten. In the undegraded form, it is toxic to intestinal cells and causes immune reactions. The synthesis of anti-gliadin antibodies occurs, and T lymphocytes appear in the small intestinal mucosa. These, in turn, prompt cytokines (that stimulate endothelial and cytotoxic lymphocytes, as well as plasma cells) to produce anti-gluten antibodies directed against tissue transglutaminase (anti-tTG) and smooth muscle endomysium (IgAEmA) [Karczewska, 2004; Jarosz and Dzieniszewski (ed.), 2005; Andrych, Iwona and Kryszewski, 2006; Czerwionka-Szafarska, Szafarska-Popławska and Muller, 2006].

In contrast to celiac disease, the mechanism of gluten intolerance pathogenesis (NCGS) has not been determined. Available evidence indicates that several mechanisms play an important role in its development: activation of innate immune response [Sapone, 2011], change in the barrier function of the intestinal mucosa [Wolta and de Gorgio, 2012], or eating foods with amylase inhibitors [Junker et al., 2012]. Non-celiac gluten sensitivity seems to be associated with the activation of a non-specific immune response to gluten – however, in a mechanism than differs from the celiac disease. Auto-immune processes do not occur then. It is assumed that this type of hypersensitivity does not have a genetic nature. Therefore, genetic examination is not reliable [Konińska et al. (ed.), 2017].

The importance of multi-component food hypersensitivity associated with a diet rich in FOODMAPs or food additives (stimulating the occurrence of gastrointestinal symptoms and stimulating the intestinal nervous system) has also been analysed [Gibson and Sheperd, 2012]. The majority of patients with hypersensitivity to gluten also present the hypersensitivity to other nutrients.

There are reports that indicate the legitimacy of a gluten-free diet for people with psoriasis. The first reports indicating a relationship between enteropathy and malabsorption syndrome with this disease ap-

peared in the 1980s [Hendel et al., 1982].There are studies that indicate a higher incidence of psoriasis among people with celiac disease, especially latent celiac disease [Humbert et al., 1991; Chalmers and Kirby, 2000]. In patients with gluten intolerance, there is a change in the structure of the small intestine and symptoms of malabsorption. This is associated with increased permeability of the intestinal mucosa. This may be the reason for an increase in migration of bacteria, which (acting as superantigens) cause or exacerbate psoriatic lesions [Humbert et al., 1991].

In the study by Michaelsson et al., it was shown that among patients with psoriasis (with anti-gliadin antibodies (AGA) in blood serum), the use of gluten-free diet led the improvement of the course of the disease. 30 AGA-positive and 4 AGA-negative patients took part in this study. After three months of using a gluten-free diet, the improvement in skin condition was observed in AGA-positive patients [Michaelsson et al., 2000]. Clinical cases confirming the coexistence of psoriasis and gluten intolerance have also been described in the literature. Addolarato et al. [2003] described a case of a patient with psoriasis and gluten intolerance. After the use of a gluten-free diet by this patient, skin lesions completely disappeared. Moreover, the study described a patient with psoriasis and diagnosed osteomalacia. One month after the introduction of the gluten-eliminating diet by this patient, all symptoms disappeared [Frikha, Snoussi and Bahloul, 2012]. Wolters [2005] cites a study, in which 33 people with psoriasis and high levels of AGA antibodies and 6 people with normal levels of gliadin anti-bodies took part. The examined people used a gluten-free diet for 3 months. In the group of 30 patients, the severity of lesions was reduced. There was no improvement among patients with normal antibody levels. Eighty-two percent of patients, who achieved improvement also had lower levels of anti-gliadin antibodies. In the next stage, the respondents returned to their old eating habits. After 3 months, the lesions intensified in 18 people out of 30 patients in the group with elevated antibody levels.

Some research presented a higher AGA antibody titre in psoriasis patients compared to healthy people. In tests, Michaelson et al. [1993] confirmed the presence (AGA in the IgA class) in 16% of the 302 tested patients. However, Sultan et al. [2010] did not confirm these observations.

Zamani et al. [2010] examined the presence of antibodies considered to be more specific for celiac disease than AGA, namely: anti-tissue transglutaminase (tTGA) and anti-endomysium (EMA) antibodies in 328 patients with psoriasis. Only 3 people showed their elevation and only 1 patient was diagnosed with celiac disease.

Wu et al. [2012] proved that patients with psoriasis have a higher risk of celiac disease compared to a healthy population. The study determined the relationship between psoriasis and 21 common autoimmune diseases. A retrospective cohort study compared 25,341 psoriasis patients with over 125,000 control patients in the American database of the Kaiser Permanente hospital system in Southern California, well-matched by age and gender. The study found that in the general population there was a higher incidence of psoriasis together the celiac disease than the celiac disease alone. A similar study was carried out. It compared 12,502 psoriasis patients with 24,285 people, who were marched for age and gender with the use of an Israeli medical database. It was found that the occurrence of celiac disease is 0.29% in patients with psoriasis compared to 0.11% in the control group ($p < 0.001$) [Birkenfeld et al., 2009].

