COVID-19: PATHOGENESIS IN THE IMMUNE, METABOLIC, CARDIOVASCULAR SYSTEM

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Annotation. This article analyzes the pathogenesis of the disease, physiological mechanisms and, of course, the post-COVID-19 recovery period. Analyzed quantitative changes in metabolism in the body, the dynamics of clinical signs. The research used more than 50 articles from the leading database, the information obtained from them is clearly explained.

The new coronavirus seriously affects the lungs not only during COVID-19 illness: a partial decrease in their function also is observed in recovered patients. Examining people who have recovered from the coronavirus, Chinese doctors found in their lungs the so-called ground glass opacity symptom: a decrease in the transparency of the lung tissue, which indicates irreversible damage.

COVID-19 and cardiovascular system

The relationship between acute respiratory tract infections and cardiovascular disease is a well-established fact; acute respiratory viral infection, influenza, respiratory syncytial infection, bacterial pneumonia are triggers of cardiovascular diseases, and the initial pathology of the cardiovascular system increases the likelihood of the development and progression of the infectious process. The demonstration of this fact is reflected within the current COVID-19 pandemic: a significant proportion of coronavirus-affected patients are reported to have cardiovascular disease.

So far, it has not been possible to link together the sequence of events occurring in the body under the influence of a new coronavirus infection, including for CCC. The course of COVID-19 varies from asymptomatic carriers to severe acute

respiratory syndrome (SARS, or SARS) [1]. The problem is exacerbated by the lack of reliable data on the role of the additional furin cleavage site. SARS-CoV-2 shares many similarities with the genome of SARS-CoV and other SARS-like pathogens. Therefore, the analysis of previous pandemics and the slowly growing base of the latest information allow us to make assumptions about the pathological physiology of COVID-19 in humans. Now, several pathophysiological mechanisms of the impact of COVID-19 on CVS are presented in the literature. Additionally, the effects of drug therapy taken by patients both for the treatment of the coronavirus infection itself and for concomitant diseases are highlighted.

Due to the immune response, high levels of chemokines are synthesized to attract effector inflammatory cells. This inappropriate immune response with the secretion of inflammatory chemokines leads to lung infiltration and hyper activation of monocytes and macrophages producing pro-inflammatory cytokines (IL-6, IL-8 and IL-1β, as well as TNFα) and chemokines (such as CCL2, IFNγ-induced protein-10 and CCL3). Increased local production of cytokines and chemokines attracts neutrophils that are more inflammatory and monocytes to lung tissue, causing oedema and reduced gas exchange in the alveoli [2].

Lung injuries described at autopsy lead to the development of hypoxemia. When arterial oxygen saturation (PaO2) falls below 40 mmHg, dyspnoea occurs. It should be noted that the normal response to hypoxemia is an increase in tidal volume and respiratory rate. However, during the COVID-19 pandemic, there have been increasing reports of patients who did not experience this compensatory response despite critically low saturation levels. In the English-language literature, this phenomenon is called “happy hypoxemia” [3].

Although the exact mechanisms of cardiovascular complications (CVD) in COVID-19 are still to be elucidated and systematized, the literature describes the predominant influence of the following processes:

1) direct cardiotoxicity;

2) systemic inflammation;

3) discrepancy between myocardial oxygen demand and its delivery;

4) plaque rupture and coronary thrombosis;

5) side effects of therapy during hospitalization;

6) sepsis leading to the development of disseminated intravascular coagulation syndrome;

7) increased systemic thrombosis;

8) imbalance of electrolytes.

According to statistics, the main cause of myocardial damage is direct viral damage to cardiomyocytes and the effects of systemic inflammation [4]. From a clinical perspective, monitoring of cardiac markers such as troponin, N-terminal natriuretic peptide B, and creatine kinase may help identify patients at risk for cardiovascular complications at an earlier stage. This factor can be useful for preventive purposes and provide timely pathogenic treatment [5].

One of the leading causes of COVID-19 deaths in cardiac patients is myocarditis. Initially, patients complain of chest pain, shortness of breath, later dysrhythmia is detected. Myocarditis was indicated by autopsy data of some patients, which revealed infiltration with mononuclear antibodies and traces of the genome of the virus itself in the myocardium [6, 7]. The long-term effects of myocarditis associated with SARS-CoV-2 are unknown. It has been suggested that subclinical myocarditis may be a risk factor for sudden cardiac death during moderate to vigorous physical activity [8].

The COVID-19 pandemic clearly demonstrates that outbreaks of new viral infections continue to be a pressing global health problem. Taking into account the spread of SARS-CoV-2 and the large number of infected, as well as the lack of pathogenic therapy, at present, the forces of all specialists should be thrown into coordinated joint work on prevention, diagnosis, development of new methods of treatment and the creation of vaccines against COVID-19 [9].

Type 2 diabetes mellitus and arterial hypertension are the most common and unfavourable comorbidities in patients with coronavirus infections. Basic research indicates a direct relationship between the pathogenesis of the disease and the most important metabolic and endocrine processes. Thus, new tasks have been set for

endocrinologists - to inform patients with endocrinopathies, primarily with diabetes, as quickly as possible about the risks that a pandemic can potentially pose to their health, about infection prevention methods, and about the tactics of managing people with endocrinopathies in case of illness [10].

Damage to the cardiovascular system in COVID-19 is multifactorial; it occurs both because of the direct effect of the virus on the elements of this system, and indirectly. The cardiovascular system in patients with comorbidity, regardless of age, is more prone to myocardial damage and the development of complications with a high risk of death. Cardiovascular status should be assessed in patients with suspected or confirmed COVID-19 who have underlying cardiovascular disease and/or risk factors; cardiovascular symptoms/signs; changes in the level of biomarkers.

Now, it is impossible to state unequivocally whether a full recovery of the cardiovascular system after COVID-19 is possible, and when the functional recovery of its elements will occur after the disease. Long-term studies and observation of patients will allow the development of preventive measures and tactics for the treatment of damage to the cardiovascular system in COVID-19.

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