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FEATURES OF THE CORONAVIRUS INFECTION COVID-19 PATHOGENESIS AND METHODS OF ITS SPECIFIC DIAGNOSIS

Abstract. *The COVID-19 pandemic has led to global health problems in many countries. The rapid spread of the disease, high mortality and the development of severe complications have posed challenges to specialists related to rapid diagnosis and provision of medical care to patients. The aim of the work is to analyze the scientific data on the mechanisms of development of the pathogenesis of COVID-19 coronavirus infection caused by SARS- CoV-2 to determine specific methods for diagnosing the virus, treatment tactics and ways to prevent the spread of infection. The pathogenesis of COVID-19 coronavirus disease occurs with primary lung damage and late hematological, tissue hypoxemia and mitochondrial dysfunction due to oxidative stress.*

Keywords: *coronavirus infection COVID-19, pathogenesis, SARS-CoV-2, Angiotensin-Converting Enzyme 2, cytokine storm*

Introduction. Coronavirus infection (COVID-19) is an acute infectious disease caused by a new strain of the coronavirus family SARS CoV-2, which was discovered in the second half of 2019 and belongs to the genus Betacoronavirus. The COVID-19 pandemic has led to global health problems in many countries. The rapid spread of the disease, high mortality rate and the development of severe complications have posed challenges to specialists related to rapid diagnosis and provision of medical care to patients. The most common clinical manifestation of the new variant of coronavirus infection is bilateral pneumonia (viral diffuse alveolar damage with microangiopathy), acute respiratory failure (ARF), and 3–4 % of patients have developed acute respiratory distress syndrome (ARDS) [1,2]. Some patients develop hypercoagulable syndrome with thrombosis and thromboembolism, and other organs and systems are also affected (central nervous system, myocardium, kidneys, liver, gastrointestinal tract, endocrine and immune systems) [2]. To prevent the further spread of the pandemic, researchers in all countries continue to intensively study the clinical and epidemiological features of the disease, and develop new means of its prevention and treatment.

The aim of the work is to analyze the scientific data on the mechanisms of development of the pathogenesis of COVID-19 coronavirus infection caused by SARS- CoV-2 to determine specific methods for diagnosing the virus, treatment tactics and ways to prevent the spread of infection.

Results and discussions.

Coronaviruses (Coronaviridae) are a large family of RNA viruses capable of infecting both humans and animals. ased on serological and phylogenetic analysis, coronaviruses are divided into two subfamilies, Letovirinae and Orthocoronavirinae, which include four genera: Alphacoronavirus, Betacoronavirus, Gammacoronavirus, Deltacoronavirus [3].

Today, four types of coronaviruses (HCoV-229E, HCoV-OC43, HCoV-NL63 та HCoV-HKU1), are known to circulate among the population of Ukraine and other countries, which are constantly identified as part of ARVI. They can cause mild to moderate damage to the upper respiratory tract. [3,4].

SARS-CoV-2 is an enveloped RNA virus with a helical capsid symmetry. The virion diameter is 50–200 nm. There are three proteins on the surface of the supercapsid: S – spike glycoprotein, which forms peplomers and gives the virus its characteristic crown shape; M – membrane glycoprotein; and E – envelope. The fourth protein is N-nucleocapsid phosphoprotein, which is a structural component of the nucleocapsid. The S protein (spike) is capable of binding to the ACE2 (angiotensin-converting enzyme 2) receptor, a membrane protein that is part of the renin-angiotensin system (RAS), regulates blood pressure, inflammation, and vascular tone in type II alveolar cells, bronchial and tracheal epithelium, nasal mucosal cells, cardiomyocytes, intestinal enterocytes, and other cells. [6,7,8].

The S protein enables the virus to enter the cell by fusing membranes. It consists of two subunits: S1, which is responsible for the receptor-binding domain (RBD), and S2, which is responsible for membrane fusion. The M protein (membrane glycoprotein) is the main structural protein of the SARS-CoV-2 virus envelope, which determines the shape of the virus, coordinates the assembly of viral particles, and interacts with the S, E, and N proteins.

