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The role of diet in the pathogenesis and treatment of acne vulgaris

Trądzik zwyczajny – miejsce diety w patogenezie i terapii

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Abstract

Acne vulgaris is a common, chronic, multifactorial inflammatory skin disease. Although the disorder occurs primarily in young people, with a peak incidence in adolescence, it quite often persists into adulthood. The views on the impact of diet on the development and course of acne have changed dramatically over the years. Since the 1930s, patients have been advised to avoid chocolate and other sugar-containing products, fatty foods and sugar, and even reduce the overall intake of carbohydrates. Research in the years that followed undermined the influence of nutrition on acne, hence the use of specialised diets was not recommended. Since the beginning of the 21st century, there has been a certain renaissance in research on this relationship and a growing body of evidence has been accumulated to support the important role of different food products in the etiopathogenesis of acne. Most investigations focus on products with a high glycaemic index, as well as chocolate and dairy products. While the current research cannot be used as a basis for developing clear recommendations for patients, there is evidence that low-glycaemic index meals with reduced dairy, sugar, saturated fat and salt consumption, as well as increased omega-3 intake and probiotic supplementation may have a positive effect on the course of acne and its treatment. It should be remembered that although dietary interventions may support the therapeutic process, they will not replace appropriate pharmacological treatment adjusted to the severity of acne.

Keywords: acne vulgaris, diet, glycaemic index, chocolate, milk

Streszczenie

Trądzik zwyczajny (*acne vulgaris*) jest powszechnie występującą, przewlekłą chorobą zapalną skóry o podłożu wieloczynnikowym. Występuje przede wszystkim u osób młodych, ze szczytem zapadalności przypadającym na okres dojrzewania, jednak u wielu osób utrzymuje się także w wieku dorosłym. Poglądy na temat wpływu diety na rozwój oraz przebieg trądziku zmieniały się diametralnie na przestrzeni lat. Już od lat 30. ubiegłego wieku pacjentom zalecano unikanie spożywania czekolady i innych słodyczy, produktów tłustych i cukru, a nawet spożywanie mniejszej ogólnej ilości węglowodanów. Badania prowadzone w kolejnych latach podważyły wpływ odżywiania na trądzik, stąd stosowanie specjalistycznych diet nie było zalecane. Od początku XXI wieku nastąpił swoisty renesans badań nad tym związkiem i coraz liczniejsze dane wskazują na istotną rolę poszczególnych produktów żywnościowych w etiopatogenezie zmian trądzikowych. Przedmiotem zainteresowania badaczy są najczęściej produkty o wysokim indeksie glikemicznym, a także czekolada oraz nabiał. Choć obecnie dostępne wyniki badań nie mogą stanowić podstawy do stworzenia jednoznacznych zaleceń dla pacjentów, istnieją dowody na to, że spożywanie posiłków o niskim indeksie glikemicznym, z ograniczeniem spożycia nabiału, słodyczy, tłuszczów nasyconych i soli, a ze zwiększeniem podaży kwasów omega-3 oraz suplementacją probiotyków, może korzystnie wpływać na przebieg trądziku i jego leczenia. Należy pamiętać, że postępowanie dietetyczne może wspierać proces terapeutyczny, jednak nie zastąpi odpowiedniego leczenia farmakologicznego, dostosowanego do nasilenia trądziku.

Słowa kluczowe: trądzik zwyczajny, dieta, indeks glikemiczny, czekolada, mleko

EPIDEMIOLOGY AND PATHOGENESIS

Acne primarily affects young people between 11 and 30 years of age. The peak incidence is between 14 and 17 years of age in women and between 16 and 19 years of age in men; however, the condition can develop before puberty and persist into adulthood[1,2]. Primary eruptions, which include comedo, papules, pustules and inflammatory lesions, appear mainly in areas rich in sebaceous glands, i.e. the face, back and chest[1]. Facial acne affects 54% of women and 40% of men over the age of 25 years. It is mild in most individuals, whereas about 15% of patients develop severe acne with scarring and discoloration. Men seem to be more likely to develop more severe forms of the disorder[1,2]. The major processes seen in acne include overproduction of sebum, abnormal keratinisation within the pilosebaceous units, colonisation of sebaceous glands by anaerobic *Propionibacterium acnes* and inflammation. The pathogenesis of the disease process is complex and depends on the interaction of multiple factors responsible for the development and intensification of skin lesions. These include e.g.:

