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GASTROENTEROLOGICAL PROBLEMS OF PSORIASIS

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Abstract. The presented article is devoted to the association of psoriasis with gastroenterological diseases. The authors report on the most significant comorbidities of gastrointestinal pathology in psoriasis. The article emphasizes the role of an interdisciplinary approach to managing patients.

Key words: *psoriasis; inflammatory bowel disease; celiac disease; Helicobacter pylori; liver pathology.*

ГАСТРОЭНТЕРОЛОГИЧЕСКИЕ ПРОБЛЕМЫ ПСОРИАЗА

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Резюме. Представленная статья посвящена ассоциации псориаза с гастроэнтерологическими заболеваниями. Авторы сообщают о наиболее значимых коморбидностях патологии желудочно-кишечного тракта при псориазе. В статье подчеркивается роль междисциплинарного подхода ведения пациентов.

Ключевые слова: *псориаз; воспалительные заболевания кишечника; целиакия; Helicobacter pylori; патология печени.*

Psoriasis is a systemic immune-associated disease of multifactorial nature with dominating genetic factors. It is characterized by accelerated proliferation of epidermocytes and impaired differentiation, immune reactions in the dermis and synovial membranes, imbalance between proinflammatory and anti-inflammatory cytokines, chemokines; frequent pathological changes in the musculoskeletal system [1].

Psoriasis is among the most common skin diseases, it occurs in 1–2% of the population in developed countries [1, 2].

In most cases, psoriasis manifestation occurs at a young socially active age, which adversely affects the quality of life of a patient. According to several studies, dermatosis has a moderate to severe course among 35–50% of patients. Genetic factors, immune system disorders and environmental factors are considered the most significant in the etiology of psoriasis [1–4]. It should be noted that patients with psoriasis have an increased risk of comorbidity, which is especially relevant in conditions of demographic aging of the population. Cardiovascular diseases are the most prevalent comorbidity, the second place is held by the pathology of digestive organs, followed by endocrine system problems, etc. [4, 5]. N. Al-Mutairi et al. analyzed the structure of comorbid pathology in 1835 patients with various forms of psoriasis and showed that the incidence of concomitant pathology increases significantly in a severe variant of the disease [6].

A number of authors note the role of metabolic processes, functional state of the stomach, in-

testines, pancreas and hepatobiliary system in the development of psoriasis [2, 3, 5]. Y.Y. Milyutin examined 136 patients with psoriasis and found that they had disorders of acid-forming and pepsin-forming function of the stomach [7]. A.V. Bogatyreva found that patients with severe and long-lasting psoriasis have significant disorders of gastric secretory function, expressed in decreased secretion of gastric juice and hydrochloric acid and increased secretion of mucoproteins [8]. Such changes may be associated with autoimmune gastritis [9].

PSORIASIS AND INFLAMMATORY INTESTINAL DISEASES

Inflammatory bowel disease (IBD) is one of the widely discussed gastroenterologic comorbidities of psoriasis.

Such IBDs as Crohn's disease (CD) and ulcerative colitis (UC) are frequently associated with psoriasis. These pathologies are more frequent (3–4 times) in children compared to adults. Genetic predisposition and peculiarities of the immune response are distinguished among the causes of IBDs in patients with psoriasis. It was established that the incidence of psoriasis in patients with Crohn's disease is significantly higher than in the population — 9.6% [10, 11].

A.B. Gottlieb et al. summarized the data of systematic literature reviews and metaanalysis of 79 studies: CD and nonspecific ulcerative colitis (NUC) were among the most frequent comorbid conditions in psoriasis [12].

Cohen et al. found that psoriasis was more commonly associated with Crohn's disease than with NUC [13]. Multivariate analysis of two nurses' health studies (Nurses' Health Study I and II) including follow-up periods of 1996–2008 and 1991–2005 showed that psoriasis in women increased the risk of developing Crohn's disease by 3.5 times, regardless of body mass index, age, physical activity, habitual intoxication, use of oral contraceptives and postmenopausal hormone therapy [14].

Another large-scale prospective observation of 5661 patients with psoriasis and psoriatic arthritis showed that their risk of DC is 6.8 times higher, especially in patients with early onset of the disease, as well as in the presence of an active skin process for 10 years [15]. However, the study did not demonstrate the correlation between the risk of CD development and the severity of psoriasis, as 87% of patients suffered from the mild form of psoriasis. The obtained data corresponded to international studies of genome screening, which revealed the interleukin-23 gene that is common for psoriasis and inflammatory bowel diseases.

