Low-Energy Diet in the Treatment of Psoriasis

Subjects: Health Care Sciences & Services

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Psoriasis is a chronic inflammatory skin disease. Immunological, genetic, and environmental factors, including diet, play a part in the pathogenesis of psoriasis. Metabolic syndrome or its components are frequent co-morbidities in persons with psoriasis. A change of eating habits can improve the quality of life of patients by relieving skin lesions and by reducing the risk of other diseases. A low-energy diet is recommended for patients with excess body weight. Persons suffering from psoriasis should limit the intake of saturated fatty acids and replace them with polyunsaturated fatty acids from the omega-3 family, which have an anti-inflammatory effect. In diet therapy for persons with psoriasis, the introduction of antioxidants such as vitamin A, vitamin C, vitamin E, carotenoids, flavonoids, and selenium is extremely important. Vitamin D supplementation is also recommended.

psoriasis skin nutrition diet fatty acids obesity

1. Introduction

Psoriasis is one of the most common inflammatory skin diseases [1]. As estimated by the WHO (World Health Organization), this dermatosis affects 0.09–11.43% of the global population, and the number of patients varies from 1.50% to 5.00% in developed countries [2] It consists of abnormal hyperplasia of keratinocytes (epidermal cells), which leads to the formation of psoriatic plaques [3].

It is a chronic disease in which we can observe periods of spontaneous regression followed by relapses [1]. The disease affects the skin but is also a systemic disease [3].

Immune disorders, which lead to increased pro-inflammatory cytokine production, contribute to the pathogenesis of psoriasis. An increase in the activity of Th1, Th17, and Th22 lymphocytes leads to the production of pro-inflammatory factors in excessive amounts. These factors include: C-reactive protein (CRP), interleukins 1, 2, 6, 8, 12, 17, 22, 23 (IL), interferon γ (IFN-γ), tumour necrosis factor (TNF-α), ceruloplasmin, α2-macroglobulin, α1-antitrypsin, and others. Concentration of these factors is increased both in the acute phase of psoriasis and in remission [1][3][4]. TNF-α plays a key role in the pathogenesis of psoriasis due to its stimulating effect on the proliferation of keratinocytes [5].

Apart from immune disorders, genetic and environmental factors also play a part in the pathogenesis of the disease [1]. Among other things, a relationship between the occurrence of psoriasis and genes in the HLA complex (in particular HLA-Cw6) has been demonstrated. However, it is often the case that the disease never develops in people carrying psoriasis-related genes [1][6].

The environmental factors that can lead to the manifestation of psoriasis or escalation of lesions are as follows [4][6]:

- physical factors (X-rays, subcutaneous and intradermal injections, surgical procedures, vaccinations, tattoos, insect bites, abrasions, burns (including sunburns), acupuncture, UV irradiation);
- chemical factors (chemical burns, topical treatments, others);
- skin diseases (rosacea, fungal infections, allergic contact dermatitis);
- infections (mainly streptococcal pharyngitis, viral infections);
- stress;
- medications (β-adrenolytics, angiotensin-converting enzyme inhibitors, lithium, terbinafine, nonsteroidal anti-inflammatory drugs, anti-malarial drugs, tetracyclines, rapid withdrawal of systemic corticosteroids);
- diet;
- tobacco smoking;
- alcohol consumption.

Despite numerous studies, the etiopathogenesis of psoriasis has not been fully explained [1][7]. It is complex and ambiguous. The above-mentioned factors (immunological, genetic, and environmental) influence the development and severity of this dermatosis to varying degrees. Moreover, it is worth noticing the connection between psoriasis and other diseases [8][9].

Psoriasis is a systemic disease often accompanied by other diseases, e.g., metabolic syndrome and cardiovascular diseases [5][8][10][11]. It is estimated that persons suffering from psoriasis live five years less on average compared to healthy people. The most common causes of death in patients with psoriasis include thromboembolic events and myocardial infarction [10][11].

The chronic inflammatory process is the element that links psoriasis with its co-morbidities [5][7][8][11].

2. Metabolic Syndrome

Metabolic syndrome and its components, which include abdominal obesity, atherogenic dyslipidaemia, insulin resistance, impaired glucose tolerance or type 2 diabetes, and hypertension, are observed more frequently in persons with psoriasis than in the general population [8][12][13]. Some people also count the following among the

symptoms: hyperhomocysteinaemia, increased concentration of procoagulant factors, microalbuminuria, and non-alcoholic fatty liver disease [1][14][15].

