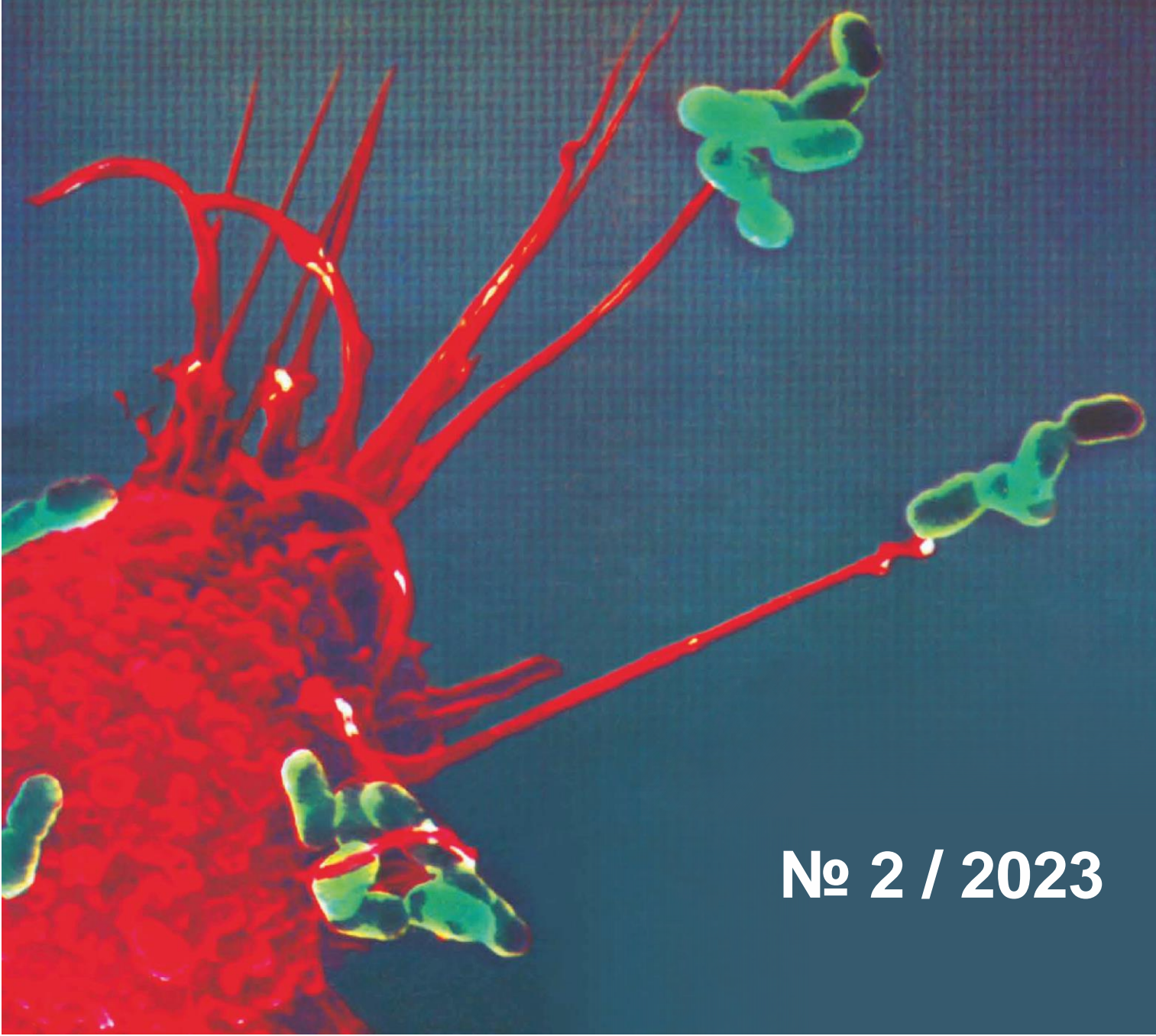


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арча меваси, наъматак меваси, дала қирқбўғими ўти, мойчечак гули, тукли эрва ўти - йиғма -3 да айниқса юқори бўлиб, тажриба натижаларига кўра йиғма -3 яллиғланишга қарши фаоллиги билан ЛИВ-52 га нисбатан юқори кўрсаткич кўрсатди.