- overproduction of sebum by sebocytes (patients with acne secrete more sebum compared to healthy individuals);
- abnormal perifollicular keratinisation (keratinised epidermal cells are not shed off, corneocytes clump together, forming blackheads);
- colonisation of sebaceous glands by anaerobic *Propionibacterium acnes* (these bacteria produce lipases that degrade skin lipid layer, releasing free fatty acids and disrupting the epidermal keratinisation process);
- androgenic stimulation of sebaceous glands during puberty (leads to increased sebum secretion);
- genetic factors – the mode of inheritance has not been clearly defined yet; however, it is suggested that there is multigene or autosomal dominant inheritance with variable gene penetration; if both parents had acne, there is a high risk (>50%) of acne in the child;
- hormonal fluctuations in women associated with the menstrual cycle (60–70% of women experience acne exacerbation prior to the onset of their menses);
- pharmacotherapy (e.g. anabolic steroids, glucocorticoids, anti-epileptics, antidepressants, anti-tuberculosis drugs, barbiturates);
- psychological factors (increased stress is responsible for peak cortisol levels with androgen-like effects)[1–3].

Environmental factors are also important. The impact of climate (climatic zones, seasons), the level of urbanisation, skin exposure to sunlight, smoking and diet is questionable. Treatment is based on the use of topical preparations containing retinoids, antibiotics and benzoyl peroxide. Commonly used retinoids and antibiotics are reserved for severe acne[1].

HIGH GLYCAEMIC INDEX FOOD

Many studies currently point to the relationship between acne and Western diet, which is rich in simple sugars, refined grains, high-protein and dairy products, as well as sweetened beverages[4]. It has also been observed that acne does not affect societies that traditionally do not consume high glycaemic index (GI) products and milk. What is more, when members of these communities begin to follow a Western diet as a result of cultural changes or migration, the incidence of acne increases, reaching levels similar to those seen in Western populations[5]. It was found in recent years that insulin-like growth factor-1 (IGF-1) seems to play a particular role in the etiopathogenesis of acne. High IGF-1 levels have been shown to reduce the forkhead box protein O1 (FoxO1) transcription factor, which leads to the activation of the mammalian target of rapamycin complex 1 (mTORC1). The mTORC1 is involved in cell proliferation and metabolism, and mediates sebaceous gland hyperproliferation, lipid synthesis and keratinocyte hyperplasia, thereby contributing to the progression of acne lesions[4]. Physiological increase in IGF-1 is seen in adolescence, i.e. at the time of the highest prevalence of acne. It positively correlates with the severity of lesions in women, and with seborrhea in both women and men. Interestingly, acne does not occur in patients with Laron syndrome, with secondary IGF-1 deficiency[6]. IGF-1 and IGF-1R are overexpressed in acne lesions[7]. Importantly, high GI foods contribute to hyperglycaemia and hyperinsulinemia, which in turn increase the production of IGF-1. Additionally, hyperinsulinemia stimulates the production of androgens, which in turn increase endogenous IGF-1. This creates a vicious cycle that stimulates sebum overproduction[8]. Many studies have shown an adverse effect of a high GI diet on acne and a reduced number of acne lesions in individuals following a low GI diet. Smith et al. reported reduced total acne counts after a 12-week low-glycemic-load diet. They also found a positive effect of diet on weight loss, body mass index (BMI) and increased insulin sensitivity compared to the control group consuming foods high in carbohydrates[9]. Kwon et al. showed that following a low-glycemic-load diet for 10 weeks reduced the counts of acne lesions, the size of the sebaceous glands and inflammation[10]. Improvement in acne was already observed after a short-term, 2-week low GI diet[11]. Sugar and sweetened non-alcoholic beverages, the consumption of which is associated with an increased risk of moderate to severe acne, probably due to their effect on insulin secretion and an increase in blood glucose levels, also play an important role[12]. The Mediterranean diet, which has a preventive effect against acne, represents a model low GI diet[13]. Interestingly, evidence has been accumulated for the beneficial effect of metformin in the adjunctive treatment of moderate to severe acne in both slim and overweight individuals[14].

CHOCOLATE

Since chocolate is one of the most popular food products in the world, its influence on acne has been discussed by scientists for decades. Although clear evidence for the effect of chocolate on acne exacerbation is missing, many doctors notice the appearance of new lesions in their patients a few days

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after consuming products containing chocolate[15]. There is growing research that shows the negative impact of chocolate consumption on the course of acne. For example, it was shown that consuming 10 g of dark chocolate with 70% cocoa content daily for 4 weeks may intensify acne lesions by increasing both epidermal keratinisation and bacterial colonisation[16]. A single consumption of chocolate was also associated with acne exacerbation[17]. The mechanism underlying the relationship between chocolate consumption and acne intensification is not fully understood. It should be noted that in addition to cocoa, chocolate usually contains significant amounts of sugar, milk, fatty acids, phenylethylamine, caffeine and tryptophan (serotonin precursor), which can also contribute to acne exacerbation[15]. Since dark chocolate contains less sugar and fat, but more antioxidants, it may exacerbate skin lesions to a lesser extent[8].