Ludvigsson et al. [2011] examined celiac patients in order to check whether they have an increased risk of psoriasis. A retrospective cohort study was performed in Sweden, and the group included 28,958 patients with celiac disease confirmed by a bowel biopsy. The control group consisted of 143,910 persons of the right age and sex. The authors showed that people with celiac disease had a higher risk factor for the development psoriasis in the future than the remaining examined persons.

Bharia et al. [2014] emphasized that there are many studies that indicate the relationship between psoriasis and a pathological response to gluten. The authors analysed reports published in the years 1960-2012 in English. A meta-analysis demonstrated that patients with psoriasis have a 2.4-fold higher risk of elevated levels of anti-gliadin antibodies. AGA IgA antibodies were present in 14% of patients with psoriasis and 5% of healthy persons covered by the tests. The authors point out that psoriasis does not need to be necessarily associated with gluten enteropathy, but it may be connected with gluten sensitivity. However, they showed that the coexistence of these diseases should be the subject of further researches. Undoubtedly, such studies

are necessary due to the low specificity of anti-gliadin antibodies in gluten-dependent enteropathy and the lack of studies on the level of anti TG and anti IgAEmA antibodies in this population.

Psoriasis is a systemic disease. It is often accompanied by other chronic inflammations. The affected patients may also have: ulcerative colitis, diabetes, hypertension or metabolic syndrome. Chronic inflammatory process plays a role in the formation of inflammatory changes in blood vessels, leading to endothelial dysfunction, changes in glucose metabolism or insulin resistance. The consequence of this state is the accelerated development of atherosclerotic plaque. People with psoriasis have an increased risk of cardiovascular disease, including acute coronary syndromes [Sikora-Grabka, Adamczak and Więcek, 2012].

There is no doubt that properly composed diet can contribute to the improvement of the health and well-being of these patients [Wolters, 2005]. Researches indicate a relationship between psoriasis and obesity [Sterry, Stober and Menter, 2007]. However, it is unknown whether increased body weight is a consequence of psoriasis or a risk factor concerning the morbidity [Mallbris et al., 2005]. However, it has been shown that psoriasis has a more severe clinical course among people with obesity than in a group of people with normal body weight [Wolk et al., 2009]. Psoriasis and obesity have common inflammatory pathogenesis. In people with excessive body weight, adipocytes are characterized by a pro-inflammatory secretory profile and this is connected with overproduction of pro-inflammatory cytokines such as: TNF-alpha, IL-6, IL-8, leptin, visfatin and plasminogen activator inhibitor 1 (PAI-1) [Cohen et al., 2008]. It has been proven that the use of a low-energy diet (that reduces body weight) also reduces the levels of inflammatory cytokines and markers in the blood serum (C-reactive protein; CRP, IL-6, receptor for TNF-alpha) and reduces systemic features of inflammation [Nicklas, Ambrosius and Messier, 2004].

Inflammatory mechanisms can also be stimulated by adequate supply of polyunsaturated fatty acids (PUFAs) that are called essential saturated fatty acids. The group of these fats is divided into omega-3 (n-3) and omega-6 (n-6) acids. The most important representatives of the group of omega-3 acids include: alpha-linolenic acid and eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA). On the other hand, in the omega-6 group, the most important are: linoleic acid and arachidonic acid. Polyunsaturated fatty acids are products necessary for the synthesis of eicosanoids. These, in turn, are compounds that regulate the activities of many tissues and organs. The high supply of linoleic acid contributes to the synthesis of arachidonic acid. Omega-6 acids intensify pro-inflammatory, thrombotic and vasoconstrictive processes [Jarosz (ed.), 2011].

The supply of omega-3 acids has the opposite effect. A properly composed diet enables to regulate inflammatory mechanisms. The supply of alpha-linolenic acid intensifies the transformation of acids from omega-3 family and inhibits reactions with the participation of omega-6 acids. The ratio of omega-6 acids to omega-3 acids in the diet plays a key role. Prevention of chronic inflammatory diseases is based on achieving their correct ratio. The source of omega-6 acids is meat and all products of its origin, as well as processed foods also called the „convenience foods". The source of omega-3 acids is oil plants, vegetable oils and (above all) fish [Jarosz (ed.) 2011].