E-protein (Envelope) is a small protein of the viral envelope that participates in the assembly and release of the virus during its replication in susceptible cells of a macroorganism, forming ion channels (virporins). N-protein (Nucleocapsid) is capable of binding viral RNA, forming a nucleocapsid and stabilizing the viral genome, participates in the replication and packaging of RNA, thereby participating in the stimulation of the cell's immune response. The SARS-CoV-2 virus genome consists of single-stranded, linear, positively charged, non-fragmented RNA, containing 26 to 32 thousand nucleotide pairs. Coronaviruses have the largest genome among all RNA virus families [3,4,7].

SARS-CoV-2 also contains non-structural proteins (nsps) nsp1-nsp16 and accessory proteins, which are not part of the virion but are necessary for viral RNA replication, protein synthesis, and suppression of the immune response. [3,4,9].

SARS-CoV-2 enters target cells expressing ACE2 via the endosomal pathway. The viral S protein binds to the ACE2 cell receptor and is translocated into endosomes, where the S protein is cleaved by endosomal acid proteases (cathepsin L) to activate the target cell nucleus enzyme complex. After deproteinization, the viral genome is released and translated into viral replicase. Subgenomic negative-strand matrices are transcribed onto the plus strand and serve as matrices for mRNA synthesis, resulting in the synthesis of viral structural proteins. Viral nucleocapsids are assembled from genomic RNA and N protein in the cytoplasm, followed by budding into the lumen of the endoplasmic reticulum (ER) and Golgi apparatus. The virions are then transported in vesicles to the cell membrane and released from the target cell by exocytosis. [10]

The entry point for the pathogen is the epithelium of the upper respiratory tract and the epithelial cells of the stomach and intestines. The initial stage of infection is the penetration of SARS-CoV-2 into target cells that have ACE2 receptors. The main and most readily accessible target is type II alveolar cells (AT2) of the lungs, which determines the development of pneumonia [6-9]. The role of CD147 in SARS-CoV-2 cell invasion is also being discussed [9]. The spread of SARS-CoV-2 from the systemic bloodstream or through the lamina cribrosa can lead to brain damage [11,12]. Hyposmia in a patient in the early stages of the disease may indicate damage to the central nervous system. Diffuse alveolar damage develops [11,12]. The virus causes increased cell membrane permeability and enhanced transport of albumin-rich fluid into the interstitial tissue of the lung and alveolar lumen. Interstitial and alveolar edema develops. This destroys the surfactant, leading to alveolar collapse, and as a result of a sharp disruption in gas exchange, ARDS develops [7,8]. Several stages can be identified in this process. The exudative (acute) stage is characterized by damage to type I alveolar cells, which leads to increased permeability of the alveolar-capillary membrane of cells, causing interstitial and alveolar edema and filling of the alveoli with leukocytes, erythrocytes, and products of destroyed cells. The proliferative (subacute) stage is characterized by damage to type II alveocytes, leading to the migration of fibroblasts into the alveolar exudate and the proliferation of type II alveocytes, resulting in a reduction in pulmonary edema. The fibroproliferative (chronic) stage is characterized by obliteration of the alveoli, leading to fibrosis of the pulmonary parenchyma [7, 8].

It has been proven that SARS-CoV-2 can directly infect human blood vessel epithelial cells in vitro. Direct damage to endothelial cells by SARS-CoV-2 or indirect damage by immune cells, cytokines, and free radicals can lead to severe endothelial dysfunction. The vascular endothelium is considered an active paracrine, endocrine, and autocrine organ that actively participates in the regulation of blood vessel tone and maintenance of their homeostasis. Endothelial dysfunction that develops in COVID-19 causes microcirculation disorders, vasoconstriction with subsequent organ ischemia, inflammation and tissue edema, and procoagulation [7,8]. Endothelial dysfunction can cause systemic microcirculation disorders in various vascular beds, with clinical consequences in patients with COVID-19 coronavirus disease. Endothelial dysfunction dictates the need for appropriate treatment to normalize endothelial function while fighting pathogen replication, especially therapy with anti-cytokine drugs, ACE inhibitors, and statins [9]. This strategic approach is particularly relevant for patients with significant risk factors for endothelial dysfunction (hypertension, diabetes mellitus, obesity, cardiovascular disease, smoking). Endothelial dysfunction and chronic inflammation go hand in hand with hypercoagulation, accompanied by increased fibrinogen levels and decreased fibrinolysis and anticoagulation, which ultimately leads to thrombus formation [12].

