FEATURES OF THE CLINICAL COURSE OF PSORIASIS IN PATIENTS WITH OBESITY

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Psoriasis is the most common chronic, genetically determined autoimmune polyetiological inflammatory disease with impaired epidermal proliferation, provoked by exogenous and endogenous factors, and manifested by erythematous and scaly elements, papules and plaques. According to the results of clinical and epidemiological studies, psoriasis affects about 3-4% of the population of our planet, regardless of gender, age, and ethnic group, and the specific gravity of this pathology in the general structure of skin diseases reaches, from the data of different authors, from 1% to 40%. However, despite the significant incidence of psoriasis and a large amount of research on this problem, there is still no single view of the pathogenesis of this dermatosis. For an objective understanding of the pathogenesis of psoriasis, it is necessary to take into account the insufficiently studied comorbidity of this pathology. Therefore, in the studies of the pathogenesis of psoriasis in recent years, more attention is paid to the impairment of metabolic processes. Recently, an indisputable link between psoriasis and obesity has been proven. Obesity has been found to increase the risk of many diseases, including psoriasis. The literature has broadly highlighted the question of the pathogenetic mechanisms of inflammatory processes in psoriasis and obesity that form a vicious circle at the level of the immune system, which must be broken for the successful treatment of these diseases. In this research, we studied the clinical presentation of the disease, measured anthropometric parameters, determined the grade of obesity by BMI, analyzed the history of life and disease, and conducted clinical and biochemical blood tests. The results of the study revealed that alimentary obesity in patients with psoriasis leads to metabolic disorders, complicating the course of dermatosis, which leads to a worsening of the DLQI of patients, the inefficiency of standard methods of therapy and frequent exacerbations of psoriatic disease. Therefore, the prospect for further research is a more in-depth study of the comorbidity of psoriatic disease, which will identify new targets for the treatment of this dermatosis to prevent complications and more effective treatment of this pathology.

Key words: psoriasis, alimentary obesity, pathogenesis, clinical presentation.

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Psoriasis is one of the most common chronic recurrent multifactorial diseases of the skin with a predominance of genetic bias, characterized by hyperproliferation of epidermal cells, impaired keratinization due to inflammatory reaction in the dermis, as well as the lesions of nails and joints [1]. According to the results of clinical and epidemiological studies, about 3-4% of the population of our planet, regardless of gender, age and ethnic group, suffers from psoriasis, and the share of this pathology in the general structure of skin diseases, according to various authors, ranges from 1% to 40% [2]. However, despite the considerable spread of psoriasis and a large amount of research on this problem, there is still no single view of the pathogenesis of this dermatosis. In order to objectively understand the pathogenesis of psoriasis, it is necessary to take into account the understudied comorbidity of this disease [3].

Recently, an indisputable link between psoriasis and obesity has been proven. Alimentary obesity develops as a result of metabolic and eating behavior disorders. It is characterized by the accumulation of adipose tissue in the body. Obesity can be both an independent multifactorial disease – primary obesity (alimentary-constitutional), and a syndrome that accompanies the course of other diseases – secondary obesity (symptomatic). In the structure of morbidity, primary obesity occurs in 95% of patients, secondary – only in 5% [4]. The main etiological reason for the development of primary obesity is the alimentary factor. The inhabitants of developed countries are currently the hostages of readily available food. In this context, the food moves from the category of necessity to the category of pleasure. Thus, eating disorders are gradually formed, which anticipates the development of obesity. Most often, obesity occurs as a result of consuming foods of high energy value (caloric content) in combination with insufficient physical activity and a factor of genetic susceptibility. However, in isolated cases, the occurrence of the disease against the background of genetic endocrine disruptions or malignancies was observed [5]. A person is considered obese if the body mass index (BMI) exceeds 30 kg / m². Obesity has been proven. Alimentary obesity develops as a result of inadequate nutrition (caloric density), triglycerides higher than 1.7 mmol / I, VLDL cholesterol less than 1.0 mmol / I. The parameters were evaluated according to the criteria of the US National Cholesterol Education Program [13, 14].

The statistical processing of the results was performed using Statistica 7.0. The difference was considered reliable with a probability of error p <0.05.

**Results and discussion**

In all patients, the clinical presentation of the disease was studied, anthropometric parameters were measured, the grade of obesity by BMI was determined, the history of life and disease was analyzed, and clinical and biochemical blood tests were conducted.

