mgr Paweł Kozłowski

Ocena markerów uszkodzenia śródbłonka i rozwoju autoimmunizacji po zakażeniu SARS-CoV-2 u osób nieobciążonych dodatkowymi czynnikami ryzyka

Rozprawa na stopień doktora nauk medycznych i nauk o zdrowiu w dyscyplinie nauki medyczne.

Promotor: prof. dr hab. n. med. i n. o zdr. Olga Ciepiela

Zakład Medycyny Laboratoryjnej
Warszawskiego Uniwersytetu Medycznego

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Streszczenie w języku angielskim

Evaluation of markers of endothelial damage and development of autoimmunity after SARS-CoV-2 infection in subjects without additional risk factors.

In November 2019, cases of respiratory infection emerged in the Chinese city of Wuhan. The new disease called coronavirus disease 2019 (COVID-19) was caused be the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). In May 2020, the World Health Organization declared a pandemic state due to the rapid spread of the virus and the exponential increase in the number of infections. From the beginning of the pandemic until March 2023, there were more than 700 million infections and almost 7 million deaths from COVID-19. At the same time, reports of extrapulmonary manifestations of the disease and delayed complications of SARS-CoV-2 virus infection began to appear. Infection-induced endothelial cell damage and the development of autoimmune processes contribute to the development of these complications. Both of these phenomena, which in the long term can lead to significant health problems in the population, require in-depth studies due to the number of potentially affected individuals. During the initial period of the pandemic, the attention of the scientific community was focused mainly on patients with a severe course of COVID-19 infection. At the same time, generally healthy people unencumbered by risk factors for a severe course of COVID-19 infection also became infected. Individuals who underwent COVID-19 were diagnosed with prolonged health dysfunctions unrelated to any other disease other than COVID-19. Such complications have been called “long COVID”. It is estimated that up to 45% of those originally infected with SARS-CoV-2 may develop “long COVID”. These estimates include those with previously confirmed infection, with a severe or moderate course of the disease. It is also important to remember that infection with SARS-CoV-2 can be mild or even asymptomatic. It therefore became reasonable to investigate whether a history of asymptomatic to moderate SARS-CoV-2 infection in individuals without additional risk factors could lead to persistent endothelial cell damage and the development of autoimmune processes.

The aim of this study is to further our understanding of the effects of SARS-CoV-2 virus infection on endothelial cells and the development of autoimmunity in individuals without additional risk factors.

A group of 294 honorary blood donors was included in the study. Strict eligibility criteria prior to blood donation allowed the study to qualify individuals as unencumbered by additional risk factors. Qualified donors were tested for antibodies to the N protein of SARS-CoV-2 virus as a marker of past infection. Based on the results, donors were divided into a test group (those with antibodies) n=215, and a control group (those without antibodies) n=79. In each group, concentrations of selected markers of endothelial damage and glycocalyx were evaluated (VCAM-1, ICAM-1, E-selectin, syndecan-1), and tests for antinuclear antibodies and against β2-glycoprotein I were performed.

Features of persistent endothelial damage were observed in the convalescents without additional risk factors not less than 6 months after infection. Convalescents had higher E-selectin levels (1754 pg/mL vs 1633 pg/mL, p=0.0135) and lower syndecan-1 levels (692 pg/mL vs 934 pg/mL, p=0.0082) than the control group. Tests for the presence of antinuclear antibodies and antibodies against β2-glycoprotein I did not show a higher prevalence in the convalescents compared to the control group.

The results allowed us to conclude that people without additional risk factors may be at risk of developing diseases associated with persistent endothelial cell damage after infection with SARS-CoV-2. There was also no increased risk of developing autoimmune processes, including those leading to thrombosis, in the studied convalescents. Due to the huge number of people who have undergone infection with the SARS-CoV-2 virus, the variability of the virus itself, the introduction of immunization, and the complex nature of autoimmune diseases, it is necessary from the public health perspective to deepen research and expand knowledge of the described phenomena.