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Diet therapy as support for the treatment of atopic dermatitis

Dietoterapia jako wsparcie leczenia atopowego zapalenia skóry

ABSTRACT

Atopic dermatitis (AD) is a chronic disease with a genetic basis. The physiological markers of the disease are inflammation and abnormalities in the efficiency of the body's antioxidant system.

The aim of this study was to analyze the possibility of using diet therapy to alleviate the course of AD. Based on information present in the world literature, available in Web of Sciences, Scopus, PubMed, and Google Scholar databases, the effect of food active ingredients in the course of this chronic disease was investigated.

In the case of dermatoses, it is important both to eliminate foods that can exacerbate the course of the disease and to include in the menu ingredients that can alleviate the development of the illness. Diet therapy is the simplest and least cumbersome way to support the treatment of AD, even leading to the elimination of symptoms of the disease.

Keywords: atopic dermatitis, AD, diet therapy

STRESZCZENIE

Atopowe zapalenie skóry (AZS) jest chorobą przewlekłą o podłożu genetycznym. Markerami fizjologicznymi choroby są stan zapalny oraz zaburzenia w wydolności układu antyoksydacyjnego organizmu.

Celem pracy było przeanalizowanie możliwości zastosowania dietoterapii jako sposobu łagodzenia przebiegu AZS. Na podstawie informacji obecnych w światowym piśmiennictwie, dostępnych w bazach Web of Sciences, Scopus, PubMed oraz Google Scholar, przeanalizowano wpływ składników aktywnych obecnych w produktach spożywczych na przebieg tej przewlekłej choroby.

Przy dermatozach, istotne jest w aspekcie dietetycznym zarówno eliminowanie pokarmów, które mogą zaostrzać przebieg choroby, jak i włączenie do jadłospisu produktów spożywczych, które dzięki zawartości substancji czynnych mogą złagodzić przebieg choroby. Dietoterapia jest najprostszym i najmniej kłopotliwym sposobem wspomagania leczenia AZS, prowadzącym nawet do wyeliminowania objawów choroby.

Słowa kluczowe: atopowe zapalenia skóry, AZS, dietoterapia

INTRODUCTION

Atopic dermatitis (AD) is a chronic disease with a genetic basis. The genes attributed to AD are mainly located in chromosomal regions 3p, 3q, 4q and 18q [1]. The decisive genetic determinant of disease development is filaggrin polymorphism, but the influence of other genes with functions related to the immune system and extracellular matrix has also been suggested [1]. The aetiology of AD has also been linked to skin barrier defects, environmental factors and food allergies [2]. Individuals with AD show increased serum total IgE levels and reduced interferon-gamma (IFN-γ) synthesis and increased levels of

the interleukins IL-4, IL-5 and IL-13 [3]. Studies in AD mice have shown that IFN-γ reduces epidermal barrier function by affecting the fatty acid composition of ceramides [4]. Inflammation is induced by the pro-inflammatory interleukins IL-1, IL-6 and tumour necrosis factor α (TNF-α), produced by inflammatory epidermal dendritic cells [5]. In turn, IL-12 and IL-18 promote the transformation of the inflammatory response from Th2 to Th1/0 and entry into the chronic phase of the disease [5]. Persistent inflammation weakens the immune system and exacerbates skin symptoms (dryness, persistent pruritus, erythema), and the recurrent nature of the disease makes life less comfortable [6]. Increased oxidative stress parameters are also found in people with AD [6].

AD is diagnosed primarily by means of the clinical picture, based on the Hannifin and Rajka criteria, which include the so-called major symptoms (pruritus, chronic and recurrent course, location of the lesions and their morphology, history of atopy) and the so-called minor symptoms, of which there are 23, including dry skin, fish scales, cheilitis and facial erythema [7]. For the diagnosis of AD, simultaneous confirmation of three major and three minor symptoms is sufficient [7]. The disease is usually diagnosed in infancy (more than 50% of cases involve children up to 12 months of age), sometimes also at a later age (3-11 years) and even in adulthood [8]. However, it should be noted that most often the symptoms resolve with age, although this is not the norm [8]. During AD, co-morbidities are often found, mainly involving the respiratory and cardiovascular systems [9], but also diabetes and hyperlipidaemia [4]. It can also cause *Attention Deficit Hyperactivity Disorder* (ADHD), which results from ongoing psychological stress and sleep disturbances [10], as well as other neurological disorders such as depression and anxiety disorders and low self-esteem [9].