DAIRY PRODUCTS

Milk is a heterogeneous product, therefore many different mechanisms underly its effects on the skin. Although milk has a low GI, it can increase blood glucose and insulin levels. Whey proteins are responsible for its insulinotropic effect, whereas casein stimulates IGF-1 production[8]. It has been shown that sports nutrition supplements containing whey proteins can cause acne[18]. Furthermore, milk contains bovine IGF-1, IGF-2 and steroidal substances such as androgens, 5α-pregnanodione and 5α-androstanedione[8,19]. High insulin levels have a negative effect on the skin condition as they may increase the secretion of androgens, gonadotropin and sex hormone binding globulin (SHBG), as well as enhance the secretion of sebum, while androgens themselves increase skin keratinisation and promote *P. acnes* colonisation of the hair follicles[20]. Whey proteins have a high leucine content, which affects the secretion of androgens and epidermal proliferation[21]. The unfavorable effects of milk were also attributed to its content of iodine or palmitic acid[21,22]. As a result of processing, skim milk has lower levels of estrogens, which have beneficial effects on the skin, and higher content of whey proteins[23]. Additionally, lower satiety after consuming skimmed milk may lead to its greater consumption compared to full-fat milk, which results in delivering more active milk substances to the body[19]. A meta-analysis of 14 studies in a total of 78,529 children, adolescents, and young adults found an increased risk of acne among those consuming skim milk, full-fat milk, yogurt, and cheese[23]. However, data on fermented milk products are inconclusive. They are believed to have a positive effect on the skin, which is attributed to the content of lactoferrin and probiotic *Lactobacillus* bacteria[8].

DIETARY FIBER

Research shows that a high-fiber diet can improve the skin condition in patients with acne[8]. The positive effect of a low GI diet may also result from its high fiber content[9].

FATTY ACIDS

The dietary ratio of omega-3 to omega-6 is a known modulator of inflammatory processes. High consumption of omega-3 fatty acids contributes to reduced production of pro-inflammatory cytokines, and thus improvement of acne lesions, as confirmed by research in groups consuming fatty fish and dietary supplements containing omega-3 fatty acids[24,25]. Omega-3 acids, eicosapentaenoic acid (EPA) found in fatty fish and seafood and γ-linolenic acid (GAP) contained in borage and evening primrose oil in particular, reduce the production of pro-inflammatory leukotriene B4 (LTB4), which stimulates sebum production in sebocytes. Additionally, omega-3 fatty acids contribute to reduced IGF-1, which is implicated in the pathogenesis of acne[4,22]. The negative impact of saturated fatty acids and trans fats on the course of acne has also been described[4]. It is worth noting that supplementation with omega-3 acids supports the proper functioning of the epidermal barrier, maintaining adequate skin hydration. Isotretinoin, which belongs to the group of retinoids generally used in moderate to severe acne, reduces the amount of sebum produced by the skin; however, most patients report dry skin and cheilitis during treatment. Omega-3 supplementation during isotretinoin treatment has been shown to reduce the incidence of these adverse effects, lowering transepidermal water loss[26]. Hypertriglyceridemia is another abnormality observed with systemic retinoids. Omega-3 acid supplementation has been shown to have a positive, stabilising effect on triglyceride levels in patients treated with isotretinoin[27].

FAST-FOODS

Attention should be paid to fast-food meals, which are usually characterised by a high GI value, and additionally a high content of saturated fats and salt. In their questionnaire study, El Darouti et al. showed a significantly higher daily sodium chloride intake in the group of individuals with acne compared to those with healthy skin[28].

PROBIOTICS

In recent years, much research has focused on the microbiome and its relationship to diseases. Considering the role of *P. acnes* in the pathogenesis of acne, attention is focused on the possible changes in the skin microbiome and disturbances in the intestinal microbiota in patients with acne[4]. A pilot study conducted in a group of adult acne patients receiving *Lactobacillus rhamnosus* GG preparation for 12 weeks showed its positive effects on reduced counts of acne lesions and reduced IGF-1, as well as increased FoxO1 in skin biopsies compared to the control group receiving placebo[29].

CONCLUSIONS

Acne vulgaris is one of the most commonly diagnosed skin diseases, which poses a major medical and psychosocial

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problem. It seems today that the Mediterranean diet, which is rich in vegetables and fruits, whole grains, vegetable oils, fish and seafood, is an optimal dietary model for individuals with acne. Also, patients should be informed that a balanced diet can have a beneficial effect on both their skin condition and overall health.

Conflict of interest

The authors do not declare any financial or personal links to other persons or organisations that could adversely affect the content of this publication or claim rights thereto.

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