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FEATURES OF THE COURSE OF CORONAVIRUS INFECTION IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AGAINST THE BACKGROUNDOF CARDIOVASCULAR PATHOLOGY.

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Summary: COVID-19, like other respiratory viruses, has extrapulmonary manifestations. In particular, exposure to the virus leads to decompensation of diabetes and damage to the cardiovascular system(CVS), the pathophysiological mechanisms of which are not fully understood; a full understanding of the mechanisms of interaction between COVID-19 and CVS is not formed. The article analyzes the current understanding of COVID-19, examines possible pathogenesis links, and attempts to systematize the pathophysiological mechanisms of CVD damage in patients with type 2 diabetes and their complications.
Key words: COVID-19, SARS-CoV-2, diabetes mellitus, cardiovascular system, cardiovascular diseases, angiotensin-converting enzyme 2 (ACE2).

DM- diabetes mellitus AG - arterial hypertension ATII - Angiotensin II ACE2 — angiotensin-converting enzyme 2 CHD — coronary heart disease IM — myocardial infarction KMP — cardiomyopathy ACS — acute coronary syndrome CVD- cardiovascular disease MTR — cardiovascular complications CVC — cardiovascular complications	CVE — cardiovascular events CVS — cardiovascular system CoV — coronavirus genome E- protein shell HCoV — human coronavirus IL — interleukin M — protein membrane N — protein nucleocapsid S —protein spike TNF — a tumor necrosis factor tPA — tissue activator of plasminogen

Introduction: The relationship between acute respiratory tract infections and cardiovascular diseases (CVD) is a generally recognized fact; acute respiratory viral infection, influenza, respiratory syncytial infection — bacterial pneumonia are triggers of CVD, and the initial pathology of the endocrine and cardiovascular systems (CVD) increases the likelihood of the development and progression of the infectious process [1]. The demonstration of this fact is reflected in the current COVID-19 pandemic: a significant proportion of patients affected by coronavirus are reported to have type 2 diabetes and CVD [2]. Hyperglycemia and diabetes are independent predictors of mortality and morbidity in patients with COVID-19.(Yang JK, et al. Diabetes Med. 2006; 23:623-628.) 78.59% of patients with diabetes mellitus had concomitant diseases, including 53.06% -cardiovascular diseases. High blood glucose levels increase the risk of developing severe pneumonia. (Joy E, et al. Abstract GP77. Presented at: European Congress of Endocrinology; May 19-22, 2018; Barcelona, Spain). Since viral infection can

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cause sharp fluctuations in blood glucose levels in diabetic patients, which negatively affects patients ' recovery, there is reason to believe that diabetes combined with pneumonia during SARS-CoV-2 has an unfavorable prognosis. In this regard, there is a need for a fundamental study of the pathophysiological changes occurring in the CVS as a result of the potential effects of coronavirus, which, however, presents a number of difficulties. However, without understanding these processes, it is impossible to predict the development of certain cardiovascular events (CVD), and, as a result, choose the right treatment strategy. In this context, the actual data obtained, as well as the analysis of previous coronavirus pandemics, are informativeкоронавируса. For endocrinologists , For cardiologists, this issue is particularly acute, since CVD damage in patients with type 2 diabetes is the second leading cause of mortality in COVID-19 and the long-term consequences of heart and vascular damage are unknown.

Biological features of SARS-CoV-2

The structure of SARS-CoV-2 is a nucleocapsid, surrounded by a protein membrane and a lipid-containing outer shell, from which club-shaped spiny processes extend. Externally, these formations resemble a crown, for which the family got its name [3].

These processes are represented by one of the 4 structural proteins of the coronavirus — the spike protein (S), which provides the process of attachment of the virus to the host cell receptor, as well as further fusion with the cell membrane. Other structural proteins are nucleocapsid protein (N), membrane protein (M), and envelope protein (E).In a living organism, the coronavirus has the greatest affinity for the integral protein of the plasma membrane of angiotensin-converting enzyme 2 (ACE2) [4].