Proper skin care is recognised as the primary prevention of AD and as a means of relieving symptoms [11]. However, this is an action that does not eliminate the problem. Current pharmacotherapy methods are effective, but not without adverse side effects. Although AD is hereditary, a common cause of AD is food allergy [7], in which case an elimination diet should be used. Only the tendency to develop dermatosis is inherited, whereas its occurrence is determined by environmental stimuli [1]. Besides, inflammation [5] and disturbances in the capacity of the body's antioxidant system [6] are physiological markers of the disease. Through appropriate nutrition and lifestyle, the risk of these pathological conditions can be minimised. Oxidative stress may be a direct or indirect risk factor for dermatoses, or it may exacerbate pre-existing conditions. There is evidence that excessive amounts of reactive oxygen species, mediated by oxidative stress, are involved in many biological reactions, leading to DNA modification and enhancing lipid peroxidation and the production of pro-inflammatory cytokines [12]. Diet therapy is the simplest and least cumbersome way to support the treatment of AD, even leading to the elimination of disease symptoms. Diet can influence the course of atopic dermatitis not only negatively, exacerbating symptoms, but also positively, alleviating the course of dermatosis [2].

EFFECT OF NUTRITION ON SKIN HEALTH IN AD

There is a special connection between the gastrointestinal tract and the skin, as the skin shows the ability to store substances supplied with food. This is crucial when supporting the pharmacological treatment of dermatoses, including AD. With dermatoses, it is important both to eliminate foods that may exacerbate the course of the disease and to include in the diet foods that may alleviate the course of the disease due to their content of active substances (Table 1). Therefore, a rationally planned diet supported by the supplementation of certain ingredients may prove effective in the quest for skin improvement [2].

Table 1 List of foods with proven positive effects on AD

Products	Presence of substances with antioxidant and anti-inflammatory effects
Meat of fatty fish: tuna, herring, salmon, sprat, mackerel	Essential fatty acids (EFAs), including omega-3 fatty acids, vitamins A, D
Vegetable oils	EFAs, including omega-3 fatty acids, vitamins A, D, E, K, tocopherols, sterols
Tea	Polyphenols (including catechins)
Vegetables: celery, lettuce, broccoli, tomato, cauliflower, radish, kale	Vitamins C, E, carotenoids
Fruit: blueberries, blueberries, apricots, strawberries, apples, pears, currants	Vitamin C, carotenoids, anthocyanins, polyphenols
Eggs	Vitamins A, D, E, biotin, carotenoids
Vegetable silages, fermented dairy products, plant products enriched with probiotics	Probiotics

Source: [2, 13, 14]

FOOD INGREDIENTS EXACERBATING THE COURSE OF AD

Certain food components trigger skin reactions that can exacerbate AD symptoms and prompt sufferers to gradually exclude certain foods from their diet. AD and food allergy can co-exist, but this is not the norm; this problem affects approximately 30-40% of people with AD [15]. Therefore, elimination diets should be conducted with caution. Exclusion of foods may lead to nutrient deficiencies, especially bearing in mind that wheat, milk, egg and fish proteins, among others, are the most common triggers of skin lesions [2]. Studies confirm that children who are subjected to strict elimination diets consume less than 67% of the recommended daily

intake of essential vitamins and minerals [16]. Analysis of the menus of adults with AD has shown deficiencies in many nutrients, most notably vitamins A and C, which have antioxidant and anti-inflammatory effects and therefore support the treatment of AD [17]. It should be emphasised that there is currently insufficient evidence to recommend elimination diets for people with AD prophylactically [15, 18]. Nevertheless, the efficacy of elimination diets in AD has been indicated; however, this is probably related to the coexistence of food allergy [2, 18]. Besides, due to the lack of appropriately designed studies and small patient groups, strict elimination diets should not be recommended as a strategy in the management of AD.