Based on the clinical presentation of psoriatic lesions, the PASI index was calculated, reflecting the area, color intensity, infiltration, and desquamation of psoriatic rash. Analyzing the results, it was found that the average group PASI index in patients with psoriasis and concomitant grade I-II alimentary obesity was - (21.8 ± 1.4), and in patients with psoriasis without obesity - (15.2 ± 1.2). The obtained results correspond to the average severity of psoriasis in both groups of patients. However, it should be noted that the average group PASI index was 30.3% higher in the group of patients with concomitant grade I-II alimentary obesity. The difference in PASI indexes depended on the severity of infiltration and hyperemia, while the area and intensity of psoriatic rash were approximately the same. Thus, in patients with psoriasis and concomitant grade I-II alimentary obesity, psoriatic rashes had a stagnant-red color with pronounced infiltration, whereas in patients without obesity, rashes had a pink color with less pronounced infiltration phenomena.

All patients complained of sleep disturbance, unrest, and anxiety, which resulted in poor quality of life. To study the impact of the disease on the patient’s quality of life, we calculated the dermatological quality of life index (DLQI). Analyzing the results of the study, it was found that in patients with psoriasis and concomitant grade I-II alimentary obesity, the DLQI index was 20.3 ± 0.5 points.
whereas in patients without obesity it was 14.5 ± 0.4 points, which indicates the pronounced impact of the disease on the quality of life for patients in both groups. It should be noted that the average group DLQI index is by 28.6% higher in the group of patients with concomitant grade I-II alimentary obesity.

From the history of the disease, it is known that all patients repeatedly received traditional outpatient and inpatient treatment for psoriasis. Patients with psoriasis and concomitant grade I-II alimentary obesity noted dissatisfaction with the treatment due to the duration of therapy and short periods of remission up to 3 months with episodes of exacerbation 3-4 times a year, and some of them did not notice complete recovery. Some of them indicated the presence of permanent (the so-called “regular”) plaques, while patients with psoriasis without obesity were satisfied with the treatment, most noted the seasonality of the disease with recurrence of psoriasis 1-2, sometimes 3 times a year.

Thus, a significantly higher number of exacerbations was observed in patients with psoriasis and concomitant grade I-II alimentary obesity, which indicates a more severe course of psoriasis in combination with this pathology. It should be noted that patients with psoriasis and concomitant grade I-II alimentary obesity, and psoriasis patients with normal body weight received only general anti-psoriatic therapy, without taking into account the comorbid conditions, which could affect the insufficient effectiveness of treatment.

In the examination of patients who made up the study group and BMI calculation according to the classification of obesity, it was found that 29 (36.25%) patients had grade I obesity (average group BMI - 34.04 kg/m²), grade II obesity - 51 (63.75%) patients (average group BMI - 38.15 kg/m²). The average BMI in the control group of patients with normal body weight was 24.5 kg/m², which is by 35.8% lower than in the group of patients with psoriasis and concomitant grade I-II alimentary obesity (Table 1).

Table N 1. The data of general blood analysis in the examined patients (M±m)

| Indicator | Value (number of patients) |
|-----------|-----------------------------|
| PASI (points) | Study group (n=80) | Control group (n=20) |
| DLQI (points) | 21.8±1.4 | 15.2±1.2 |
| BMI (kg/m²)  | 24.5 (20) |
| Normal weight | 0 |
| grade I obesity | 34.04 (29) |
| grade II obesity | 38.15 (51) |

Number of psoriasis relapses:
- Once a year: 0 - 6
- 2 times a year: 0 - 12
- 3 times a year: 13 - 2
- 4 times a year: 67 - 0

In order to evaluate metabolic disorders in patients with psoriasis and concomitant grade I-II alimentary obesity, we conducted studies of general and biochemical blood tests (Tables 2, 3).

Table N 2. The data of general blood analysis in the examined patients (M±m)

| Indicator (reference value) | Patients with psoriasis with grade I-II obesity (n=80) | Patients with psoriasis without obesity (n=20) |
|-----------------------------|---------------------------------------------|---------------------------------------------|
| Hemoglobin, (g/l)           | 128.5±5.8                                   | 147.3±6.5                                   |
| Men – 130-160               | Women – 120-140                             |
| Erythrocytes, (10⁹/l)       | 4.2±0.4                                     | 4.6±0.5                                     |
| Men – 4.5-5.0               | Women – 4.0-4.5                             |
| Color index 0.85-1.1        | 0.93±0.09                                   | 1.2±0.1                                     |
| Leukocytes, (10⁹/l) 4.0-9.0 | 7.2±1.3                                     | 5.2±1.2                                     |
| Stab neutrophils, (%) 1.0-6.0 | 1.53±0.21                                  | 2.4±0.35                                    |
| Segmented neutrophils, (%) 47.0-72.0 | 63.25±3.1                                  | 65.8±3.2                                    |
| Eosinophils, (%) 0.5-5.0    | 3.8±1.2                                     | 2.5±0.7                                     |
| Basophils, (%) 0-1.0        | 9.0±0.2                                     | 0.7±0.4                                     |
| Lymphocytes, (%) 19.0-37.0  | 26.73±2.62                                  | 20.1±1.9                                    |
| Monocytes, (%) 3.0-10.0     | 4.65±0.58                                   | 5.3±0.73                                    |
| ESR, (mm/h) 2-15            | 11.5±1.1 *                                  | 7.3±1.0                                     |

