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DAMAGE TO HEMOPOIESIS WITH SARS-COV-2

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Annotation
The characteristics of the SARS-CoV-2 coronavirus infection seriously complicate the prediction of the course of the disease and make it almost unpredictable. Viral infections cause acute respiratory distress syndrome, severe pneumonia, hematological changes, and decrease the quality of life. In this regard, an extremely important scientific direction is the search for ways of interaction between the virus and cells, as well as the study of aspects of the virus that increase its pathological effect on the body and lead to hyperactivation of the immune system. is considered It is very important to identify additional factors that affect the blood system of SARS-CoV-2, to focus on clinical improvement, and to improve the results of treatment of patients.

Keywords: SARS-CoV-2, coronavirus, hematopoiesis, angiotensin-converting enzyme.

At the moment, the new coronavirus infection that dominates the whole world is the SARS-CoV-2 virus, whose pandemic began in 2019. More than 430 million people have been infected with this infection, and about 6 million of them have died [2]. In the Russian Federation, more than 16 million new coronavirus infections and 350,000 deaths were recorded during the pandemic [1,3]. The prognosis for the severity of infection and possible transfer from the upper respiratory tract to the lower respiratory tract depends on the virulence of the viral agent, possible co-infections and the age of the patient, the main or concomitant respiratory and cardiac– depending on the presence of vascular diseases. Immune Deficiency Syndrome [9] The lack of information on the virulence factors specific to SARS–CoV–2 currently makes the treatment of patients infected with the novel coronavirus very difficult. A cluster of studies has been published that provides empirical evidence for virulence factors specific to SARS–CoV–2 that may explain key elements of the pathology of COVID–19. These studies partially reveal structural and non-structural features of SARS-CoV-2 that give it an advantage in virulence over previous pandemic coronaviruses [11,13,17]. A cluster of studies has been published that provides empirical evidence for virulence factors specific to SARS–CoV–2 that may explain key elements of the pathology of COVID–19. These studies partially reveal structural and

non-structural features of SARS-CoV-2 that give it an advantage in virulence over previous pandemic coronaviruses [11,13,17]. A cluster of studies has been published that provides empirical evidence for virulence factors specific to SARS–CoV–2 that may explain key elements of the pathology of COVID–19. These studies partially reveal structural and non-structural features of SARS-CoV-2 that give it an advantage in virulence over previous pandemic coronaviruses [11,13,17].

The age aspect is very important, because respiratory viral infections are the main cause of death in young children as well as in the elderly [3]. According to a meta-analysis by Gallo Marin et al. (2021), being over 55 years of age in patients infected with SARS-CoV-2 is associated with increased disease severity and/or mortality [4].

Comorbidities that significantly worsen the prognosis of respiratory infections include: chronic diseases of the cardiovascular and respiratory systems, diabetes, kidney or liver diseases, blood diseases, malnutrition, and immunodeficiency [6,15]. Immunocompromised patients include transplant recipients, cancer patients undergoing chemotherapy, rheumatic patients treated with immunosuppressants, and individuals with primary immunodeficiency or human immunodeficiency virus infection [16]. In the latter categories of patients, respiratory viral infections occur as often as in immunocompromised people, but upper respiratory tract infections, lower respiratory tract infections, associated with high mortality [8]. The difficulty of predicting disease severity in novel coronavirus infection is also emphasized by the fact that the virus has tropism for various tissues, including primarily the respiratory tract, as well as the brain, endothelium, heart, kidneys, and liver [10]. According to recent studies, a D-dimer level ≥2.0 μg/mL, elevated troponin I (>13.75 ng/L), total lymphocytes, CD4+ and CD8+ T cells, B-cell depletion several markers, hypoalbuminemia, elevated liver fibrosis index (FIB-4), lactate dehydrogenase, and ferritin levels are considered poor predictors of novel coronavirus infection [11]. The difficulty of predicting disease severity in novel coronavirus infection is also emphasized by the fact that the virus has tropism for various tissues, including primarily the respiratory tract, as well as the brain, endothelium, heart, kidneys, and liver [10]. According to recent studies, a D-dimer level ≥2.0 μg/mL, elevated troponin I (>13.75 ng/L), total lymphocytes, CD4+ and CD8+ T cells, B-cell depletion several markers, hypoalbuminemia, elevated liver fibrosis index (FIB-4), lactate dehydrogenase, and ferritin levels are considered poor predictors of novel coronavirus infection [11]. The difficulty of predicting disease severity in novel coronavirus infection is also emphasized by the fact that the virus has tropism for various tissues, including primarily the respiratory tract, as well as the brain, endothelium, heart, kidneys, and liver [10]. According to recent studies, a D-dimer level ≥2.0 μg/mL,