In response to SARS-CoV-2 infection during exudation and proliferation, T-cell immunity prevails. When these cells are replaced by fibrosis, the total number of T-lymphocytes decreases sharply, while humoral immunity cells have not yet appeared. The predominance of CD8+ suppressor T-lymphocytes over CD4+ helper T-lymphocytes is likely associated with mechanisms of autoimmune damage. Lymphopenia in patients may indicate both hyperactivation and migration of lymphocytes to the lungs, as well as apoptosis and suppression of T-lymphocytes [13,14].

Susceptibility to SARS-CoV-2 infection remains high among people of all age groups. The main routes of transmission are: airborne droplets; airborne dust; fecal-oral; through infected objects and surfaces (fomites). It is known that the virus can be transmitted through blood from mother to child. Infection with SARS-CoV-2 can occur through direct or indirect contact [4]. Some publications also report the detection of the virus in wastewater, indicating the possibility of transmission through water. This increases the risk of COVID-19 spreading, especially in areas with poor hygiene [4]. Individuals with asymptomatic forms of COVID-19 may also be a source of infection for patients in the high-risk group. Coronaviruses can cause disease in both domestic and wild animals, but in most cases, infections remain asymptomatic. These animals include horses, camels, cattle, pigs, dogs, cats, rodents, birds, ferrets, minks, bats, rabbits, snakes, and other wild animals. The incubation period is 2 to 14 days, with an average of 5 to 7 days. There are mild, moderate, and severe forms of COVID-19 infection. In severe cases, there is rapidly progressive lower respiratory tract involvement, pneumonia, ARF, ARDS, sepsis, and septic shock [3,4]. Cytokine storm is an important factor influencing the outcome of COVID-19. Cytokine storm syndrome is an immunopathological condition characterized by a sharp increase in pro-inflammatory cytokines after stimulation of the body by microorganisms or drugs [14]. Under normal conditions, the levels of pro-inflammatory cytokines and anti-inflammatory cytokines in the body remain relatively balanced. When a virus enters the human body, excessive activation of the immune system is triggered, including dendritic cells, macrophages, lymphocytes, and natural killer cells. These cells secrete large amounts of cytokines: IL1, IL2, IL6, IL7, IL8, IL9, IL10, IL12, IL17, IL18, granulocyte colony-stimulating factor (G-CSF), granulocyte-macrophage colony-stimulating factor (GM-CSF), tumor necrosis factor α, IFNγ-inducing protein 10, IFNα and IFNβ, monocyte chemoattractant protein 1 (MCP-1), macrophage inflammatory protein 1α (MIP-1α) [13,14]. At the same time, the level of inflammatory markers such as C-reactive protein and ferritin increases. The difference between cytokine storm in COVID-19 is that the target organ is the lungs. This is due to the tropism of the coronavirus to lung tissue. Hyperactivation of the immune response in COVID-19 is often limited to the pulmonary parenchyma adjacent to the bronchial and alveolar lymphoid tissue and is associated with the development of ARDS [15].

The cytokine storm in COVID-19 usually leads to the development of multiple organ failure and can be fatal. Cytokines can increase the permeability of the blood-brain barrier, allowing the virus to enter the brain. Once in the CNS, the virus can infect astrocytes and microglia, activating a cascade of neuroinflammation and neurodegeneration through the release of tumor necrosis factor, cytokines, and other inflammatory mediators [15].

SARS-CoV-2 infection is associated with a decrease in blood oxygen levels even in patients without hypoxia. This discrepancy illustrates the need to explain whether the virus directly or indirectly affects erythropoiesis. A significantly higher number of erythrocyte precursors – CD71+ cells – were found in the blood circulation of patients with COVID-19. These cells were found to have pronounced immunosuppressive properties. A strong negative correlation was found between the number of these cells and the T- and B-cell ratios in patients with COVID-19 [13-15].