SUMMARY

INFLUENCE OF A COMPOSITION OBTAINED FROM A MIXTURE OF DRY EXTRACTS OF LOCAL MEDICINAL PLANTS ON THE COURSE OF ASEPTIC ARTHRITIS

Khakimov Ziyoviddin Zainutdinovich¹, Rakhmanov Alisher Khudaiberdievich¹, Khadjieva Umida Abdulxaevna², Tursunova Laziza Ikramdjanovna²

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The article presents the results of the study of the antiexudative activity of the preparation based on the dry extracts of local medicinal plants in the model of aseptic arthritis induced by dextran in experimental animals. Mixture of dry extracts of medicinal plants in the following ratio (0.5:1.0:0.5:1.0:0.5) - juniper fruit, sedum fruit, field sedge herb, chamomile flower, hairy pea herb - in aggregate -3 is particularly high, and according to experimental results, compound-3 showed a superior index to LIV-52 with anti-inflammatory activity.

УДК: 616.085-72-002.77: 616-002.77

SPECIFIC CLINICAL FEATURES OF REACTIVE ARTHRITIS

(Literature review)

Xoshimov Abrorjon Anvarjonovich, Nurmatova Nargiza Fatxullayevna, Habibulloyeva Bibiroziya Rahmatullozoda

Tashkent Medical Academy

Key words reactive arthritis, joints syndrome, cytokines, IL 17, IL 18.

Purpose of the study. To study the cytokine profile, cellular and humoral immunity, as well as nonspecific protection factor in patients with reactive arthritis. Patients with reactive arthritis make up 1/3 of patients with rheumatological patients, and in the chronic form of the disease, a significant loss of the functional abilities of the joints and the occurrence of severe complications can develop, which leads to disability of patients. Interest in reactive arthritis is also associated with the fact that a number of patients, especially when the process is chronic, develop destructive processes in the joints up to ankylosing.

The inflammatory diseases of joints are one of the current problems in modern rheumatology. The main reason is it widespread among population and

lasts for years. Moreover, this disease sometimes turns to chronic and leads to early disability. It is known that the term "reactive arthritis" (ReA) was introduced into scientific and medical literature in the early 70s of the 20th century by scientists K. Aho and P. Ahvonen, and was used to identify arthritis developed after yersiniosis infection. Later, this term took a strong place in all modern classifications of rheumatological diseases, and the terminology of arthritis, which was previously used, was changed to "infectious-allergic arthritis" and "postinfectious arthritis".

Nowadays, there are no clear data on the epidemiology of ReA, and this is explained by the difficulty of comparing previously less obvious infections, as well as the similarity of clinical symptoms with other arthritis. In recent years, the rate of occurrence of ReA disease tends to increase, and this is caused by the frequent occurrence of urogenital infections, the observation of cases of disease among family members, and the increase of diseases among military officers.

Based on different literature, ReA is a disease belonged to the group of seronegative spondyloarthritis, characterized by joint inflammation due to previous intestinal or urogenital infections. Accordingly, there are postenterocolitic and urogenital forms. By the way, the postenterocolitic form is observed in 5.2 cases per 100,000 population, and the urogenital form in 4.5 cases. Urogenic reactive arthritis (UReA) takes the major place in the general composition of ReAs and is observed more often in men than in women (according to various authors, from 10:1 to 2-3:1). At the same time, UreA is observed more often in women compared to the literature review, so it is necessary to identify the focus of urinary tract infection in patients with seronegative arthritis and associate it with joint syndrome.

According to modern concepts, in the development of arthritis caused by intestinal or urogenital infections, the location of the primary infectious process, the characteristics of the microorganism that caused it, and a certain genetic predisposition of the macroorganism (*Chlamydia trachomatis*, *Yersinia enterocolitica*, *Yersinia pseudotuberculosis*, *Shigella flexneri* and *Campylobacter jejuni*), including factors such as HLA-B27 - histocompatibility antigen transportability. Organs of the nasopharynx, genitourinary system and gastrointestinal system are the "gateways" for negative factors that cause disease.

Undoubtedly, the role of intestinal and urinary tract infections as an initial factor in the development of ReA is confirmed by the stable chronological relationship of the development of arthritis after the infection. The processes observed in chlamydia have been studied in depth, but the identified features can also be observed in other infections that lead to the development of ReA. It was found that chlamydia are distinguished by two main forms, namely elementary bodies (infectious form) and reticular bodies (vegetative form). Chlamydia possesses all the cellular machinery to synthesize their own DNA, RNA, and proteins, but depend on the host cell for the supply of nucleotides, amino acids, vitamins, co-factors, nutrients, and energy compounds. The elementary bodies

phagocytosed by the host cells transform into reticular bodies, which proliferate and transform back into elementary bodies, breaking the host-cell integrity and exiting into the intercellular space. Elementary bodies have the characteristic of living outside the cell - this is an infectious particle, while reticular bodies break down in the extracellular environment, but have high metabolic activity inside the cell. Chlamydia transforms into an L-shaped form and persists for a long time, then transforms into elementary bodies and maintains resistance to antibacterial treatment.