It is common for parents to link their children's disease exacerbation to food additives (colours, sweeteners, preservatives). The fact is that carmine and tartrazine have been documented to be associated with AD [18]. Carmine (a red dye) is often added to cheese, processed fruits and vegetables, chewing gum, breakfast cereals, meat products and processed fish, among others. Tartrazine, a lemon-yellow dye, is added primarily to cheese, canned fruit and vegetables, soups, desserts and condiments [18].

FOOD INGREDIENTS THAT ALLEVIATE THE COURSE OF AD

The physiological markers of AD are inflammation and oxidative stress, and it is therefore necessary to minimise the risk of these pathological conditions or attenuate their impact on the body through appropriate nutrition [5, 6]. Food components that stabilise these parameters are mainly probiotics, essential fatty acids EFAs, antioxidant vitamins: A, C, D and E, as well as carotenoids and polyphenols (Fig. 1).

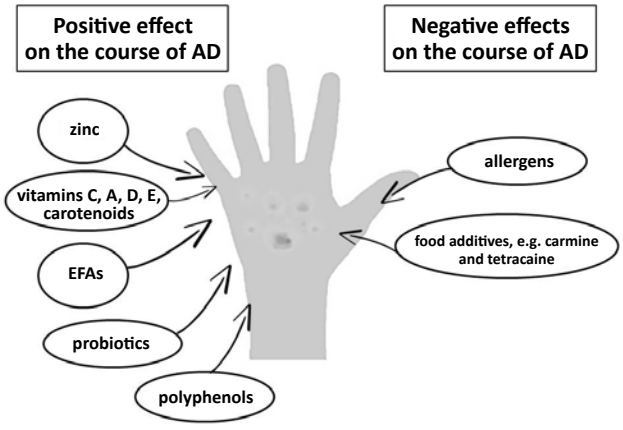

Fig. 1 Influence of food components on the course of AD
Source: Own elaboration based on [2, 14, 18]

Probiotics

Probiotics are live micro-organisms that exhibit immunomodulatory effects. The primary purpose of dietary supplementation with probiotics is to increase the population of beneficial bacteria in the gut, eliminate pathogens and bring about a permanent change in the composition of the intestinal flora. Probiotic bacteria carry out fermentation, primarily converting sugar to lactate, as well as to acetate and formate, so that the resulting acidic environment inhibits the growth of pathogenic microorganisms [19]. In the large intestine, probiotic bacteria compete for nutrients with pathogens and prevent them from adhering to epithelial cells. They also stimulate the production of immunoglobulins and, therefore, the amelioration of intestinal dysbiosis by probiotics is associated with a decreased autoimmune response, reduced inflammation and improved intestinal integrity by increasing protein expression in the intestinal epithelium [19]. The gut microbiome participates in nutrient metabolism, influences the production of pro-inflammatory and anti-inflammatory cytokines in the body, and exerts a destructive effect on pathogenic gut flora and neutralises toxins [2].

There is conflicting information in the available literature regarding the relationship between AD and the gut microbiota; however, inadequate microbial structure may be responsible for abnormal immune function, resulting in the development of AD [20]. The gut microbiota is able to modulate systemic inflammatory and immune responses, influencing the development of allergies; however, the molecular mechanisms underlying the anti-allergic effects of probiotics remain unclear and their efficacy is controversial [19]. Authors Kaliomaki et al. investigated the long-term effects of probiotic supplements in the nutrition of pregnant women and infants. The efficacy of the probiotic was verified after 2 and 4 years [21]. The cited authors found that already after two years, the incidence of AD among supplement users was twice as low as in the control group. Furthermore, it has been proven that newborns who had a varied intestinal microflora in the first week of life showed a lower predisposition to AD [20]. The inclusion of probiotics in the diet improves atopic skin as early as eight weeks [22]. The diet of people with AD should include probiotic products, primarily pickled vegetables and fruits, as well as fermented dairy products containing strains of probiotic bacteria from the *Lactobacillus, Streptococcus* and *Bifidobacterium* groups [14]. Dietary supplements and prebiotics, which selectively stimulate the growth and/or activity of the intestinal microflora, may also be good additions to the diet [19]. Paying attention to frequent consumption of probiotic foods may be one very important factor in eliminating the bothersome symptoms of AD.