Post-COVID long-haul syndrome is quite common in people who have had COVID-19. Post-COVID long-haul syndrome can affect anyone who has been diagnosed with COVID-19 caused by SARS-CoV-2 and who has not returned to their previous level of health and functioning six months after the illness. According to various data, such symptoms are observed in 10 to 50 % of people who have had COVID-19. There are two groups of such patients: those with irreversible damage to the lungs, heart, kidneys, or brain that affects their ability to function; and those who continue to experience debilitating symptoms despite the absence of noticeable organ damage [16]. Whatever the nature of the changes associated with the prolonged persistence of clinical manifestations or the appearance of new symptoms after an acute illness, their presence necessitates the rehabilitation of such patients. The manifestations of post-COVID syndrome are very diverse and are observed for several months after the infection. The main symptom is increased fatigue (in 100 % of cases). In 90 % of patients after recovery, intense headaches are noted, which are not associated with intoxication and may be accompanied by vestibular disorders, hearing and vision impairment [16]. Ophthalmological changes associated with COVID-19 are related to eye diseases such as conjunctivitis. 60 % of patients experience mental disorders, 20 % experience depression and low mood, 28 % experience symptoms of anxiety, and 28–56 % experience various problems with memory and mental acuity [16,17]. For a long time, 75 % of patients may experience hypothermia or hyperthermia that cannot be controlled with paracetamol [16,17]. In addition, there may be sore throat (85 %), painful lymph nodes (80 %), myalgia (80 %), joint pain (80 %), paresthesia, and impaired movement and sensitivity in various parts of the body. Hearing problems, hair loss, and tooth decay may also occur. Some men experience impotence [17].

Elderly patients may present with atypical symptoms without fever or cough due to reduced reactivity. COVID-19 symptoms may be mild and not reflect the severity of the disease and prognosis. Atypical symptoms of COVID-19 in elderly patients include delirium and confusion [18].

The clinical symptoms of COVID-19 in children correspond to the clinical picture of SARS caused by other viruses: fever, cough, sore throat, sneezing, weakness, myalgia. The most common manifestation of severe acute respiratory syndrome is bilateral viral pneumonia complicated by ARDS or pulmonary edema. Respiratory arrest is possible, requiring emergency care and the use of mechanical ventilation (MV). Adverse outcomes develop with progressive respiratory failure and secondary infection in the form of sepsis. Possible complications include ARDS, acute heart failure, abdominal pain, and diarrhea [19]. Tachycardia is observed in half of hospitalized children, and tachypnea in one-third of children [19]. Not all children with suspected COVID-19 who had severe forms of the disease were laboratory-confirmed to have SARS-CoV-2, which does not rule out combined infections or the presence of other respiratory diseases in children with suspected cases based on clinical and epidemiological data [19].

For specific laboratory diagnostics, PCR (polymerase chain reaction) methods are used to detect SARS-CoV-2 RNA, and immunochromatographic methods are used to detect the virus antigen. In modern laboratory diagnostics, quantitative real-time reverse transcription PCR (rqRT-PCR) is popular for detecting coronavirus due to its high specificity, simple quantitative analysis, and greater sensitivity compared to conventional RT-PCR. Today, for more effective diagnostics, a variety of methods are used to improve the quality of real-time reverse transcription PCR (RT-PCR). Detection of SARS-CoV-2 RNA by RT-PCR with hybridization-fluorescence detection involves three steps: RNA extraction from samples of the test material, reverse transcription reaction, and amplification of the complementary DNA (cDNA) region of the coronavirus with hybridization-fluorescence detection [20]. At the present stage, a method based on microchips is also used to detect coronavirus. The principle of the method is that, with the help of reverse transcriptase, the coronavirus RNA produces cDNA labeled with specific probes. These labeled cDNAs are then placed in the wells of a plate and hybridized with solid-phase oligonucleotides fixed on a microchip, followed by washing to remove free DNA. As a result, coronavirus RNA can be detected by examining specific probes [21].

Loop-mediated Isothermal Amplification (LAMP) is a new method of nucleic acid amplification for detecting SARS-CoV-2 RNA. The essence of this method is to double a DNA segment with high specificity, efficiency, and speed under constant temperature conditions. When combined with reverse transcription, LAMP can amplify RNA sequences with high efficiency. This method is quite fast and highly sensitive and does not require complex equipment [22].

Recombinase Polymerase Amplification (RPA) is another method of isothermal nucleic acid amplification that can be used to detect SARS-CoV-2. The advantages include the speed of the method and the need for moderate heating to 37–42°C, which makes the temperature control module of the equipment much simpler and more economical.

CRISPR-based tests can detect viral genomes using fewer steps than PCR diagnostics. They are highly sensitive and specific, but cannot be performed quickly if the virus concentration is below 10 nM [23].