In a population-based cross-sectional study by Langan et al. [16], 34% of persons with psoriasis and 26% of the control group had metabolic syndrome. A positive relationship between the occurrence of metabolic syndrome and the severity of psoriasis (determined by the BSA—Body Surface Area indicator) was also observed. In the studied group, metabolic syndrome was diagnosed in: 32% of patients with mild psoriasis, 36% of those with moderate psoriasis, and as many as 40% of those with a serious form of dermatosis [16]. Thus, the metabolic syndrome is more likely to affect patients with moderate to severe psoriasis, especially patients who developed the disease at a young age [1][16].

Pro-inflammatory cytokines as well as Th1 and Th17 lymphocytes play an important role in psoriasis. Levels of cytokines such as IL-6, TNF-α, angiogenic factors, and adhesion molecules are elevated in obesity psoriasis and ischaemic heart disease. In addition, these inflammatory mediators have been shown to influence fat deposition, insulin action, and lipid metabolism. Thus, chronic inflammation in psoriasis may predispose to diabetes, atherosclerosis, and obesity. On the other hand, inflammatory mediators, whose production accompanies metabolic disorders, may initiate the manifestation of psoriatic lesions or exacerbate existing psoriatic symptoms [10][17].

In patients with psoriasis, TNF-α is found in blood serum and skin lesions, while it is absent in healthy skin. TNF-α is also secreted in adipocytes and has a role in the development of insulin resistance. In addition, the presence of TNF-α leads to an increase in the concentration of free fatty acids and triglycerides in the blood, which may cause atherogenic dyslipidaemia [10][18].

IL-6, found in high concentrations in psoriatic lesions, also plays an important role in metabolic disorders. Its production is three times higher in visceral adipose tissue than in subcutaneous adipose tissue and correlates with the possibility of developing type 2 diabetes. Moreover, elevated IL-6 levels are also found in patients with unstable coronary artery disease [10][18].

3. Obesity

Persons suffering from psoriasis are more often overweight or obese compared to the general population [1][19][20].

In the meta-analysis of 18 studies carried out by Armstrong et al. [19], which covered over 200,000 persons suffering from psoriasis, it was calculated that the risk of obesity is over 50% higher in patients with psoriasis compared to those without the disease. In patients with more serious forms of psoriasis, the risk of obesity is higher compared to mild forms of the disease. In addition, patients with normal body weight and psoriasis have a higher risk of becoming obese in the future [19].

Obesity (especially android obesity) promotes the occurrence of psoriasis and worsens its course. On the other hand, psoriasis increases the risk of obesity. Moreover, the more severe the lesions, the higher the risk of obesity is; as BMI increases, the risk of psoriatic arthritis increases [1][6][10][11][14][20]. It has been observed that a body mass index (BMI) > 29 kg/m^2 is associated with a more than two-fold-increased risk of psoriasis. Furthermore, the severity of psoriatic symptoms is correlated with an increase in BMI [1][20].

BMI is not an ideal indicator for assessing a patient's nutritional status, as it does not take into account body composition and body fat distribution. Such parameters can be assessed, for example, by using the bioelectrical impedance (BIA) or dual energy X-ray absorptiometry (DXA). In a study by Galluzzo et al. [21], body composition analysis by bioelectrical impedance analysis (BIA) was performed in a group of 164 patients with psoriasis. In that study, 22.50% of men and 5.50% of women with a BMI indicating normal weight and 50% of men and 50% of women with a BMI suggesting overweight were obese according to body fat percentage. This indicates the much greater diagnostic value of the BIA method compared to BMI alone.

Similar results were obtained by Diniz et al. [22]; however, in this study in 42 patients with psoriasis and 41 controls, body weight was measured by DXA. In both study groups, DEXA showed a higher prevalence of obesity compared to BMI and waist circumference.

Blake et al. [23] performed a systematic review of 25 research papers on the relationship between psoriasis and body composition, measured by various methods (BIA, DXA, CT—computed tomography, and others). Their conclusion was that the presence of psoriasis is associated with higher levels of body fat, visceral fat, and reduced muscle mass.