Essential fatty acids

EFA deficiency is one of the main causes of skin disorders, as they are part of cell membranes and are precursors of immunomodulators [2]. EFAs maintain the proper structure, elasticity and function of cell membranes, are essential in the synthesis of intracellular lipids in the stratum corneum, and are precursors of eicosanoids and have regulatory properties [23].

EFAs are incorporated, among other things, into ceramides and metabolised in the dermis by cyclooxygenases and lipoxygenases, and thus act as bioprotectors [23].

EFAs cannot be synthesised by the human body and must therefore be supplied with food. The most important of these are *linolenic acid* (LA) (18:2 n-6), which is a precursor of arachidonic acid (ARA), prostaglandins, leukotrienes and thromboxanes. And also, α-linolenic acid (ALA) (18:3 n-3), which is a precursor of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) [2]. EPA is considered a major anti-inflammatory agent due to its competitive activity against ARA for metabolites, and the strong anti-inflammatory and immunomodulatory effects of DHA have also been demonstrated [23]. Importantly, LA is also a precursor of *gamma-linolenic acid* (GLA), which exhibits potent anti-inflammatory properties, so LA deficiency results in reduced levels of GLA in the body [2]. In people with AD, the conversion of LA to GLA may be impaired or insufficient due to a deficiency of delta-6-desaturase, so the dietary supply of EFAs plays a key role in the course of dermatosis [24]. Sources of LA are mainly sunflower, corn, peanut, soybean and grape seed oils. GLA is naturally found in evening primrose oil, hemp seed and borage oil [2].

Clinical studies to date support the efficacy of EFA supplementation in the treatment of AD, but the results are inconclusive. Studies in children with AD have shown that dysregulation of n-6 fatty acid metabolism is not associated with AD, but rather with IgE production and atopy of various origins [25]. On the contrary, in a study by Yen et al., deficits in n-6 EFAs in children were correlated with the severity of AD through effects on skin barrier function and inflammation [26]. An analysis of women's diet during pregnancy showed that prenatal EFA exposure was associated with the development of AD in the infant during the first 12 months of life [27]. In the study cited above, atopic eczema in the infant was associated with lower maternal n-3 and higher n-6 EFA supply, due to low fish consumption and high consumption of meat from slaughter animals. If this is the case, AD may also be associated with the presence of significant amounts of ARA in slaughtered animal meat [28]. Although metabolites derived from the oxidation of ARA do not initiate inflammation but contribute to it, they also lead to the generation of mediators responsible for the resolution of inflammation and wound healing [28].

Vitamin D

Vitamin D is recognised as effective in the treatment of various skin pathologies. It is involved in regulating the production of antimicrobial skin peptides, participates in the maintenance of the skin barrier and in the differentiation of keratinocytes [2]. Its deficiency weakens the protective skin barrier, which can lead to skin infection by, among others, *Staphylococcus aureus,* as people with AD show an ease of microbial colonisation on the skin [29]. It also shows immunomodulatory effects on both the innate and adaptive immune systems, which may explain the growing body of evidence linking vitamin D to allergic disease [2]. People with AD have lower serum vitamin D levels than healthy individuals [2]. These findings applied to people of all ages, but were particularly pronounced in children. In contrast, few studies have found no association between vitamin D levels and the risk of AD, such as a study conducted in Korea in a group of schoolchildren [30]. Other Korean studies have clearly indicated that low vitamin D levels are associated with AD in adults and are associated with an increased risk of osteoporosis and bone fractures [31].

