THE COURSE OF CORONAVIRUS AGAINST THE BACKGROUND OF CHRONIC HEPATITIS

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Annotation: *The liver is a vital organ. The liver has many functions in the human body. Participates in the reactions of the immune system. In addition, it determines the overall level of resistance to infections and is responsible for the production of antibodies to fight viruses and bacteria. Liver diseases are the most common in the world. Especially in the context of the COVID-19 pandemic. World Health Organization February 11, 2020*

Key words: *COVID, chronic hepatitis, coronovirus, clinical course.*

Relevance. News by the end of 2019 In the People's Republic of China, an unknown pneumonia caused universal attention. Pneumonia caused by the new coronavirus SARS-CoV-2 severe acute respiratory syndrome coronavirus is a coronavirus of severe acute respiratory syndrome. On February 11, 2020, the World Health Organization officially assigned the name SARS-CoV-2 infection, COVID-19 "CoronaVirus Disease 2019" - a disease caused by the new coronavirus 2019). Pneumonia is the leading clinical form of COVID-19. Nevertheless, as the COVID-19 pandemic spread and the analysis of clinical data, patients began to identify symptoms that are not characteristic of "abnormal" pneumonia. Neurological manifestations, skin changes, eye lesions, etc. are described. The extrapulmonary presence of SARS-CoV-2 was also detected in cholangiocytes. Among the possible factors of liver damage, the virus-induced effect, systemic inflammation ("cytokine storm"), hypoxia, hypovolemia, hypotension in shock, drug hepatotoxicity, etc. are considered. In 14-53% of patients with COVID-19, changes in biochemical parameters may be registered, which, as a rule, do not require medical correction. Acute hepatitis develops very rarely.

However, special attention should be paid to patients with COVID-19 belonging to the risk group — those who have undergone liver transplantation, receiving immunosuppressants, as well as in cases of decompensation of cirrhosis, the development of acute liver failure against the background of chronic, hepatocellular carcinoma, during antiviral therapy. We need constant exchange and open access to scientific data, new technologies, updated guidelines.

Coronaviruses are widespread in nature and are the causes of various colds (up to 25%). Most of them cause a viral infection that does not cause serious harm to health, but some, such as SARS-CoV severe acute respiratory syndrome coronavirus — severe acute

respiratory syndrome coronavirus and MERS-CoV (Middle East respiratory syndrome coronavirus-Middle East respiratory syndrome coronavirus) lead to the development of severe respiratory syndrome with high mortality.

In nature, many species of bats serve as the natural host for coronaviruses. Evolving due to mutations, preadaptation processes, they periodically cause epidemics in human populations. Thus, the outbreak of an unknown pneumonia that began at the end of December 2019 in China caused the development of a public health emergency, which subsequently led to a pandemic caused by the new coronavirus SARS-CoV-2 (severe acute respiratory syndrome coronavirus - severe acute respiratory syndrome coronavirus). On February 11, 2020, the World Health Organization (WHO) assigned the official name of SARS-CoV-2 infection, COVID-19 ("CoronaVirus Disease 2019" - a disease caused by the new coronavirus 2019).

The mortality rate from this infection is 0.5-3%. SARS-CoV-2 and possible mechanisms of COVID-19 pathogenesis.The new coronavirus is a single-stranded RNA-containing virus belonging to the Coronaviridae family, the genus Betacoronavirus. SARS-CoV-2 is a zoonotic virus, which follows from phylogenetic analysis, which showed the closest relationship with the isolate of the SARS-like bat coronavirus BM48-31/BGR/2008 (identity — 96%). Apparently, bats are the reservoir of SARS-CoV-2, and other small mammals in particular, pangolins are intermediate hosts, possibly infecting the "patient zero". In addition, the phylogenetic analysis of SARS-CoV-2 obtained data indicating 88% identity of sequences with SARS-CoV and about 50% with MERS-CoV. The structure of respiratory syndrome coronaviruses is very similar. Among the structural proteins of SARS-CoV-2, S-proteins or "protein spikes" (from the English Spike — spike), membrane protein, protein and nucleocapsid are distinguished. Protein S plays an important role in the attachment, fusion and penetration of the virus into cells, which allows it to be considered as a possible target for the production of antibodies and vaccines. The pathogenesis of the new coronavirus infection has not been sufficiently studied. The key virulence factor is the interaction of the receptor-binding domain (receptor—binding domain - RBD) of protein S located on the outer membrane of SARS-CoV-2 with the receptor of angiotensin-converting enzyme 2 (angiotensin—converting enzyme 2 receptors - ACE2) activated by transmembrane serine proteases (TMPRSS2 -Transmembrane protease, serine 2) of humans.

ACE2 is expressed in a surfactant secreted by type II alveolocytes from blood plasma components. Surfactant is a surface-active monomolecular film, which is located at the interface of the air—liquid phases in the alveoli, alveolar passages and respiratory bronchioles of the 1st-3rd order and prevents the collapse (adhesion) of the walls of the alveoli during breathing. ACE 2 expression protects against lung damage, but it decreases due to its binding to the spike protein SARS-CoV, which increases the risk of infection. At the same time, it was shown in the experiment that an increase in the expression of ACE2

does not exclude the possibility of an increase in binding to SARS-CoV. Up to three viruses can attach to one target.