When assessing the nutritional status of patients with psoriasis, it is also worth considering the phase angle measured by BIA. The phase angle allows the assessment of cell size and cell membrane integrity as well as lean body mass and tissue hydration. A decrease in phase angle values suggests cell membrane breakdown and a decrease in intracellular water, while a larger phase angle reflects greater amounts of intact cell membranes and lean body mass. Phase angle may be a predictive marker of mortality in many chronic diseases. Decreased phase angle may be associated with metabolic syndrome and its components [24]. The study by Barrea et al. [24] showed that the phase angle in patients was lower compared to the control group. Phase angle values were correlated with psoriasis patients' quality of life, disease severity, and the presence of metabolic syndrome.

Studies have shown that when obesity and the HLA-Cw6 gene coexist, the risk of developing psoriasis increases 35-fold compared with the risk in individuals free of these factors [1].

Adipose tissue is the largest endocrine organ, where many proinflammatory cytokines (e.g., IL-6, TNF-α) and bioactive factors called adipokines are produced. These are not only related to metabolic disturbances but may also be responsible for the severity of the psoriatic process [14][15][17][25].

In patients with a severe course of the disease, increased blood levels of pro-inflammatory adipokines (e.g., leptin, visfatin, chemerin) are observed, and during remission their levels decrease. In contrast, anti-inflammatory adipokines (omentin and adiponectin) inhibit the development of psoriatic lesions. Serum levels of these anti-inflammatory factors are significantly lower in patients with severe disease compared to patients with mild forms of psoriasis [1][15].

The higher prevalence of obesity in psoriasis patients may be related not only to the overproduction of pro-inflammatory factors, but also to the fact that psoriasis is often accompanied by stigma and prolonged stress, which often lead to reduced levels of physical activity as well as adverse changes in eating habits and broader lifestyle. These, in turn, may contribute to weight gain [26][27].

On the other hand, obesity may contribute to lower self-esteem, increased stress levels, and even the development or worsening of depression and anxiety disorders [26]. The relationship between psoriasis and obesity in a psychological context is shown in **Figure 1**.

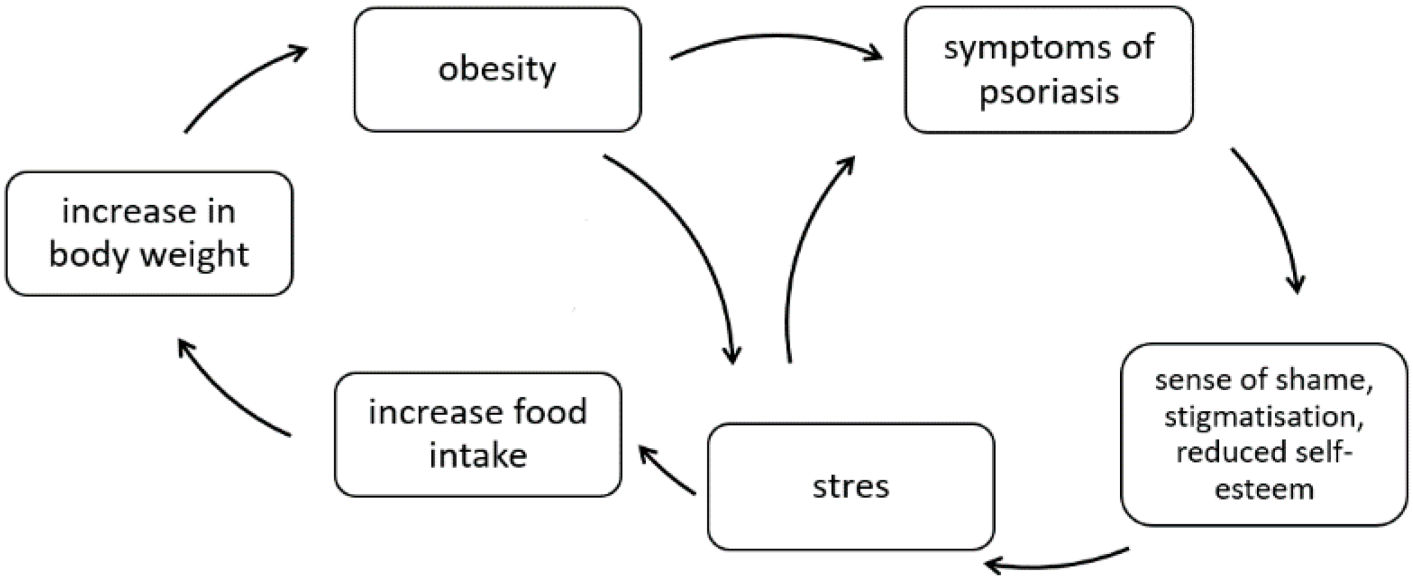

Figure 1. The correlation between psoriasis and obesity in a psychological context (own elaboration based on [26]).