Vitamin D synthesis in the human body takes place in the skin thanks to sunlight (UVB). A certain amount is also supplied through the diet: from foods of plant origin as vitamin D2 and of animal origin in the form of D3 [2]. Vitamin D is lipophilic, so its sources are fat-containing foods such as oily fish, oils, eggs, milk and dairy products, and it can also be supplemented [32]. The daily diet provides 100-200 IU of vitamin D, which covers about 10-20% of the requirement [32].

Vitamin C

Vitamin C (ascorbic acid) plays an important role in maintaining skin health and can promote keratinocyte differentiation and reduce melanin synthesis, leading to antioxidant protection against UV-induced photodamage [2]. It is involved in the formation of the dermal barrier and collagen in the dermis and in modulating cell growth and differentiation signalling pathways [2]. Vitamin C has strong antioxidant properties and therefore alleviates the course of dermatoses, in which oxidative stress is one of the characteristic physiological parameters. On the other hand, high doses have a pro-oxidant effect, which is probably why plasma vitamin C levels are regulated at the level of absorption and excretion [33]. In addition, there is a proven correlation between deficiency of this vitamin and the occurrence of allergies. Furthermore, the use of high doses of intravenous vitamin C has been shown to be effective in alleviating allergic symptoms [34]. It has been shown that as the clinical severity of AD increases, plasma vitamin C levels and epidermal ceramide levels decrease, suggesting that vitamin C metabolism and/or absorption may be impaired during AD [35]. Vitamin C is essential for the *de novo* synthesis of ceramides in the epidermis [35], and also exhibits anti-inflammatory effects [34]. The primary dietary sources of vitamin C are fresh fruit and vegetables [13].

Vitamin E

Vitamin E shows great potential as an AD treatment aid due to its antioxidant and anti-inflammatory properties. However, rare cases of skin allergy should be taken into account. Nevertheless, vitamin E shows moisturising and protective effects and protects cell membranes from lipid peroxidation and consequent oxidative damage [2]. Furthermore, lower levels of vitamin E are found in people with AD than in the

plasma of healthy individuals. A study in people with AD showed that orally applied vitamin E can improve symptoms and quality of life in patients with AD, and that no side effects are found at a dose of 400 IU/day [36]. Also, a study conducted in Italy among people with AD confirmed that there is a correlation between vitamin E intake, IgE levels and clinical symptoms of atopy [37], indicating that vitamin E can be a very good therapeutic tool in AD. Therefore, people struggling with AD should provide the body with adequate amounts of this vitamin in their daily diet. The best sources of vitamin E are vegetable oils, which additionally contain EFAs [2].

Carotenoids

Carotenoids have a positive effect on skin structure and function. In AD, photoprotective, antioxidant and anti-inflammatory effects are most important [2]. Carotenoid levels in the skin have been found to increase within 1-3 days after consumption of carotenoid-rich foods, including fruit and vegetables [38]. Beta-carotene has potent anti-inflammatory and anti-allergenic effects – improves skin condition in AD by strengthening the protective barrier. Lower levels of carotenoids and retinoids, as well as an altered ratio of lycopene isomers, have been shown in the plasma of people with AD compared to those without symptoms of the disease [39]. In contrast, studies in HR-1 mice with AD showed attenuation of skin symptoms as a result of dietary provision of carotenoids (lycopene and beta-carotene). The results were confirmed by histological analysis of skin sections taken [40]. Similarly, NC/Nga mice with AD showed an improvement in skin condition after oral astaxanthin treatment for 26 days, while blood analysis showed a reduction in inflammatory markers [41]. Very good sources of carotenoids are towering fruits and vegetables, as well as eggs, salmon meat and shellfish [2, 13].