Possible explanations for the identified association between psoriasis and IBDs include genetic abnormalities, immune dysfunction, systemic inflammation, and dysregulation of the gut microbiota. Several studies have examined the genetic link between psoriasis and GCD. The chromosomal locus 6p21, a region involving genes related to the major compatibility complex (MHC), is the most extensively studied genetic region. Psoriasis and IBDs share the same genetic susceptibility loci on chromosome 6p21, which corresponds to PSORS1 in psoriasis and IBD. It is known that genes that are not associated with the major compatibility complex, including *IL23R* and *IL12B*, have also been identified in the pathogenesis of both psoriasis and IBD. The *IL23R* gene encodes a subunit of the IL-23 receptor and affects IL-23 binding ability. Interleukin-23 is necessary for differentiation and activation of Th17 lymphocytes, which produce IL-17 [16, 17]. IL-17 binding to its receptor stimulates hyperproliferation and differentiation of keratinocytes, maturation of myeloid dendritic cells, and recruitment of neutrophils and macrophages in psoriatic lesions. Increased expression of IL-17 was detected in the intestinal mucosa and serum of patients with IBD compared to the control group. IL-17-producing T lymphocytes in the intestine appeared be involved not in disease formation, but in elimination of infection and protection of the immune barrier. The *IL12B* gene encodes the p40 subunit, which is involved in both IL-12 and IL-23 signaling pathways; therefore, IL-12B can be suggested as an important cytokine subunit in the pathogenesis of both psoriasis and IBD [16, 17].

In addition, the skin and intestine in IBD and psoriasis share similarities regarding their great microbial diversity and abundant blood supply. The microbiota influences the physiology and immune response of the skin and gut epithelium by regulating biological metabolites. Among other things, the microbiota can lead to the expression of antimicrobial particles, increased levels of cytokines and consequently regulation of T cell activity and differentiation. Thus, microbiota dysfunction may cause systemic immune dysregulation [18]. Current evidence supports the gut-skin axis theory, which describes a close relationship between gut dysbiosis and skin manifestations. It was found that patients with psoriasis have a reduced diversity and abundance of gut microbiota, which is also observed in patients with IBD [19].

A study by Scher et al. [19] indicates a significant reduction ($p < 0.05$) of bacterial diversity in the

gut of patients with psoriasis and psoriatic arthritis. Another study found a reduction in *Akkermansia muciniphila* in psoriasis patients; this species plays a protective role against inflammation and is involved in strengthening the intestinal barrier. Further studies showed that an increased ratio of *Firmicutes* to Bacteroidetes in patients with psoriasis correlates with the presence of greater inflammation [20]. On the contrary, the work of Codoner et al. [21] showed an enhanced bacterial diversity with increased representation of *Faecalibacterium, Akkermansia* and *Ruminococcus* and decreased *Bacteroides*. The latter genus produces polysaccharide A and activates regulatory T cells, so its reduction may be associated with an altered immune response. However, the increase in Faecali bacterium did not correspond to an increase of F. praunitzii, which was found in low concentrations in both psoriasis and Crohn's disease patients. This species produces butyrate, which can inhibit the NF-κB pathway and therefore block the inflammatory response.

Gut dysbiosis has been extensively studied in patients with IBD and, particularly, in patients with BC. Most studies reported a decrease in *Bacteroides* and *Firmicutes* bacteria belonging to *Clostridium* species (17 strains of groups IV and XIVa and butyricum) and an increase in *Gammaproteobacteria* and *Actinobacteria*. In addition, patients with CD demonstrated a relative increase in Proteobacteria, mainly *E. coli*, concentrated in the mucosal-associated microbiota compared to fecal samples [21, 22].

Taking into account the fact that both psoriasis and Crohn's disease are associated with persistent immune dysfunction caused by dysbacteriosis of the gut microbiota, which can negatively affect immunologic homeostasis, restoring normal microbiota composition is crucial for disease therapy, even when genetic, epigenetic, and environmental risk factors are involved.