Moreover, psoriasis, together with comorbidities (including obesity), leads to a chronic inflammatory process, changes in glucose metabolism, and subsequent development of atherosclerosis and cardiovascular disease. The association of these changes is illustrated by the concept of the so-called “psoriatic march” (**Figure 2**) [1][17].

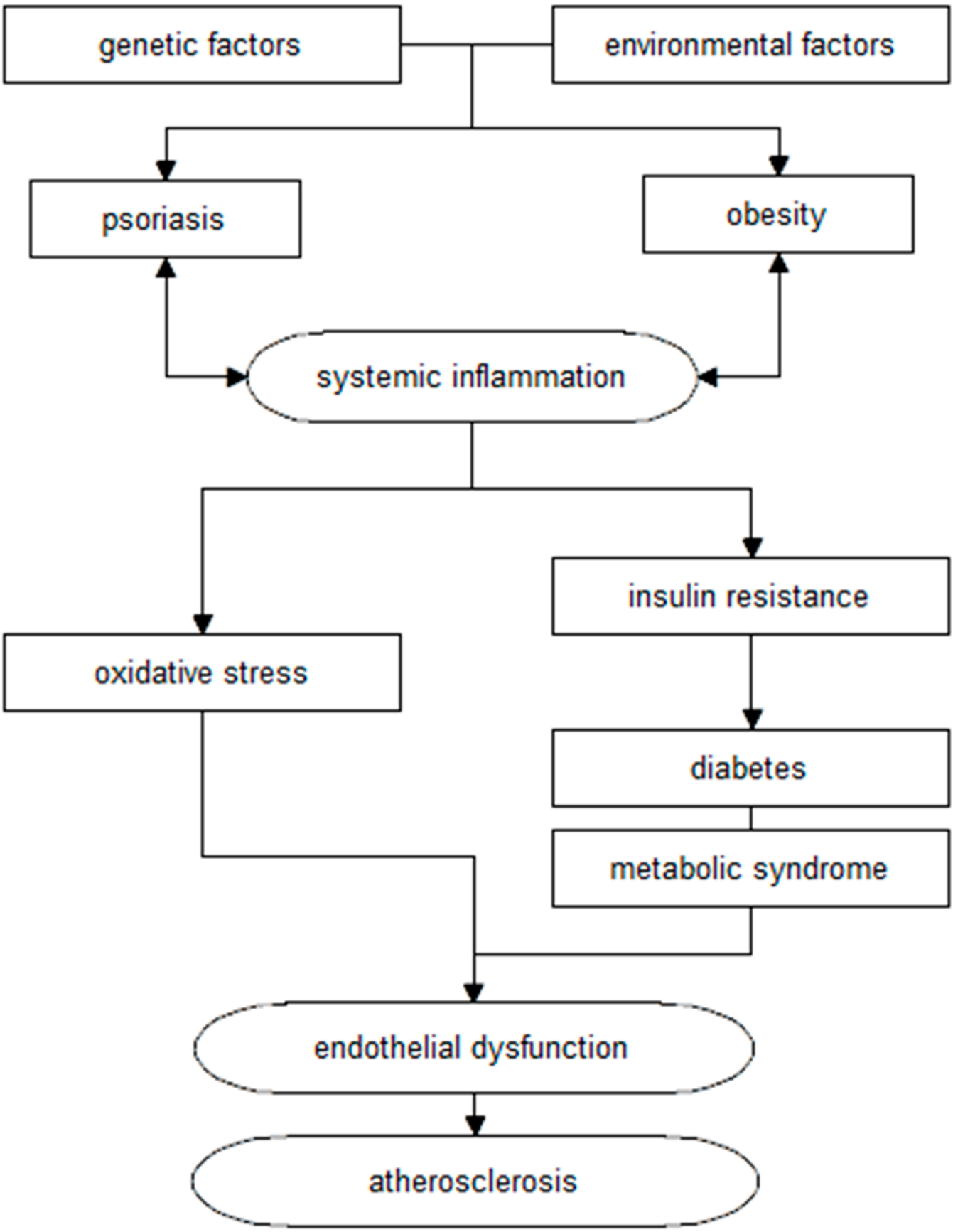

Figure 2. Diagram of the relationship between psoriasis and cardiovascular disease, according to the "psoriasis march" concept (own elaboration based on [1][17]).

