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Morpho-Functional State of Gastrointestinal Tract Cells in Patients with Rheumotoid Arthritis

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Annotation: This review is devoted to the characteristics of the cause of the risk of damage to the gastrointestinal tract in patients with rheumatoid arthritis. One of the urgent problems of modern medicine is the lesion of the gastrointestinal tract in rheumatoid arthritis. The issues of prevalence and risk factors of chronic diseases of the gastrointestinal tract are considered. The gastrointestinal tract is affected in rheumatoid arthritis more often than it is diagnosed. As a result, early diagnosis of lesions of the gastrointestinal tract in patients with rheumatoid arthritis has an important clinical and prognostic value.

Possible morphological changes and mechanisms of damage to the gastrointestinal tract are also considered. In rheumatoid arthritis, the occurrence of a chronic disease of the gastrointestinal tract depends, first of all, on the duration of the disease and the nature of the inflammatory process. These data are fully confirmed at the present time. The problem of lesions of the gastrointestinal tract in rheumatoid arthritis is poorly understood and requires further research.

Keywords: rheumatoid arthritis, dyspepsia, gastrointestinal tract, chronic gastritis.

Rheumatic diseases are the oldest human pathology, and are considered the most common ailments of the XXI century. In recent decades, there has been some progress in the field of theoretical and clinical rheumatology. According to E.A. Galushko and E.L. Nasonov rheumatic diseases include more than 80 diseases and syndromes [21]. Rheumatoid arthritis (RA) is an autoimmune disease characterized by the development of chronic destructive polyarthritis with frequent involvement of other systems in the pathological process.Extra-articular systemic lesions in RA can have a serious impact on the prognosis of the disease [6,30].

Pathology of the gastrointestinal tract (GIT) is detected in 13-62% of patients with RA [15] and occupies an important place among the extra-articular manifestations of this disease. Many works are devoted to the study of lesions of the gastroduodenal zone in RA [14]. Chronic gastritis in patients with rheumatoid arthritis develops primarily as a manifestation of systemic disorders characteristic of RA (lesion of the microvasculature, lymphoplasmacytic cell reactions), and the stages characteristic of gastritis of any origin occur, namely, superficial, chronic atrophic gastritis. On the other hand, damage to the mucous membrane of the upper gastrointestinal tract is aggravated by the use of non-steroidal anti-inflammatory drugs and glucocorticosteroids. The main morphological substrate of these ulcerations is an ulcerative defect of the mucous membrane and the chronic pangastritis associated with it.

Morphological manifestations of active gastritis were characterized by the fact that against the background of a pronounced inflammatory infiltration of the gastric mucosa by lymphocytes and plasmocytes, a large number of neutrophils appeared in the infiltrate and integumentary epithelium [37]. Most often, when taking drugs of this class, adverse reactions from the gastrointestinal tract

occur [37,13]. Adverse events (AEs) range from moderate symptoms such as dyspepsia, heartburn, and abdominal discomfort to more serious events such as peptic ulcer and associated life-threatening complications, bleeding, and perforated ulcer. The mucous membrane (CO) has certain protective mechanisms; when taking NSAIDs, the functioning of most of these mechanisms is disrupted [16].

Intestinal pathology in RA is considered in the literature in terms of side effects of basic therapy [1,7]. Functional and structural features with different disease activity remain less studied, their role in maintaining autoimmune systemic inflammation has not been established. The role of biogenic amines and peptide hormones produced by the diffuse endocrine system (DES) in the regulation of motility, absorption in the gastrointestinal tract, nociception, tissue trophism, and induction of the inflammatory process is widely discussed. Several works have been devoted to the study of the concentration of neuropeptides in RA in the synovial membrane and blood plasma [5,13], the quantitative density of the DES components of the intestinal mucosa in RA and the relationship with the activity of the autoimmune process have not been studied.

The role of intestinal pathology in RA remains unexplored. Changes in the intestine may be a consequence of the development and manifestation of immune inflammation and may be an inducer of a pathological process during which the body is sensitized to autoflora components. Intestinal microecology has a significant impact on homeostasis, being directly involved in the formation of the immune response. Data have been published that patients with RA have defective circulating T cells (Treg) [16], an increased titer of Th17 cells in plasma and synovial fluid is observed [8,18], the role and significance of which is being studied. It is possible that dysregulatory and dysbiotic disorders of the intestine can lead to impaired immune tolerance, being one of the triggers for a systemic response.

