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EVIDENCE-BASED TECHNOLOGY TO DETERMINE THE IMMUNE RESPONSE TO THE EPIDEMIC OF CORONAVIRUS INFECTION

Abstract: Aim: The determination of lymphocyte subpopulations in patients with rheumatoid arthritis using the CD antigen markers, including CD3, CD4, CD8, CD16, CD20, CD25 and CD95. Materials and methods: Was examined 60 people aged 30-65 years were divided into the following 3 main groups: I group: 20 participants (16 female and 4 male) with RA , II group: of 20 individuals (14 female and 6 male) who had previously been diagnosed with SARS-CoV-2 infection, III group: control healthy participants20 healthy volunteers (15 female and 5 male).Results: This finding suggests that CD3+, CD4+, CD8+, CD16+ and CD20+ T-lymphocytes are the predominant CD25-producing cells and affect the content of this molecule not by altering the expression density of the CD25 receptor on their surface, but rather due to the increased number of these cells. Conclusion: The study of T lymphocyte subpopulations, which we have traced in our research, makes it possible to diagnose inflammation in an early stage and serves as an effective tool in primitive medicine.

Key words: lymphocyte subpopulations, rheumatoid arthritis, SARS-CoV-2 infection, inflammation.

INTRODUCTION

Coronavirus disease (COVID-19), which has emerged as a serious concern for the global health community, has also provided an opportunity for a better understanding of the true potential of modern medicine[5]. During this pandemic, medical practitioners have been asked to determine the link between viral infections and a variety of chronic, non-transmissible conditions. Among these conditions, immune-inflammatory rheumatic disorders (IRV) stand out as an area of particular interest in rheumatology, as they are significantly influenced by genetic factors and acquired immunity. IRVs are caused by defects in genetic material, which can be monogenic or polygenic, and result in the development of autoimmune and/or auto-inflammatory processes. These processes are determined by common pathological mechanisms, which lead to the activation of immune cells and the production of antibodies. It is important to note that autoimmune mechanisms can reduce immune tolerance, resulting in pathological immune reactions to autoantigens, which are molecules produced by the body and recognized as foreign by the immune system. This can lead to the development of various IRVs, including systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and other conditions that affect the joints, skin, and other organs. These reactions are associated with the activation of immune cells acquired (T cells and B cells). According to the World Health Organization's (WHO) expert group, the "post-COVID condition" can be discussed in individuals who, according to their medical records, have a possible or definite infection with SARS-CoV-2. We suggest that SARS-CoV-2 infection or vaccination against the virus may be a triggering factor for joint damage and may cause an exacerbation of rheumatoid arthritis (RA), or lead to the development of the disease. RA is an inflammatory autoimmune disorder characterized by a breakdown in T cell tolerance and B cell function in the body, leading to various pathological abnormalities.[5] A population of helper T cells plays a central role in RA development, engaging in inflammation through the use of chemokines

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and cytokines. According to several authors, when assessing the functional activity of immune cells, it is recommended to evaluate the expression of CD25+ and CD95+ surface markers on lymphocyte membranes [6]. CD95+ regulates the response of lymphocytes to antigenic stimulation, determines the nature, kinetics, and duration of the immune response, as well as the formation of immune tolerance [7].

The main aim our research determination of lymphocyte populations and subpopulations taking into account CD antigen indices: CD3, CD4, CD8, CD16, CD20, CD25, CD95 in patients with rheumatoid arthritis by immunofluorescence using monoclonal antibodies.