PSORIASIS AND *HELICOBACTER PYLORI*

Helicobacter pylori infection plays an important role in a number of extraintestinal pathologies, including metabolic, autoimmune, hematologic, cardiovascular, neurologic and skin diseases [23, 24]. On average, this chronic infection affects about 4.4 billion people worldwide. *H. pylori* infection is usually asymptomatic, however, as a frequent cause of peptic ulcer disease, chronic gastritis and gastric cancer, it might include such symptoms as nausea, vomiting and abdominal pain. There are numerous testing methods available for the diagnosis of *H. pylori* infection. They are characterised by high specificity and sensitivity, namely the urea breath test (UDT), which is the most popular and accurate non-invasive method [25].

Several studies have documented that seropositivity to *H. pylori* is associated with elevated levels of C-reactive protein (CRP) and platelet activation factors [26].

According N.V. Pavlenk and E.N. Makhnovets, 76% of 50 patients suffering from the vulgar form of psoriasis were infected with *H. rulori*. As a result of the study, the following clinical features were revealed in the vulgar form of psoriasis with chronic helicobacteriosis: severe exudative component, more frequent deformation of nail plates with a «thimble» type [27]. The studies of S. Qayoom, Q.M. Ahmad showed 50 patients with psoriasis were infected with *H. pylori* significantly more often than the control group [28]. Some authors reported remission of psoriasis after *Hp* eradication therapy. [26]. Moreover, N. Onsun et al. demonstrated a correlative relationship between *Hp* infection and the severity of psoriasis course in the largest research ion this issue in 2014. Moreover, they emphasized that *Hp* eradication accelerates the resolution of psoriasis [29].

Campanati et al. demonstrated that *Hp*-positive patients with psoriasis had more severe clinical forms which rapidly regressed during eradication therapy [30]. Mesquita et al. demonstrated that the prevalence of anti-Hp antibodies was significantly higher in patients with psoriasis compared to healthy individuals [31].

A large meta-analysis conducted in Saudi Arabia in 2019 confirmed the correlation between *H. pylori* infection and psoriasis. The study reported a significant reduction in psoriasis area and severity index (PASI) in patients treated for *H. pylori* infection compared to the control group. The authors suggested that *H. pylori* treatment may reduce the severity of psoriasis and improve the effectiveness of treatment. The study revealed that psoriasis patients had a statistically significant attenuation of platelet P-selectin and CRP levels, CD4/CD8 ratio and percentage of lymphocytes after treatment of *H. pylori* infection compared to psoriasis patients without *H. pylori* eradication [32]. In addition, haptoglobin levels were substantially reduced in psoriasis patients treated for *H. pylori* infection compared to patients without therapy. The authors recommended the use of haptoglobin as a biomarker of psoriasis with a threshold value of 1.95 g/L and routine screening for *Hp*. The findings are consistent with the study of Onsul. Thus, the researchers concluded that *Hp* may be a trigger for psoriasis as well as a possible marker of its severity.

At the same time, according to E. Dauden et al. the most virulent strains of *H. pylori*, CagA+, have no significant influence on the course of psoriasis [33]. Conflicting literature data concerning the role of *H. pylori* in the development of psoriasis requires further scientific research and development.

PSORIASIS AND CELIAC DISEASE

Celiac disease, or gluten-sensitive enteropathy (GSEP), is a disease of the small intestine caused by an immune response to the plant protein gluten. The difficulties in diagnosing GSEP are related to the extreme variety of clinical manifestations — from extremely severe absorption disorders with chronic diarrhea to asymptomatic course or manifestation of extraintestinal symptoms, including psoriasis [34, 35]. Psoriasis and celiac disease are associated with dysregulation of Th1- and Th17-cell pathways, gamma-delta T cells and increased intestinal permeability, vitamin D deficiency, as its absorption is reduced by gluten enteropathy [36].

A recent retrospective cohort study conducted in California revealed that psoriatic patients have a 2.2-fold increased risk of being diagnosed with celiac disease compared to healthy individuals.

The meta-analysis showed that IgA AGAs were positive in about 14% of psoriatic patients compared to 5% of matched controls. Moreover, there was a correlation between celiac antibody positivity and severity of psoriatic manifestations [37].

At the same time, increased antibodies which are specific for celiac disease did not always lead to histologic markers of intestinal mucosa damage in psoriasis patients. However, adherence to a gluten-free diet resulted in rapid resolution of the clinical picture of the disease.

There is also an Italian multicenter study showing that 7 out of 8 patients with celiac disease and psoriasis who underwent a gluten-free diet had a significant improvement in psoriasis area and severity index (PASI), suggesting a role of gluten in the pathogenesis of both diseases [38].