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Damage to hematopoiesis in SARS-CoV-2 is one of the main glycolipid components of the outer membrane of gram-negative bacteria, which makes up about 70% of the intestinal bacterial flora [4,9]. The interaction between cells of the monocyte-macrophage series is carried out mainly through receptors mCD14 (membrane CD14) and TLR-4/MD-2 (type 4 receptors with adapter protein - MD-2). General blood analysis is the most important part from the point of view of immunogenicity [5, 16]. Depending on the number of acyl chains, endotoxin is divided into several subtypes. Hexaacylated (containing six acyl linkages) lipid A has the greatest ability to activate the TLR4/MD–2 complex and subsequently trigger the pro-inflammatory cascade, and its anti-inflammatory activity is mediated by the lipid A pathway. binds to the TLR4/MD–2 complex [17]. There are now reports of several forms of anti-inflammatory activity that can compete with the immunogen for binding to the TLR4/MD–2 complex, thereby triggering the next phase of the pro-inflammatory response. but this data is limited due to the small number of studies [5]. The "anti-inflammatory" type of lipopolysaccharide, which contains tetraacyl lipid A, is characteristic of bacteria of the genus Bacteroides. Thus, endotoxin can be transported into the blood circulation by direct diffusion with absorption by intestinal epithelial cells during paracellular permeability of the intestine or secretion of chylomicrons. In addition, it binds to HDL, LDL or chylomicrons in hepatocytes, and is subsequently deacylated (inactivated) and excreted with bile [1,3,8]. Gas exchange in the lungs can be affected by a number of conditions such as viral infections, dysfunction of the capillary network in the alveoli, antibiotic therapy, and the balance of micro- and macroelements in the diet [11, 12]. The impact of these pathological conditions on the increase in blood volume was estimated by local scientists at the end

of the 20th century. According to a study by Hennezel et al. (2017) in overweight and obese individuals, alveolar imbalance leads to an increase in the entry of hexaacyl lipid A into the systemic circulation [5,15]. High-calorie, high-lipid diets also alter the distribution and architecture of tight junctions through various mechanisms, activate alveolar cell apoptosis, stimulate a cascade of pro-inflammatory signals known to increase intestinal permeability, and produce alveolar-damaging cytokines. will increase reduces the level of barrier cytokines; the composition of the alveoli is modulated and the alveoli is enriched with mucolytic species [11, 13].

A study by Petruk et al. (2021) described interactions between the coronavirus C-reactive protein and, leading to increased inflammation in vitro and in vivo. Together with protein C-endotoxin, THP-1 enhances nuclear factor-kappa B (NF-κB) activation in monocyte cells and cytokine response in monocyte-macrophage cells. Dynamic light scattering, transmission electron microscopy, and –FITC analysis showed that C modulates the aggregation state of protein, which provides an explanation for the observed enhancement effect at the molecular level.

Also, the first link in the pathogenesis of viral lung damage - the development of endothelial dysfunction and the impact on the development of pulmonary edema - is very interesting. Initially, these changes are caused by the retention of microaggregates with a decrease in pulmonary capillary perfusion by the pulmonary endothelium, the destruction of stored products with the formation of anti-inflammatory mediators that damage the interstitium, and the inhalation of toxic substances or microbial flora damages alveolocytes and occurs as a result of lung exposure to surfactant [6].

One of the main roles in the novel coronavirus infection is played by a decrease in the level of angiotensin-converting enzyme 2 (ACE2), which converts angiotensin II to angiotensin 1–7, which stimulates the production of nitric oxide (NO) in endothelial cells. NO promotes vasodilation and suppresses platelet aggregation. In SARS-CoV-2 infection, the level of angiotensin II increases, which leads to vasoconstriction and a decrease in blood flow. After SARS–CoV–2 activation, monocytes and other cells express tissue factor and phosphatidylserine on their surface and initiate procoagulant changes in the pulmonary vascular bed [1,2].

Sodhi et al (2018) tested the effect of ACE2 on des–Arg9 bradykinin in respiratory epithelial cells in laboratory animals, as DABK is a biological substrate of ACE2 in the lung and ACE2 plays a role in the pathogenesis of acute disease. Pulmonary inflammation, in part due to modulation of DABK axis/bradykinin B1 (BKB1R) receptor signaling. Loss of ACE2 function in the lungs of laboratory mice by endotoxin inhalation was found to lead to activation of the DABK/BKB1R axis, release of

chemokine 5 (CXCL5), macrophage inflammatory protein-2 (MIP2), and pro-inflammatory cytokines. This interaction leads to increased infiltration of lung tissue by neutrophils, causes increased inflammation and damage to the parenchyma. In this regard, decreased ACE2 activity in the lung during loading exacerbates lung inflammation, in part due to impaired ability to suppress signaling through the DABK/BKB1R axis, resulting in increased neutrophil recruitment and increased pulmonary inflammation. it can be assumed that [4,7,12].

The complement system may also be involved in the pathological interaction of SARS–CoV–2. In the case of SARS–CoV–2, complement activation is excessive, leading to life-threatening acute inflammatory processes, endothelial cell damage, and promoting intravascular coagulation [16]. Also, because it is a potent activator of the complement system, triggering the lectin-mediated cascade described in SARS–CoV–2, endotoxin is a molecule that enhances this type of immune response.

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