MATERIALS AND METHODS

The study included 60 participants with a confirmed diagnosis of rheumatoid arthritis (RA) and post-COVID-19 syndrome (average age: 54.2 years [47;62]). The first group consisted of 20 participants (16 female and 4 male) with RA who met the criteria for diagnosis according to the American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) criteria in 2010. The second group consisted of 20 individuals (14 female and 6 male) who had previously been diagnosed with SARS-CoV-2 infection. The control group included 20 healthy volunteers (15 female and 5 male), (median age: 58.5 years [53;62]), who were not diagnosed with either RA or SARS-CoV-2. Peripheral blood T cell counts were determined basedon molecular marker expression analysis for CD3, CD4, CD8, CD4/CD8, CD16, CD20, CD25, a nd CD95 in each participant. The number of lymphocytes was compared with markers for early and late activation stages and compared with the numbers of T- and B-lymphocytes and their subsets in patients in three groups. All participants in the study underwent flow cytometry using a standard protocol with immunophenotyping of T and B cells. The study sample was peripheral blood drawn in the morning on an empty stomach from the ulnar vein and placed in a test tube containing sodium citrate. Commercial monoclonal antibodies labeled with FITC (Sorbent, Russia) were used to determine the number of CD25+ lymphocytes, which are markers for early activation and participate in protecting cells from apoptosis. CD95+, markers for late activation indicating the readiness of lymphocytes to trigger apoptosis, were also determined.[3,4]. The total number of T lymphocytes (CD3+) and helper T lymphocytes (CD4+) were also evaluated, as well as the number of cytotoxic T lymphocytes (CD8+) and natural killer cells (CD16+). Additionally, the number of B lymphocytes (CD20+) was assessed in accordance with the methodology outlined in the provided panel of monoclonal antibodies.

RESULTS.

As a result of research in patients with rheumatoid arthritis (RA) and post-COVID syndrome, there has been a decrease in the relative number of CD25+ lymphocytes (10 (9-12)% compared to the control group (15% (10-20%)) with p=0.002. As for lymphocytes expressing CD95 receptors on their cell surface: Apoptosis, then, their number is relatively (10(7.5–12.5)%), so is the absolute number (0.37(0.31–0.67).109/L) in patients with a cancerous syndrome. The number of these lymphocytes did not change in patients with the cancerous syndrome compared to the control group. In this regard, correlation analysis data we have obtained demonstrate the presence of positive correlations between the number of CD25+ lymphocytes and the numbers of major lymphocyte subpopulations (CD3+, CD4+, CD8+, CD16+, and CD20+). These correlations were present in patients with both

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the cancerous andrheumatoid arthritis (RA) syndromes, as well as in the control group, indicating that there may be a relationship between the levels of theselymphocyte populations and the clinical status o f the patients.R =0.69 and p < 0.00001 respectively). This finding suggests that CD3+, CD4+, CD8+, CD16+ and CD20+ T-lymphocytes are the predominant CD25-producing cells and affect the content of this molecule not by altering the expression density of the CD25 receptor on their surface, but rather due to the increased number of these cells. [1,2]

The results obtained confirm those of other researchers who have found that the CD25 molecule is present on various lymphoid cell types, and its expression level increases as the body's response to an infection or other inflammatory process intensifies. When comparing the patients in the two groups to controls, the relative levels of all T-ell subpopulationswere significantly higher. These findings indicate a rapid response of T cells were significantly higher. These findings indicate a rapid response of T cells to a pathogen.

Discussion.

Based on the results obtained, a significant increase in the number of T-lymphocyte subpopulations was revealed. We believe that a moderate increase in these lymphocytes' subpopulations in our study can be used to characterize the disease activity as being either low or moderate, which corresponds to the level of activity in RA and post-COVID-19 RA in the patients studied.

Of particular note is the increased number of cytotoxic lymphocytes, by 51.1%, compared to those in the control group. This corresponds to much of the data from other researchers and completely aligns with the general picture of immune changes in inflammation. Currently, determining the level of activated T-cells is crucial for diagnosing and predicting the course of autoimmune and lymphoproliferative disorders.[8,9] Thus, a decrease in the level of these cells, when prescribing treatment, is a favorable prognostic factor for another autoimmune disease - rheumatoid arthritis. An increase in the number of these cells is accompanied by a deterioration in the overall condition of the patient. The study of T lymphocyte subpopulations, which we have traced in our research, makes it possible to diagnose inflammation in an early stage and serves as an effective tool in primitive medicine.

Conclusions

The significant violations of specific cellular immunity revealed in patients with rheumatoid arthritis with post-COVID syndrome, as well as correlations found with general immunity, suggest that these indicators may play an important role in the mechanisms underlying autoimmune pathology and may act as an additional triggering factor for the development and maintenance of autoimmune processes.

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