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Clinical and Morphological Manifestations and Risk Factors of Small Intestine Damage in Rheumatoid Arthritis

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Annotation: 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.

Keywords: rheumatoid arthritis,small intestine, lesion, clinical manifestations.

Rheumatoid arthritis (RA) remains a serious disease with an unfavorable prognosis, which is steadily progressing in a fairly large number of patients (Nasonova V.A., 2003). In the USA, over a ten-year period, more than half of RA patients become incapacitated. The mortality rate of RA patients is higher than in the general population. It is believed that men and women suffering from this disease die 4 and 10 years earlier, respectively, than their peers in the population. The prognosis is most unfavorable in patients with severe course of the disease and systemic manifestations, in particular vasculitis. The five-year survival rate of patients with severe joint mobility disorders does not exceed 50% (similar data were obtained in patients with advanced coronary atherosclerosis and lymphogranulomatosis).As the duration of the disease increases, the mortality of patients increases (Sherrer Y. et.al ., 2014). All of the above makes RA an important socio-medical problem.In order to achieve remission and relapse-free course of RA, as well as to prevent the development of irreversible joint deformities, patients are forced to constantly take basic drugs: kuprenil, crisanol, delagil, methotrexate, etc.. In combination with them, nonsteroidal 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 well-being. Such therapy, initiated from the moment of the diagnosis of RA, can create prerequisites for the correction of immunoregulatory disorders, affect the course of the disease, slow its progression, and have a symptomatic effect (Sigidin Ya.A. et al., 2014).The prevalence of small intestine lesions and its severity depends on the underlying disease, as well as on the aggressiveness of therapy. The undoubted and justified interest of researchers in comorbid conditions in rheumatic diseases has not faded in the last decade, however, the concept of multimorbidity has not yet been integrated either into clinical practice or into scientific research in the field of rheumatology. Thus, it is important to diagnose and treat them at an early stage. To determine the frequency of small intestine lesions, as well as clinical and morphological variants and risk factors for the development of secondary diseases in RA patients.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. 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 [8]Major studies conducted in recent years have demonstrated the association of RA with a high risk of chronic kidney disease (CKD) and cardiovascular complications, which is associated with increased mortality in this category of patients [9, 27,18]. The spectrum of renal pathology underlying CKD in RA is quite wide. Secondary amyloidosis for many years occupied the main position among the variants of nephropathy in patients with RAAccording to some studies, there is a tendency to change the structure of kidney damage in RA [5], taking into account the use of highly effective therapy regimens, including genetically engineered drugs, which serves as an additional prerequisite for studying this category of patientsIn addition, a higher incidence of RA was found among relatives of patients of the first degree of kinship than in the general population. These data are fully confirmed at the present time [3].The formation of nephropathies in RA is multifactorial, which is represented by a variety of their clinical and morphological variants with minor, nonspecific changes in urine tests. The course of rheumatoid nephropathy, as well as other chronic kidney diseases, is progressive with the development of nephrosclerosis and a decrease in the bridgehead of functioning nephrons, with an outcome in chronic renal failure, with an extremely unfavorable prognosis, which determines the importance of early diagnosis and treatment of nephropathies in RA.Renal pathology is detected in RA with a high frequency - about 60%, according to various authors [36].Patients with RA may have various renal diseases: secondary amyloidosis of the kidneys, glomerulonephritis, interstitial nephritis, renal vascular vasculitis, nephrosclerosis, and in some cases, their combinations [30,37]. Etiologically, kidney lesions in patients with RA can be divided into 2 groups: firstly, nephropathy as one of the extraarticular manifestations or complications of RA itself, for example, renal vascular vasculitis, chronic glomerulonephritis, secondary amyloidosis, and secondly, as a complication of drug therapy P A: analgesic nephropathy (AN), drug glomerulonephritis. Pathogenesis of such different kidney diseases can't be the same. A certain contribution to the progression of chronic kidney disease is made by disorders in the hemostasis system, endothelial dysfunction [41,43,24], the frequency of exacerbations of the disease, the presence of half-moons and the severity of tubulointerstitial changes in the nephrobioptate [21].In patients with rheumatoid renal vascular vasculitis, a slight transient decrease in renal function is more often detected along with transient hematuria, indicating local inflammation, and severe renal insufficiency is rarely observed [38,1].The spectrum of renal pathology underlying CKD in RA is quite wide. Secondary amyloidosis for many years occupied the main position among the variants of nephropathy in RA patients [45, 28]. According to some studies, there is a tendency to change the structure of kidney damage in RA [6].Many researchers have noted that in RA patients, the development of CKD and the severity of its manifestations are determined by the duration and activity of the underlying disease, age, the presence of arterial hypertension (AH), lipid metabolism disorders and hyperglycemia [46, 2, 17].The unfavorable prognostic significance of kidney damage in rheumatoid arthritis (RA) has been actively attracting the attention of researchers in recent years [10].Certain clinical variants of involvement of the kidneys in the pathological process in rheumatoid arthritis are noted in most patients [33].Various variants of kidney damage in rheumatoid arthritis are described, in particular, glomerulonephritis, amyloidosis, vasculitis, as well as iatrogenic forms (analgesic tubulopathy, membranous nephropathy, etc.) [35,29,32].Early manifestations of functional renal disorders, especially with their moderate severity, do not always attract the attention of clinicians, while the progression of chronic kidney disease (CKD) in RA can be rapid, especially in old age, as well as in association with cardiovascular pathology [11,14].According to some researchers, the development of CKD in RA may be associated with cardiovascular damage in большей степени, чем с активностью самого РА [16].Currently, the leading pathogenetic mechanism for the development of glomerular and tubuointerstitial changes in the kidneys is chronic inflammation. In particular, elevated levels of C-reactive blood protein (CRP) in patients with RA cause glomerular vascular endothelial dysfunction and trigger the synthesis of proinflammatory cytokines. [20,25]. Previously published studies have shown that in RA patients treated with cytokine inhibitors, kidney function remained stable for a long time [19]. According to other data, in RA and amyloidosis of the kidneys, therapy with tumor necrosis factor alpha inhibitors led to a simultaneous decrease in proteinuria [4,7].The study of the pathogenesis of glomerulonephritis continues, since existing therapies do not

have the desired effectiveness [4,]. The connection of glomerulonephritis with changes in the equilibrium of cytokine synthesis associated with the mechanisms of the immune response has been proven [13,22].It has been established that cytokines take part in the regulation of proliferative processes, differentiation, growth, and cell activity [2,6]. The quantitative content of cytokines and their ratio reflect the dynamics of the pathological process, correlate with the activity of the disease, which allows us to judge the effectiveness of the therapy and predict the outcome of the disease [2]. The creation of an experimental model of chronic autoimmune inflammation allows us to study its effect on the condition of the small intestine in rheumatoid arthritis. With this in mind, the need to continue morphological, experimental studies on this issue has not lost its relevance.

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