To date, no single specific reason for the association between the prevalence of metabolic disorders and psoriasis has been established. However, this relationship has been confirmed by many studies, so every patient with psoriasis should be diagnosed for these conditions, as early implementation of appropriate treatment can prevent the development of these diseases; in addition, dermatologists can play an important role in the early diagnosis and evaluation of metabolic disorders [5][15][28].

Thus, in patients with psoriasis the following is recommended [1][13][15]:

- body weight assessment;
- BMI assessment;
- assessment of waist/hip ratio (WHR);
- fasting blood glucose determination at least once a year;
- more frequent testing for hypertension;
- determination of serum lipids;
- determination of serum uric acid and liver enzymes;
- in patients with other cardiovascular risk factors (e.g., hypertension, obesity), performing an Oral Glucose Tolerance Test (OGTT).

Genetic factors are responsible for the occurrence of psoriasis, yet the manifestation of lesions is determined by environmental factors such as infections, stress, and diet. Therefore, a change of eating habits can significantly improve the quality of life of patients, both through a beneficial effect on psoriatic lesions and through reducing the risk of other diseases, e.g., cardiovascular events [14][29].

A severe course of psoriasis can be associated with nutritional deficiencies caused by faster loss of nutrients, resulting in exfoliation of the affected epidermis [29].

The diet and nutritional status of the patient affect the severity of psoriasis, its course, and the body's response to pharmacological therapy [11][30].

4. Low-Energy Diet in the Treatment of Psoriasis

Obesity maintains systemic inflammation in the body, which can contribute to the intensification of psoriasis symptoms [3]. It is not known, however, whether obesity is a consequence of psoriasis or a risk factor for

developing this dermatosis. It is suggested that this relationship is two-way. Obesity is a predisposing factor for psoriasis and intensification of its symptoms, and psoriasis promotes the development of obesity [3][29].

It was observed that a BMI (Body Mass Index) > 29 kg/m^2 more than doubles the risk of developing this disease, and a reduction in body mass contributes to a reduction of blood serum inflammatory factors, significantly improves the course of the disease, and causes faster regression of psoriatic lesions compared to persons not following the diet [1][14][30].

A randomised study by Jensen et al. [31] proved that a low-energy diet (of 800–1000 kcal/day) followed for a period of up to 8 weeks contributes both to body weight loss (15 kg on average) and to reducing lesions and even improving the Dermatology Life Quality Index (DLQI) [31]. Furthermore, in the following publication, Jansen et al. [32] presented the results of continuing the programme for the next 48 weeks after cessation of the low-energy diet. Although the patients' body weight increased by 4.90 kg on average compared to the results obtained immediately after implementing a low energy diet, the PASI (Psoriasis Area and Severity Index) was further reduced [32].

An increased risk of side effects is observed in systemically treated obese patients, and body mass reduction leads to a decrease in toxicity of medications and an increase in their effectiveness [29]. A study by Gisondi et al. [33] showed that in obese patients, a 5–10 percent body mass reduction improves the therapeutic response to treatment with cyclosporine A (at a dose of 2.50 mg/kg b.w./day) [33]. Therefore, persons who follow dietary recommendations can reduce the dose of medication, and, consequently, reduce the side effects, including nephrotoxicity [11][30][33]. In turn, in the case of patients after successful therapy with methotrexate, a low-energy diet contributes to prolonged remission of psoriatic symptoms [14]. In persons using biologic medicines and a low-energy diet at the same time, greater relief of psoriatic symptoms was observed (greater improvement in PASI and BSA) compared to the group that underwent biological therapy only, without diet modifications [30].

Thus, in patients with psoriasis, a low-energy diet combined with regular physical activity and possible psychological support focused on patient motivation can complement their therapy [3][4][30][34].

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