



**KLINIK LABORATOR
DIAGNOSTIKADA INNOVATSION
TEXNOLOGIYALARDAN
FOYDALANISH, MUAMMOLAR VA
YECHIMLAR**
**xalqaro ilmiy-amaliy
anjuman**
18 aprel 2023 yil



O'zbekiston Respublikasi Sog'liqni saqlash vazirligi

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HEMOSTASIOLOGICAL CHANGES IN RHEUMATOID ARTHRITIS

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A study was made of the features of hemostatic disorders in patients with rheumatoid arthritis, depending on the activity and duration of the pathological

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process. It has been shown that changes in the hemostasis system with a tendency to hypercoagulability and thrombosis are formed in patients with rheumatoid arthritis against the background of inflammation, depending on the activity of the pathological process and the duration of the disease. Persons with the debut of rheumatoid arthritis, not exceeding 18 months from the onset of the first clinical manifestations, in the absence of disease-modifying therapy, have laboratory manifestations corresponding to the activation of the hemocoagulation cascade. Inflammation in active articular syndrome at the onset is characterized by an increase in the adhesive properties of platelets, hypercoagulability, thrombinemia, and a decrease in the reserves of the fibrinolytic system. The most important prognostic laboratory sign associated with the severity of inflammation in rheumatoid arthritis is thrombinemia.

Materials and methods of research. The study included two groups of patients diagnosed with RA. The first group included 101 patients with newly verified very early and early RA (7.6 ± 3.9 months) who were followed up for 10 years, the second group included 363 patients with advanced and late RA (7.3 ± 6.4 years). In the control group, the study of the hemostasis system and general clinical laboratory parameters was performed in 36 practically healthy individuals, comparable in sex and age with patients suffering from RA. The hemostasis system was studied at the stage of diagnosis, in the early and very early phases of RA before the start of the use of basic anti-inflammatory drugs (DMARDs), in dynamics after 5 and 10 years (group 1). In patients with advanced and late stages of the disease, with the ineffectiveness of previous therapy, the dose of the selected drug was adjusted (group 2). Patients in the debut were examined before the appointment of DMARDs. Subsequently, they were followed up at points 5 and 10 years from the onset of the disease and, like RA patients with a long history of the disease, they received methotrexate therapy at a dose of 10–17.5 mg per week, sulfasalazine 2 g per day, or combination therapy with these drugs.

Mathematical processing of the obtained results was carried out using the Statistica 6.0 software package. Comparison of variation series was carried out using Student's t-test and non-parametric Wilcoxon-Mann-Whitney U-test. Correlation analysis was carried out using Spearman's rank correlation method. Informed consent was obtained from all patients.

Thus, in patients with rheumatoid arthritis, significant deviations in the coagulation, anticoagulation and fibrinolytic blood systems are revealed, the severity of which varies depending on the activity of the pathological process and the duration of the disease.

The revealed inhibition of XIIa-dependent fibrinolysis deficiency was associated with the severity of inflammation and was maximal at high clinical and laboratory activity of rheumatoid arthritis. It can be assumed that fibrinolysis depression develops as a result of current inflammation and has a protective function in terms of limiting the aggressive destruction of articular cartilage during rheumatoid inflammation.

A hypercoagulable shift was detected by us in patients with rheumatoid arthritis, regardless of the severity of inflammation and the duration of the disease

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history. At the same time, according to the results of the study, the antithrombin potential of the blood increases with the progression of the disease, which can be considered as a consequence of the anti-inflammatory effect of prolonged immunosuppressive therapy and a decrease in the exudative component in the evolution of the disease. The increase in antithrombin potential, according to our data, does not include the activation of antithrombin III, which requires a study to clarify the involvement of other natural anticoagulants in the formation of increased antithrombin activity in the blood of RA patients.

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