ACE 2 and TMPRSS2 are unevenly distributed among patients of European and Asian origin, which can also affect the intensity of infection. It has been suggested that non-structural SARS-CoV proteins are capable of modifying the structure of hemoglobin in the erythrocyte, which leads to a violation of oxygen transport, causes iron dissociation, porphyrin formation, and an increase in ferritin. Such exposure can lead to increased inflammatory processes in the lungs, oxidative stress, hypoxemia, hypoxia, the development of symptoms of acute respiratory distress syndrome ARDS and multiple organ oxygen deficiency. However, the basis for this hypothesis was the creation of a biotransformation model without conducting experimental and clinical studies of SARS-CoV-2 tropen to goblet cells contained in the mucous membrane of the respiratory tract, intestine, conjunctiva of the eyes, ducts of the pancreas and parotid salivary glands. Active replication of the virus significantly reduces the protective functions of goblet cells (mucus formation), which also contributes to the penetration of the virus into the human body. In response to the spread of the coronavirus, the development of a hyperimmune reaction is observed — the so-called "cytokine storm", characterized by the synthesis of a significant (abnormal) amount of pro-inflammatory interleukins (IL-1ß, IL-6, tumor necrosis factor, etc.) and chemokines with a simultaneous decrease in the content of T-lymphocytes in the blood. In addition, SARS-CoV-2, infecting the endothelium of blood vessels, interacts with the ACE2 located there and leads to the development of endothelial dysfunction, hyper permeability, microcirculation disorders, the development of vascular thrombophilia and thrombosis.

The progression of COVID-19 is determined by diffuse alveolar damage with the formation of hyaline membranes, the development of pulmonary edema. The histological picture of the lungs during autopsy is characterized by the organization of alveolar exudates and interstitial fibrosis, the formation of hyaline membranes, the presence of interstitial mononuclear inflammatory infiltrates, numerous fibrin microthrombs, pronounced edema, hyperplasia and focal desquamation of type II alveolocytes, a significant content of macrophages with viral inclusions in the alveolar exudate. Hemorrhage, necrosis, hemorrhagic infarction are observed in more affected areas. Clinical picture and diagnosis of COVID-19 infection with SARS-CoV-2 in humans occurs in the last days of the incubation period and as much as possible in the first three days from the onset of the disease. In the vast majority, infection occurs due to contact with a COVID-19 patient in cases of clinically manifested disease (up to 75-85% — in contact with infected relatives from the family environment). It should be emphasized that the release of the virus, as a rule, lasts up to 12 days in mild /moderate cases and more than 14 days in severe cases. However, in patients who have recovered from COVID-19, the RNA of the new coronavirus can be positive even after the disappearance of clinical symptoms. A patient with COVID-19 can infect 3-5 people around him, and a person with the flu can

infect only 1-2 people. Epidemiological data indicate that patients with cardiovascular diseases, arterial hypertension, diabetes mellitus, and malignant tumors are the most susceptible to SARS-CoV-2.

The incubation period is 2-14 days, the average period is 5-6 days. SARS-CoV-2 is transmitted by airborne droplets when coughing, sneezing, talking, airborne dust with dust particles in the air, contact through handshakes, household items and fecal-oral routes. The identified adverse signs require monitoring of liver function and electrocardiography, especially among risk groups — patients with CKD and myocardial repolarization disorders. The scale in the use of experimental treatments for COVID-19 is unprecedented. However, evidence of their effectiveness has yet to be established. In this regard, the issue of drug interaction remains very important for clinical practice. The website of the University of Liverpool presents the main experimental drugs that are currently used in COVID-19 therapy with an indication of their mechanisms of action; an assessment of joint use with other means is given, taking into account the risks and benefits, duration of use, patient's condition, taking medications for previously established diseases.

Conclusion: More than 3.5 million cases of COVID-19 have been reported worldwide, resulting in more than 250,000 deaths. Unfortunately, there are currently no effective specific treatments for COVID-19. Numerous clinical randomized trials of various drugs are being conducted. There is no evidence yet that patients who have recovered from COVID-19 are protected from re-infection. It is necessary to monitor individuals with antibodies against SARS-CoV-2 in comparison with individuals without them with an assessment of the frequency of infection with SARS-CoV-2 and the development of COVID-19 over a long period (at least one year).

However, the first experimental experience of using plasma containing class G immunoglobulin antibodies in patients who have undergone COVID-19 has shown encouraging results. Recovered patients from COVID-19 and people with an asymptomatic course who secrete the virus with feces can be considered as a possible source of infection. In addition, since SARS-CoV-2 RNA was detected in wastewater samples, the question of the viability of the virus in an environment through which a fecal-oral transmission pathway can also be implemented remains unresolved.

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