PSORIASIS AND LIVER PATHOLOGY

In recent years, a significant number of publications indicate the relationship between psoriasis and pathology of the hepatobiliary system, particularly nonalcoholic fatty liver disease (NAFLD) and cholelithiasis. It corresponds to modern ideas of psoriasis pathogenesis. The development of NAFLD is based on excessive accumulation of triglycerides in the liver tissue. The disease begins with fatty hepatosis, and then progresses to steatohepatitis and, in case of further progression, to fibrosis and cirrhosis [39, 40].

NAFLD is considered to be one of the metabolic syndrome components and might be regarded as its hepatic manifestation [41].

The prevalence of NAFLD and metabolic syndrome among psoriasis patients is 10–25% which is higher than in the general population [40].

NAFLD includes a wide range of liver diseases with two main histological forms: simple hepatic steatosis (fat deposition without liver cell damage) and non-alcoholic steatohepatitis (NASH), which is characterized by liver inflammation, liver cell damage and fibrotic changes. A distinctive feature of NASH is excessive accumulation of fat in the liver associated with insulin resistance (IR). NASH is determined by the presence of fatty hepatosis (steatosis) in more than 5% of hepatocytes according to histological analysis or proton density of fat fraction (determined by the proportion of adipose tissue in the liver). Fat fraction which amounts to more than 5, 6% assessed by proton magnetic resonance spectroscopy (H-MRS) or quantification of the fat/water ratio by selective magnetic resonance imaging (MRI) is also specific for NASH [42].

The aberrant pathophysiologic processes underlying the progression of NASH are not fully understood, although they are assumed to include imbalances in fatty acid metabolism and cytokine production, increased inflammatory response as a result of oxidative or metabolic stress, leading to steatosis thereafter. In terms of clinical presentation, most cases of NAFLD are either asymptomatic or present with nonspecific symptoms such as fatigue and abdominal pain, and/or abnormal liver function test results [40, 41, 42].

The first data devoted to the association between psoriasis and NAFLD was published in 2001, it involved three patients with overweight/obesity and NASH, which was confirmed by liver biopsy [39, 40, 43]. Since then, various observational and controlled studies have revealed an increased prevalence of NASH in patients with psoriasis. In a large Dutch population-based study (2292 participants aged ≥55 years), the incidence of NAFLD was 46% in 118 patients with psoriasis and 33% in patients without psoriasis. Moreover, elderly patients with psoriasis were 70% more likely to develop NAFLD than those without psoriasis [44].

Patients with both NAFLD and psoriasis have an increased risk of developing more severe fibrotic changes in liver tissue compared to patients with NAFLD without psoriasis. In a case-con-

trol study conducted by P. Gisondi et al. NAFLD was diagnosed in approximately half of patients with psoriasis [45]. At the same time, the majority of patients had NAFLD combined with metabolic syndrome and higher levels of inflammatory markers (C-reactive protein).

At the same time, the majority of patients have a combination of NAFLD with metabolic syndrome and higher levels of inflammatory markers (C-reactive protein). It is important to note that the presence of NAFLD correlates with the prevalence and severity of psoriasis index (PASI). Similar data were obtained in a large cohort study by E.A. Van der Voort et al. Psoriasis was recognized as one of the independent risk factors for the development of NAFLD [46].

A study in Taiwan confirms a bidirectional association between NAFLD and psoriasis, especially in patients under 40 y.o. [47].

A recent single-center cross-sectional study conducted in Spain identified that 52% of patients among 71 patients diagnosed with psoriasis suffered from NAFLD. It is worth mentioning that 14% of patients had hepatic fibrosis diagnosed by transient fibro-controlled elastography [48].

Several foreign studies indicate that the presence and severity of psoriasis correspond to higher prevalence and greater severity of NAFLD, as well as NAFLD is a strong predictor of higher Psoriasis Area and Severity Index (PASI) [49, 50]. Using an attenuation control parameter to assess the extent of fatty liver disease and the severity of psoriasis on body surface area, Candia et al. confirmed a positive correlation between the two diseases [49]. In addition, progression to more severe forms of liver disease was higher in patients with psoriasis. Roberts et al. reported that 48 of 103 (47%) patients with psoriasis had NASH and 23 of 103 (22%) had biopsy-confirmed NASH, 35% of which had stage II-III fibrosis [50]. The prevalence of NASH was markedly higher than 12%. This amount was registered at the same medical center among patients with similar demographic characteristics but without psoriasis. In addition, concomitant NAFLD in patients with psoriasis may lead to a higher 10-year cardiovascular risk compared to patients without psoriasis [50].