Therefore, in the case of psoriasis, a Mediterranean diet is recommended. This diet is rich in fish. There are researches indicating that the supply of fish oil among people with psoriasis may reduce the severity of skin lesions [Maurice, Allen Barkley, 1987; Lassus, Dahlgren and Halpern, 1990]. Another diet that also reduces the supply of arachidonic acid is a vegetarian diet. It is based on the consumption of groats, cereals, legumes, vegetables and fruits, nuts or mushrooms. Products of animal origin are eliminated. Since oxidative stress plays a key role in the pathogenesis of psoriasis, the use of this type of diet may reduce the severity of psoriatic lesions due to the consumption of large amounts of vegetables and fruits, which are a source of antioxidants [Lithell et al., 1983; Yildirim, Inaloz and Baysal, 2003; Szeto, Kwok and Benzie, 2004].

Numerous studies confirm the increasing incidence of tobacco and alcohol addiction among patients with psoriasis. Studies concerning this disease in the Chinese population have shown a relationship between smoking, alcohol addiction and the HLA-DQA1 * 0201 allele, which is more frequently detected in patients with psoriasis [Zheng et al., 2004].

Nicotine promotes inflammatory processes mediated by Th1 cells. This may explain the relationship between smoking and the described dermatosis [Guliver, 2008]. It has been shown that people suffering from psoriasis smoke cigarettes twice as often compared to the general population. The majority of patients

smoked before the appearance of the first symptoms of the disease [Guliver, 2008]. Wolk et al. [2009] theorized that tobacco addiction is associated with even 70% risk of the occurrence of first symptoms of psoriasis. Particularly strong association has been demonstrated between smoking and pustular psoriasis of the hands and feet [Naldi, Peli and Parazzini, 1999; Guliver, 2008]. Thomsen and Sorensen [2010] have shown that smoking raises the risk of appearance of the disease, but also increases the symptoms of existing psoriasis. An Italian study has shown that smoking more than 20 cigarettes a day doubles the risk of developing a more severe clinical psoriasis [Fortes et al., 2005]. In Sweden, attention has been paid to the increased incidence of smoking-dependent cancers among psoriasis patients [Thomsen and Sorensen, 2010]. In Norway, the study was conducted on a population of nearly 19,000 people with psoriasis, atopic dermatitis and allergic eczema. At that time, the influence of smoking on the occurrence of these diseases was assessed. This relationship has only been demonstrated for psoriasis in a group of men [Bø et al., 2008]. On the other hand, Behnam et al. suggested 3.3-times higher risk of developing plaque psoriasis among women, who smoke cigarettes [Behnam, Behnam and Koo, 2005]. There is also another Italian multicentre study reporting that the risk of developing psoriasis is higher among smokers, but also among people, who smoked in the past – compared with a population of people, who did not report this addiction. This correlation was stronger in women than in men [Naldi, Peli and Parazzini, 1999]. Setty et al. suggested that exposure to tobacco smoke in the prenatal period and childhood may result in an increased risk of psoriasis in the future [Setty, Curhan and Choi, 2007].

Furthermore, there are studies in the literature that indicate more frequent and excessive alcohol consumption by patients with psoriasis. Among persons hospitalized for this reason in Finland in the years 1973-1995, a large cohort study was conducted. It was observed that the main cause of mortality in this group of patients was excessive alcohol consumption [Poikolainen, Karvonen and Pukkala, 2008]. Gulliver [2008] demonstrated that patients with moderately severe and severe psoriasis abuse alcohol up to twice as often. Wolk et al. [2009] noticed a positive correlation between alcohol consumption and the occurrence of the first symptoms of psoriasis among men. This thesis was confirmed by Poikolainen et al. [1990]. They stated that alcohol addiction is a risk factor for the development of this disease, especially in young and middle-aged men.

Conclusions

Psoriasis is a chronic, inflammatory skin disease. Mechanisms of psoriasis pathogenesis are still under investigation. Currently, it is believed that genetic and environmental factors in connection with impaired mechanisms of the immune response play a role in psoriasis. Based on the collected data, it can be assumed that a broadly understood healthy lifestyle plays a role in the prevention and treatment of this disease. There are no precisely defined standards regarding nutritional recommendations related to psoriasis. Nutritional treatment in psoriasis is determined by the patient's comorbidities and requires an individual approach. Limitation in kilocalories contributes to the normalization of body weight, and thus reduces the inflammation process and improves the condition of the state of the affected skin. It should be noted that an increase of the supply of fruits and vegetables rich in carotenoids, flavonoids and vitamin C in the diet is required. It is important to ensure adequate supply of omega-3 polyunsaturated fatty acids. The main source of these acids is marine fish. The consumption of animal products should be limited. Elimination of the use of stimulants improves the skin's condition in the course of the disease. On the other hand, introduction of a gluten-free diet without confirmation of gluten-sensitive enteropathy or gluten sensitivity is not justified. In the case of improvement of psoriatic lesions after elimination of gluten, the presence of markers of pathological response to gluten should be confirmed in diagnostic examinations.

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