An inflammatory process develops in the organs, joints and extra-articular tissues affected by the pathogen with the participation of biochemical and immunological reactions, these reactions are universal in relation to tissue damage, including microbial agents. Currently, specific aspects of the pathogenesis of ReA have been identified. According to the results of many studies, a number of microorganisms called ReA (*Yersinia*, *Salmonella*, *Shigella*, *Campilobacter*, *Chlamydia*) contain lipopolysaccharides that induce the synthesis of interleukin-1 cytokine and tumor necrosis factor by macrophages, which trigger the development of the inflammatory process. Lipopolysaccharides are detected in synovial cells. Furthermore, the importance of bacterial lipopolysaccharides in the pathogenesis of ReA is further supported by the strong IgA response against lipopolysaccharides in ReA patients with yersiniosis and salmonella. It is assumed that a persistent increase in IgA concentration reflects persistent antigenic stimulation.

The long course and recurrence of the disease is associated with the weakening of non-specific protection in these patients, which may be caused by long-term inadequate medical treatment (antibiotics, non-steroidal anti-inflammatory drugs, etc.). The severity of the disease, the transition of the process to a chronic type, the activation of the bacterial agent in the body, the presence of the HLA-B-27 antigen in the body, and genetic predisposition.

In recent years, the role of the gastrointestinal system in the development of specific positive pathological processes in reactive arthritis has been revealed. The mucous membrane of this system has the property of neutralizing bacteria, parasites, exogenous toxins, non-infectious antigens. Due to certain reasons, the imbalance in this system changes the stability of the body and causes a tendency to the development of the disease. Since the reactivity of the body is not the same in different periods of a person's life, the course of the disease can be different in patients of all ages. Despite the positive results in the treatment of the disease, the development of disability in patients remains high.

Based on the newest data, experts working in ReA diagnostics today, modern laboratory diagnostic methods include a wide spectrum of biomarkers: autoantibodies, indicators of the acute phase of inflammation, cytokines, endothelium-activating markers, lymphocyte subpopulations, and genetic markers in blood, synovial fluid, and synovial tissue. These markers make it possible to assess the general condition of the organism in relation to infection,

reflecting joint syndrome of one or another degree. However, they do not have the ability to accurately assess the complications of the disease or serve as a predictor of the destruction of ReA joint structures.

A significant breakthrough in the development of new approaches to the treatment of rheumatological diseases is the development of biological therapy methods. Cytokines of the IL-17 family play an important role in the pathogenesis of immunoinflammatory rheumatic diseases, so the use of an immunotherapeutic approach based on the inhibition of their activity is very relevant. Inhibition of immune responses dependent on IL-17A(F) is a promising direction in the treatment of a wide range of autoimmune diseases. The main requirements for cytokine inhibitor drugs under development include a combination of such factors as high efficiency and acceptable safety for the body. It should be noted that only classical antibodies specific to IL-17A(F) are present on the drug market. Other types of antibodies, bi(tri)specific antibodies and nanoantibodies blocking the interaction of IL-17A(F) with the receptor, have been developed relatively recently. Small molecule antagonists of IL-17A (peptides and macrocyclic compounds) may have more side effects compared to other inhibitors, and their use also requires further research.

While a very large number of fundamental and clinical studies have been devoted to the significance of IL-17 in the immunopathogenesis of rheumatological diseases and the possibilities of its inhibition for the treatment of these diseases, the study of the role of IL-18 as a mediator and biomarker of immune inflammation and a “target” for anticytokine “targeted” therapy is just beginning. The functional activity of IL-18 is regulated by IL-18-binding protein (IL-18SB), which, having a high affinity for IL-18, suppresses IL-18-dependent cellular effects. Normally, the concentration of IL-18SB is more than 20 times higher than IL-18. Imbalance between IL-18 and IL-18SB, developing which occurs with hyperproduction of IL-18, leads to IL-18- mediated "dysregulation" of the immune response and the progression of inflammation.