One of the key factors in the development of NAFLD is obesity and metabolic syndrome. Obesity also predisposes to the development of psoriasis, and simultaneously, the presence of psoriasis increases the risk of obesity. The risk factor for developing obesity in patients with psoriasis is 1.18. In addition, obesity directly correlates with the severity of psoriasis manifestations [48, 50].

Chronic moderate inflammation is noted in both NAFLD and psoriasis. Adipose tissue produces a complex of biologically active substances that affect different metabolic parameters and the activity of inflammatory reactions. As the mass of adipose tissue increases, the production of adipokines and cytokines such as leptin, tumor necrosis factor alpha (TNF-alpha), interleukin (IL)-6, -17 and resistin is induced, which have proinflammatory properties and play a key role in the pathogenesis of metabolic syndrom. At the same time, synthesis of such anti-inflammatory substances as adipocytokine and adiponectin, is decreased in adipocytes. NAFLD and psoriasis are characterized by an imbalance of pro- and anti-inflammatory cytokines [51, 52]. The content of TNF-alpha, which regulates immune and inflammatory reactions, is determined by body mass index, percentage of adipose tissue, and hyperinsulinemia. Its level is elevated in patients with NAFLD. In addition to obesity, insulin resistance, hyperinsulinemia, dyslipidemia are considered as factors provoking NAFLD. TNF-alpha inhibits the phosphorylation of tyrosine and substrate 1 of the insulin receptor, which lead to a reduced biological response of tissues and impaired glucose transport into cells. The development of insulin resistance is one of the first steps towards NAFLD. On the contrary, reduction of body weight contributes to the reduction of TNF-alpha levels and insulin resistance [53].

The proinflammatory cytokines IL-6 and IL-17 are involved in immunoinflammatory reactions in both psoriasis and NAFLD. IL-17 is supposed to influence the progression of steatohepatosis to steatohepatitis in NAFLD. Consequently, IL-17 stimulates the production of IL-6 by keratinocytes in psoriasis. In turn, IL-6 induces Th migration into the skin and regulates proliferation and differentiation of dermal and epidermal cells [52, 53].

The degree of cutaneous manifestations of psoriasis positively correlates with serum levels of IL-6 and IL-17 [53, 54].

Adiponectin produced by adipose tissue belongs to the anti-inflammatory adipocytokines. It has an opposite effect on metabolism. Its content in the blood is inversely proportional to body mass index and is significantly reduced in patients with obesity, type 2 diabetes mellitus and NAFLD. Adiponectin promotes tissue sensitivity to insulin [54]. Its anti-inflammatory effect in both psoriasis and NAFLD is realized through suppression of Th1-immune response. This effect consists of inhibition of the synthesis of proinflammatory cytokines TNF-alpha, IL-6, decreased production of vascular endothelial adhe-

sion molecules, reactive oxygen species and the expression of anti-inflammatory cytokine IL-8 [53–55]. Adiponectin production is suppressed by high concentrations of proinflammatory agents, and its level is inversely proportional to the levels of TNF-alpha and IL-6 both in psoriasis and NAFLD [55]. It is noteworthy that patients suffering from psoriasis combined with NAFLD have lower serum adiponectin levels than psoriasis patients without liver lesions.

Thus, the relationship between psoriasis and NAFLD is due to disorders of carbohydrate and fat metabolism, as well as a complex of local and systemic immune disorders that support persistent inflammation.

The study of L.X. Tong et al. presents noteworthy data regarding the increased risk of psoriasis development in individuals with cholelithiasis [56]. The grounds are not completely clear. However, a recent study found that hypercholesterolemia is one of the risk factors for the development of psoriasis. Hence, it may be a common pathogenetic link between psoriasis and cholelithiasis. It was found that long-term (more than seven years) hypercholesterolemia significantly increases the probability of psoriasis development.

Summarizing all the above mentioned, it is possible to conclude that liver pathology is an increasingly common and clinically important comorbidity, occurring in up to 65% of patients with psoriasis. Liver impairment supports systemic inflammation, contributes to disease progression and often develops tolerance to therapy.

Conflict of interest. The author declares that there is no conflict of interest.

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