Note: * The probability of error of differences p<0.05
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Table 3. The data of biochemical analysis of blood in the examined patients (M±m)

| Indicator (reference value)          | Value | Patients with psoriasis with grade I-II obesity (n=80) | Patients with psoriasis without obesity (n=20) |
|--------------------------------------|-------|--------------------------------------------------------|------------------------------------------------|
| Total protein, g/l 64.0-83.0         | 61.7±2.4 | 68.3±2.8<br>60.3±3.4                                   | 60.0±2.5<br>61.7±2.8                                   |
| Albumin, g/l (38-54)                 | 45.3±2.6 | 46.5±2.5<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Globulin, g/l (20-30)                | 26.8±2.3 | 24.7±2.5<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Albumin-globulin ratio (1.2-2)       | 1.7±0.19 | 1.83±0.17<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Total bilirubin, µmol/l 8.0-21.0     | 18.6±1.7 | 14.7±1.5<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| ALT, units/l: men – 5.0-41.0 women – 5.0-31.0 | 38.9±2.9 | 36.5±2.3<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| AST, units/l: men – 5.0-41.0 women – 5.0-31.0 | 38.6±2.7 | 36.3±2.4<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Thymol test, IU (H-S) (0-4)          | 1.4±0.15 | 1.3±0.17<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Total cholesterol, µmol/l 3.0-6.2    | 7.5±0.3* | 5.2±0.2<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| HDL (1.04-1.55 µmol/l)               | 0.98±0.1 | 1.3±0.2<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| LDL (0-2.59 µmol/l)                  | 3.95±0.25* | 2.9±0.15<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Triglycerides, mmol/l (0-1.7)        | 2.4±0.05* | 1.5±0.08<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Glucose, 3-6 mmol/l                  | 6.05±0.7 | 5.5±0.5<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Urea, mmol/l 2.5-8.3                 | 5.04±0.23 | 5.2±0.21<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |
| Creatinine, mmol/l 50.0-120.0        | 87.95±6.7 | 89.2±6.3<br>61.7±2.8                                   | 60.0±2.5<br>61.7±2.8                                   |

Note: * The probability of error of differences p<0.05

Analyzing the data presented in Table 2, it can be noted that the level of all indicators is within the norm, both in patients with psoriasis and concomitant grade I-II alimentary obesity and in patients with psoriasis and normal body weight. However, it should be noted that in general blood analysis of patients with psoriasis and concomitant grade I-II alimentary obesity, we revealed moderate differences, characterized by a decrease in the amount of hemoglobin by 12.8%, the total number of erythrocytes by 8.7%, Cl by 5%, stab and segmented neutrophils by 36.3% and 3.9% respectively, monocytes by 12.3%, and leukocytes increase by 38.5%, eosinophilia by 52%, basophils by 28.6%, lymphocytes by 33% and an increase in ESR by 57.5% as compared to the rates of patients with psoriasis and normal weight.

When analyzing the data of biochemical blood analysis in patients with psoriasis with concomitant grade I-II alimentary obesity, significant differences were found in comparison with patients with psoriasis without obesity. Thus, in patients with psoriasis with concomitant grade I-II alimentary obesity, there was an increase in glucose by 10%, total cholesterol by 44.2%, triglycerides by 60% and LDL by 36%, as well as a decrease in HDL by 4.6%, total protein by 10.5% as compared to patients with normal body weight psoriasis, indicating impaired hepatic function by reducing the total protein and disrupted lipid metabolism with the effects of dyslipoproteinemia, characterized by an increase in the content of total cholesterol, LDL, triglycerides, decreased HDL levels. It should be noted that in patients with psoriasis with concomitant grade I-II alimentary obesity, there were more significant disruptions of the above indicators. Thus, the study found that patients with psoriasis with concomitant grade I-II alimentary obesity have a more severe course of dermatosis as compared to patients with psoriasis with normal body weight, which is well observed by the PASI and DLQI of patients and the incidence of psoriasis exacerbations.

Conclusions

Alimentary obesity in patients with psoriasis leads to metabolic disorders, complicating the course of dermatosis and worsening the patient’s DLQI, leading to inefficiency of standard methods of therapy and frequent exacerbations of psoriatic disease.

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