The molecular biological method of genome sequencing is also used to diagnose SARS-CoV-2, but it is a complex technology that requires special equipment to identify complete genome sequences in the sample being tested. Although nucleic acid amplification-based tests are currently the gold standard for COVID-19 diagnosis, their value for identifying the main modes of transmission and localizing foci of infection is limited. Scientists have developed a combined approach that combines whole-genome sequencing of SARS-CoV-2 and epidemiological data to track the epidemic spread of the virus. Third-generation sequencing technologies based on nanopores were used. These technologies can contribute to the development of more effective measures to combat COVID-19 [24].

Direct detection of SARS-CoV-2 antigens is used to confirm the presence of the virus. Antigens are molecules that are specifically recognized and bound by antigen receptors on the surface of T and B lymphocytes, can trigger antibody production, and induce cell-mediated immune responses. Rapid tests for the detection of SARS-CoV-2 antigens are based on the principle of immunochromatographic analysis with visual recording of test results. As a rule, antigens are detected in nasopharyngeal swabs in patients with suspected coronavirus infection during the first 5–7 days of acute respiratory disease. Express tests are qualitative methods and can only be used to detect SARS-CoV-2 antigens in nasopharyngeal swabs from individuals suspected of having coronavirus infection. The test cannot be used to determine the quantitative content of the pathogen or the concentration of SARS-CoV-2 antigens in the test material. A positive result only indicates the presence of SARS-CoV-2 antigens in the sample and cannot be the sole criterion for diagnosing coronavirus infection. It is important to remember that a negative test result does not rule out coronavirus infection, especially when it comes to contact persons. A negative result is also possible when the antigen concentration in the sample is very low [25].

The choice of diagnostic method depends on the stage of the disease. The main type of biomaterial for laboratory testing for SARS-CoV-2 RNA is material obtained from a nasopharyngeal and oropharyngeal swab. If there are signs of lower respiratory tract involvement and the results of nasal and oropharyngeal swabs are negative, sputum (if available) or bronchial lavage fluid obtained during fiberoptic bronchoscopy (bronchoalveolar lavage), endotracheal, and nasopharyngeal aspirate. In intubated patients (on mechanical ventilation), tracheal aspirate should be collected and examined to detect SARS-CoV-2. Biopsy or autopsy material from the lungs, whole blood, serum, and feces can be used as additional material for examination [25,26].

Serological methods for detecting antibodies to SARS-CoV-2 are of secondary importance for diagnosing current infection and of primary importance for assessing the immune response to current or past infection. SARS-CoV-2 antibodies are detected using an ELISA test to determine immunoglobulins of classes A, M, and G (IgA, IgM, and IgG) to SARS-CoV-2 (including the receptor-binding domain of the surface glycoprotein S). Class A antibodies (IgA) begin to form and are detectable approximately 2 days after the onset of the disease, peak after 2 weeks, and persist for a long time. Class M (IgM) antibodies begin to appear approximately 7 days after the onset of infection, peak after a week, and can persist for 2 months or more. Class G (IgG) antibodies appear approximately 3 weeks or earlier and are specific to SARS-CoV-2. A distinctive feature of the humoral response to infection is the short time interval between the appearance of IgM and IgG antibodies, and sometimes their simultaneous formation. For the indirect diagnosis of COVID-19, it is recommended to conduct separate testing for IgM/IgA and IgG antibodies, as well as to monitor the appearance of antibodies over time (seroconversion detection) by retesting after 5–7 days [25,26].

Conclusions. Thus, the pathogenesis of COVID-19 coronavirus disease proceeds with primary lung damage and late hematological and tissue hypoxemia and mitochondrial dysfunction due to oxidative stress. Research into the pathogenesis of COVID-19 is being conducted widely around the world, but the results obtained are often contradictory and preliminary. It is not yet clear why some patients respond too weakly to SARS-CoV-2, while others respond too strongly. It is unclear whether immune responses are beneficial or harmful in this disease. An increase in cytokine levels probably plays a role in the development of severe lesions in some patients. The multi-organ failure that occurs in COVID-19 is not caused by sepsis, which is uncommon in coronavirus disease, but by the universal damage to blood vessels in organs caused by SARS-CoV-2. In any case, research into the pathogenesis of coronavirus disease and methods for its diagnosis remains a pressing issue at the current stage of medical science development.

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