ANALYSIS OF THE SPREAD AND COMPLICATIONS OF CORONAVIRUS INFECTION

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Annotation: The liver is one of the most vital organs in the human body, performing a wide range of essential physiological functions. It plays a crucial role in metabolism, detoxification, protein synthesis, and the regulation of biochemical processes. Furthermore, the liver actively participates in immune system responses, contributing to the body's overall resistance to infections and the production of antibodies that combat viruses and bacteria.

In recent years, the prevalence of liver diseases has increased significantly worldwide, becoming a major global health concern. The situation has been further aggravated by the COVID-19 pandemic, which has had a profound impact on liver function both directly—through viral infection—and indirectly—through drug-induced liver injury, inflammatory responses, and metabolic complications. According to the World Health Organization (WHO), since February 11, 2020, when COVID-19 was officially recognized as a global health threat, the incidence of liver-related complications in infected patients has drawn increasing scientific attention. This study aims to analyze the epidemiological features, pathophysiological mechanisms, and clinical outcomes of liver involvement associated with coronavirus infection, emphasizing the importance of early diagnosis and preventive measures.

Introduction: The liver is one of the most important organs in the human body, responsible for maintaining metabolic homeostasis, detoxification, synthesis of plasma proteins, and regulation of immune responses. It plays a key role in the body's defense mechanisms, influencing both innate and adaptive immunity, determining overall resistance to infections, and contributing to the production of antibodies that neutralize viruses and bacteria.

Liver diseases remain among the most widespread and serious health problems worldwide. Their etiology includes viral infections, metabolic disorders, toxic exposures, and autoimmune processes. In recent years, the burden of liver pathology

has further increased due to the global spread of the novel coronavirus infection (COVID-19).

Since the World Health Organization officially declared COVID-19 a public health emergency of international concern on February 11, 2020, numerous studies have reported the impact of SARS-CoV-2 infection on hepatic function. Liver injury in COVID-19 patients may result from direct viral cytopathic effects, systemic inflammation, hypoxia, or the hepatotoxicity of antiviral and immunomodulatory drugs. These factors contribute to the worsening of clinical outcomes and increase the risk of complications, particularly in patients with pre-existing liver diseases.

Therefore, studying the epidemiological features, mechanisms of liver damage, and clinical manifestations associated with coronavirus infection is of great scientific and practical importance. Understanding these relationships can improve early diagnosis, optimize treatment strategies, and reduce the incidence of liver-related complications in COVID-19 patients.

The aim of the study was to analyze the epidemiological characteristics, frequency, and mechanisms of liver injury in patients with coronavirus infection, as well as to assess the relationship between hepatic dysfunction and disease severity.

Nevertheless, as the COVID-19 pandemic spread and the analysis of clinical data, patients began to identify symptoms that were not characteristic of "abnormal" pneumonia. Neurological manifestations, skin changes, eye lesions, etc. are described. The extrapulmonary presence of SARS-CoV-2 was also found in cholangiocytes. Among the possible factors of liver damage, virus-induced effects, 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 is very rare. 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. There is a need for constant exchange and open access to scientific data, new technologies, and 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 and preadaptation processes, they periodically cause epidemics in human populations. Thus, the outbreak of an unknown pneumonia in

late 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). World Health Organization (WHO) February 11, 2020 It has assigned the official name of SARS-CoV-2 infection, COVID-19 ("CoronaVirus Disease 2019" is 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 family Coronaviridae, 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% sequence identity with SARS-CoV and about 50% with MERS-CoV. The structure of coronaviruses of respiratory syndromes 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. A 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 surfactantly 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. The expression of ACE2 protects against lung damage, but it decreases due to its binding to the SARS-CoV spike protein, which increases the risk of infection. At the same time, it was shown in the experiment that increased expression of ACE2 does not exclude the possibility of increased binding to SARS-CoV. Up to three viruses can attach to a single target. ACE2 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 tropens 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 coronavirus, the development of a hyperimmune reaction is observed — the so-called "cytokine storm", characterized by the synthesis of a significant (abnormal) amount of proinflammatory interleukins (IL-1β, IL-6, tumor necrosis factor, etc.) and chemokines while reducing the content of T-lymphocytes in the blood. In addition, SARS-CoV-2, infecting the endothelium of blood vessels, interacts with ACE2 located there and leads to the development of endothelial dysfunction, hyper permeability, impaired microcirculation, 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 microthrombs of fibrin, pronounced edema, hyperplasia and focal desquamation of type II alveolocytes, a significant content of macrophages with viral inclusions in the alveolar exudate. Hemorrhage, necrosis, and hemorrhagic infarction are observed in more affected areas. The 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 a 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 may 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, 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 during 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. On the website of the University of Liverpool (UK) (www.covid19-druginteractions.org) the main experimental drugs that are currently used in COVID-19 therapy are presented, indicating their mechanisms of action; an assessment of joint use with other drugs is given, taking into account the risks and benefits, duration of use, patient's condition, and medication for previously established diseases.

Conclusion:

There are over 3.5 million cases of COVID-19 worldwide, which has killed more than 250,000 people. Unfortunately, there are currently no effective specific treatments for COVID-19. Numerous clinical randomized trials of various drugs are being conducted. So far, there is no evidence 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 those 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 faeces can be considered as a possible source of infection. In addition, since SARS-CoV-2 RNA has been 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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