The prognosis is especially unfavorable in patients with RA with systemic manifestations: generalized vasculitis, rheumatoid nodules, lymphadenopathy, damage to the lungs, heart, liver, kidneys and other organs and systems. Among the extra-articular manifestations of RA, lesions of the gastrointestinal tract (GIT) are the least studied, although the most severe process is well known - intestinal amyloidosis, which occurs in 11% of patients and is usually combined with amyloidosis of other internal organs [22,24, 25,32].

Patients with RA have been noted to have impaired motility and secretory function of the stomach [26,28,34], the development of chronic atrophic gastritis [29], which is three times higher than its occurrence in the general population [12], as well as the frequent occurrence of mucosal ulcers [2,3 ,ten]. A number of researchers considered the nature of these changes in the context of the systemic nature of rheumatoid inflammation, believing that immune disorders are the basis of atrophic gastritis [35,4]. So, A.I. Strukov [36] emphasized that cell infiltration of the gastric mucosa fits into the concept of immune inflammation. D.Malone noted that the occurrence of ulcers correlates more with the nature of the course of RA than with anti-inflammatory drugs used by patients [11].

Nevertheless, the question of the specific gravity, on the one hand, of immune disorders in the stomach caused by the underlying disease, on the other hand, of the damaging effect of the mucous membrane of drugs that patients are forced to take constantly, is still debatable. In the literature of recent years, the main emphasis in the development of gastric disorders is on drug-induced gastropathy [23,27,33]. The pathogenesis of these gastropathy has not been fully deciphered and, probably, should not be considered outside of the processes that may be due to general immunopathological patterns inherent in RA as a systemic disease. Moreover, at present, a position is postulated that represents any chronic gastritis as an immune pathology that proceeds according to the standard scheme: superficial gastritis - atrophic gastritis [20].

In order to achieve remission and relapse-free course of RA, as well as to prevent the development of irreversible deformities of joint deformities, patients are forced to constantly take basic drugs (Zhukovsky G.N., 1990; Sigidin Ya.A., 1988; Borg G. et .al.; 1991 ; Contreras M. et. al., 1993). In combination with it, non-steroidal anti-inflammatory drugs (NSAIDs) and quite often glucocorticoids (GC) are prescribed to suppress inflammation in the joints, eliminate pain in them, morning stiffness, poor general health. Such therapy, started from the moment RA was diagnosed, can create preconditions for the correction of immunoregulatory disorders, affect the course of the disease, slow down its progression, and have a symptomatic effect (Sigidin Ya.A. et al., 1994).

However, the constant intake of NSAIDs, immunosuppressants and GC causes a high level of damage to the gastrointestinal tract (Muraviev V.S., 1995). It is well known from literature data that NSAIDs, by inhibiting the production of prostaglandins (PG), burns the resistance of the gastric mucosa to the aggressive effects of hydrochloric acid and pepsin, leads to the development of gastropathy, which, in some cases, can threaten the life of patients (Svintsitsky A.S. , 1994). Reduction of pain and inflammation in the joints in patients with RA in the treatment of NSAIDs is often achieved at the cost of significant side effects from the digestive tract. So, more often than in the general population, erosive and ulcerative lesions develop (Nasonova S.V. et al., 1994). The risk of NSAIDs - gastropathy increases with simultaneous therapy with GC (Prokaeva T.B. et al., 1994). The use of synthetic analogues of PG did not lead to complete elimination, although it reduced the incidence of erosive and ulcerative lesions of the stomach (Graham D. Et.al., 1990); Hegnier P., 1993; Verdicrt W. et.al., 1992. From the literature data, it is known that NSAIDs more often cause the occurrence of gastric ulcers (Roth S., 1986).

The role of intestinal pathology in RA remains unexplored. Changes in the intestine may be a consequence of the development and manifestation of immune inflammation and may be an inducer of the pathological process, during which the body is sensitized to the components of the autoflora. Intestinal microecology has a significant impact on homeostasis, being directly involved in the formation of the immune response. Data have been published that patients with RA have defective circulating T cells (Treg) [17], an increased titer of Th17 cells in plasma and synovial fluid [9,19] is observed, the role and significance of which is being studied. It is possible that dysregulatory and dysbiotic disorders of the intestine can lead to impaired immune tolerance ranciency, being one of the triggers of a systemic response.

It is currently difficult to say which is primary - RA or bowel changes. Obviously, there is a combination of violations of the structural and functional characteristics of the joints and intestines on against the background of a systemic imbalance of DES components. Against the background of an imbalance of hormones and neurotransmitters, inflammatory-dystrophic mucous membranes develop, which facilitate the penetration of antibodies.Microbial and viral antigens of the intestinal ecosystem, in turn, cause endogenous intoxication, initiate immune inflammation, and exacerbate the course of RA.

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