Taking this into account, at present, in the diagnosis of ReA , in the treatment of patients by specialists, in the early stages of the disease, the identification of signs of damage to the joint tissue (synovial shell, cartilage and related bone tissue), assessment of injury level and information on disease complications; recommending treatment measures depending on the severity of the process; Much attention is being paid to the search for markers that allow early detection of damage to joint structures, allowing for monitoring of the treatment measures.

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РЕЗЮМЕ
РЕАКТИВ АРТРИТНИНГ КЕЧИШИДА ЎЗИГА ХОС КЛИНИК
ЖИҲАТЛАРИ

(Адабиётлар шарҳи)

Хошимов Аброржон Анваржонович, Нурматова Наргиза
Фатхуллаевна, Хабибуллоева Бибирозия Рахматуллозода

Тошкент тиббиёт академияси

Калит сўзлар: реактив артрит, цитокинлар, бўғим синдроми, интерлейкин 17, интерлейкин 18.

Тадқиқот мақсади. Реактив артритга чалинган беморларнинг цитокин профилини, хужайра ва гуморал иммунитетини, носпецифик химоя омилини ўрганиш. Реактив артритга чалинган беморлар ревматологик шифохоналарда беморларнинг тахминан 1/3 ташкил қилади ва касалликнинг сурункали шаклида бўғимларнинг функционал қобилиятининг сезиларли даражада йўқолиши ва оғир асоратлар пайдо бўлиши мумкин, бу эса беморларнинг ногиронлигига олиб келади. Реактив артритга бўлган қизиқиш, шунингдек, бир қатор беморларда, айниқса жараён сурункали бўлса, бўғимларда анкилозланишгача бўлган деструктив жараёнларни ривожланиши билан боғлиқ.

РЕЗЮМЕ
КЛИНИЧЕСКИЕ ОСОБЕННОСТИ ТЕЧЕНИЯ
РЕАКТИВНОГО АРТРИТА

(Литературный обзор)

Хошимов Аброржон Анваржонович, Нурматова Наргиза
Фатхуллаевна, Хабибуллоева Бибирозия Рахматуллозода

Ташкентская медицинская академия

Ключевые слова: реактивный артрит, цитокины, суставной синдром, интерлейкин 17, интерлейкин 18.

Цель исследования. Изучить цитокинового профиля, клеточного и гуморального иммунитета, а также фактора неспецифической защиты у больных реактивного артрита. Больные реактивным артритом составляют 1/3 больных ревматологических больных, причем при хронической форме

заболевания может развиваться значительная утрата функциональных способностей суставов и возникновение тяжелых осложнений, что приводит к инвалидизации пациентов. Интерес к реактивному артриту связан также с тем обстоятельством, что у ряда больных, особенно при хронизации процесса развиваются деструктивные процессы в суставах вплоть до анкилозирования.

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ЗНАЧИМОСТЬ В РИСКЕ РАЗВИТИЯ ИНФАРКТА МИОКАРДА ГЕНЕТИЧЕСКОГО ПОЛИМОРФИЗМА ASP919GLY В ГЕНЕ MTR

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Ключевые слова: инфаркт миокарда, COVID-19, генетический полиморфизм ASP919GLY в гене MTR.

Введение. За последние пару лет были опубликованы результаты исследований, ряд обзоров и описания клинических наблюдений, которые были посвящены осложнениям вызванным вирусом SARS-CoV-2, в частности со стороны сердечно-сосудистой системы [1]. Развитие сердечно-сосудистых нарушений усугубляло тяжесть состояния больных и повышало риск летальности. Сообщали о случае с 53-летней больной, у которой клиническими проявлениями COVID-19 стал тяжелый перикардит с лихорадкой, а не пневмония [4]. Действительно, создается впечатление, что риск ССО новой коронавирусной инфекции выше, чем это было отмечено во время эпидемий, вызванных SARS-CoV (тяжелый острый респираторный синдром) и MERS-CoV (ближневосточный респираторный синдром) [7]. У пациентов, умерших от COVID-19, уровни биомаркеров перед смертью были в 12 раз выше при наличии морфологических признаков поражения миокарда, чем при их отсутствии. Повышение значений биомаркеров является признаком неблагоприятного исхода имеющегося заболевания. Несомненно, необходимы дальнейшие исследования диагностической и прогностической роли биомаркеров миокардиального стресса при COVID-19.

Цель исследования. Изучить и оценить вклад полиморфизма Asp919Gly, в гене MTR, в риске развития инфаркта миокарда у больных с вирусной инфекцией COVID-19 в анамнезе и у условно-здоров доноров.