Zinc

Zinc (Zn) is involved in the production of prostaglandins that regulate the secretory functions of the skin, influences collagen metabolism, and is also involved in the processing of fatty acids, thus influencing skin regeneration [2]. Zn deficiency impairs the capacity of the skin's immune system, increasing its susceptibility to dermatoses and allergies [2]. Zinc is part of the active centres of superoxide dismutase (SOD), a key endogenous antioxidant enzyme, and also displaces prooxidant iron and copper from lipids, proteins and *deoxyribonucleic acid* (DNA), inhibiting the production of oxygen free radicals [2]. This is all the more important as oxidative stress is one of the main physiological markers of AD. Zn deficiencies have been found in the hair and serum of children with AD [42]. Studies in mice with AD showed that zinc deficiency caused exacerbation of skin symptoms, increased serum IgE levels and increased production of pro-inflammatory cytokines, including IL-13 and γ-IFN, as well as reduced cell proliferation [43]. On the other hand, it has also been shown that Zn can decrease levels of pro-inflammatory cytokines, such as IL-6 and IL-1, while increasing levels of anti-inflammatory cytokines (IL-10) in mechanically damaged skin in mice [44]. Dietary zinc is primarily provided by milk, meat, liver, eggs, whole grain products and nuts and seeds [2].

Polyphenols

Phenolic compounds have a beneficial effect on the course of AD, as they exhibit antioxidant and anti-inflammatory effects [2]. They inhibit T-cell activation and proliferation, and thus can alleviate symptoms of dermatitis and contact hypersensitivity [45]. A study in mice with AD showed an improvement in skin health and a reduction in serum inflammatory markers after oral application of acacia bark polyphenol extract [46]. Furthermore, the cited authors linked the result to an improvement in the structure of the gut microbiota in AD mice. Oral administration of a mixture of polyphenols and anthocyanins isolated from *Vaccinium uliginosum* L. to AD mice for nine weeks resulted in relief of skin symptoms, as well as a reduction in IgE levels and pro-inflammatory markers [47]. A supplement containing isolated polyphenols from apples, administered to patients in oral doses of 10 mg/kg per day for 8 weeks, reduced inflammation, lichenisation, cracking and pruritus of the skin, with improvement sustained for 2 weeks [48]. In clinical studies, oolong tea was found to be therapeutically effective in recurrent AD, which may be due to the anti-allergic properties of tea polyphenols [49]. Studies conducted on isolated human keratinocytes have shown that quercetin present in tea may serve as a potential bioactive substance in the treatment of AD, due to its anti-inflammatory and antioxidant activity, leading to accelerated wound healing [50].

Polyphenols are found in fresh fruits (especially chokeberries, strawberries, strawberries, strawberries, raspberries, blueberries, blueberries, and blackberries) and vegetables (especially broccoli, red cabbage, garlic, red peppers, red beetroot) and their preparations, as well as herbs and spices [13]. The best dietary source of polyphenols is tea, especially green tea. Its antioxidant properties are due to its high content of polyphenols, primarily catechins, including epigallocatechin gallate (EGCG) found in green tea, quercetin, theaflavin and thearubigin present in black teas, and tannic acid [12]. The particularly high antioxidant activity of EGCG is determined by the presence of as many as eight -OH groups [12]. The content of total polyphenols in tea is 25-35% of the dry weight of the leaves [12].

SUMMARY

Atopic dermatitis (AD) is a chronic disease with a genetic basis. The physiological markers of the disease are inflammation and disturbances in the capacity of the body's antioxidant system, so it is necessary to minimise the risk of these pathological conditions or weaken their impact on the body

through appropriate nutrition. The simplest way to support the treatment of AD is through diet therapy, above all by eliminating foods that exacerbate the course of the disease (e.g. those containing allergenic substances, dyes, sweeteners, preservatives) from the diet. It may also be helpful to regularly consume foods that may alleviate the symptoms of AD due to their content of active substances. It should be emphasised that there is currently insufficient evidence to recommend an elimination diet for people with AD prophylactically, while food exclusion may lead to nutrient deficiencies. However, there is evidence that the introduction of foods with antioxidant and anti-inflammatory components into the diet of people with AD leads to an alleviated course of the disease. Probiotic foods, fruit, vegetables, whole grain cereal products, fish, nuts, oils and green tea should be included in the daily diet. These foods contain live cultures of probiotic bacteria and/or prebiotics, vitamins and provitamins, EFAs, phenolic compounds and antioxidant minerals.

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