Systemic inflammatory response

Systemic inflammatory shifts and immune disorders characteristic of DM can play an important role, including suppression of neutrophil chemotaxis and T-lymphocyte-mediated immune response, impaired cytokine production with acceleration of the inflammatory cascade, and reduced elimination of microorganisms [8-9], including SARS-CoV-2 [10]. The presence of obesity in many patients with diabetes contributes to the maintenance of systemic inflammation: excess adipose tissue, on the one hand, additionally stimulates it due to increased production of pro-inflammatory cytokines, adipokines and chemokines, and, on the other hand, is associated with vitamin D deficiency, which is also an immunomodulator and inhibits excessive production of inflammatory mediators (both mechanisms can be associated with the development of increase the severity of COVID-19) [11]. Another potential point of intersection of pathogenetic pathways in DM and COVID-19 is the relationship with the expression of angiotensin-converting enzyme 2 (ACE 2). ACE2 inhibition may be one of the factors of lung damage, as well as the cause of systemic inflammation with the release of cytokines, which contributes to the development of acute respiratory distress syndrome and multiple organ dysfunction [5]. Reduced ACE-2 levels lead to increased blood levels of angiotensin II, mediating pulmonary vasoconstriction. Liu et al. (2020) [6] showed that elevated serum angiotensin II (ATII) levels were significantly associated with high viral load and more severe lung damage in COVID-19. 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 hyperactivation of monocytes and macrophages that produce pro-inflammatory cytokines (IL-6, IL-8, and IL-1b, as well as TNF a) and chemokines (such as CCL2, IFN y-induced protein-10, and CCL3). High levels of circulating complement proteins such as C3, C3a, C4, and C5a

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have been associated with an increased risk of SSR. It is assumed that острых CVE the complement system contributes to both thrombosis and ischemic reperfusion injury after restoration of blood flow in the ischemic tissue in acute SSSs [7]. Histopathological studies have confirmed direct viral infection of endothelial cells, endotheliitis (inflammation of the blood vessel wall), and microvascularo- and macrovascular thrombosis in both venous and arterial blood flow [14]. Based on these data, it can be concluded that SARS-CoV-2 promotes the induction of endotheliitis in various organs, which is both a direct consequence of viral damage and a secondary inflammatory response of the body to infection. COVID-19 - associated endothelitis It may explain the systemic impairment of microcirculatory function in various vascular beds and their clinical consequences in patients with COVID-19 [15].

Thromboembolic complications

Patients with COVID-19 are at an increased risk of developing thrombotic events. This is due to systemic inflammation, multiple disorders of the hemostatic system and multiple organ involvement and directly depends on the severity of the disease. A number of studies in patients with COVID-19 pneumonia have shown a significant increase in blood levels of the D-dimer. D-dimer levels greater than 1 microgram/ml were found to be associated with an increased risk of death during hospitalization. It is assumed that anticoagulation with low-molecular-weight heparin is associated with a 6-fold increase in survival in patients with an increase in serum D-dimer, as well as in patients with severe COVID-19 [9, 10].

Type 2 diabetes and COVID-19

Currently, according to a few studies, it has been determined that the prognosis of those infected with COVID-19 is significantly affected by the presence of DM2, namely, uncontrolled levels of hyperglycemia. An increase in blood glucose is associated with a massive release of contrainsular hormones and a violation of the immune response in the context of the development of COVID-19 infection [11, 12]. Specific damage to the microcirculatory bed, reduced gas exchange in the lungs, and chronic tissue hypoxia aggravate the development of infectious pneumonia [13]. Hyperglycemia can also increase the replication of respiratory viruses in lung cells. It is known that inDM2, an imbalance in the hemostatic system is formed, which is manifested by an increase in coagulation activity and a decrease in fibrinolysis, contributing to an increased risk of life-threatening complications of COVID-19 [14]. Another aspect of this problem is the possibility of the onset (or detection) of DM against the background of the course of COVID-19. It is known that pancreatic islets are very vulnerable to coronaviruses due to the pronounced expression of ACE type 2 molecules. Another provoking factor may be the use of hormone therapy in severe cases of COVID-19 [3].

Cardiovascular complications of COVID-19

SARS-CoV-2, which, based on the supposed pathophysiological mechanisms of its action, leads to the development of events such as myocarditis, pericarditis, acute coronary syndrome (ACS), decompensation of heart failure (HF), sudden cardiac death, cardiomyopathy (CMT), arrhythmias, cardiogenic shock and venous, arterial thromboembolic complications. According to the results of one study, 7% of patients in a cohort of 150 people revealed irreversible myocardial damage and developed HF; these conditions were accompanied by an increased level of troponin in the blood [16]. Although the exact mechanisms of the development of cardiovascular complications (CVD) in COVID-19 still need to be clarified and systematized.

Conclusion

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Literature data indicate a high incidence of comorbidity in patients with COVID-19. Among CVD, such cases as hypertension and CHD are most often registered, and the prevalence of type 2 diabetes is also high2. Patients with COVID-19 and cardiovascular comorbidity are highly likely to develop complications and death. The results of clinical observations also indicate the difficulties of treating these patients. It is advisable to further study the features of screening, diagnosis, clinical manifestations and course of diseases in patients with COVID-19 and concomitant pathology, which will help determine the risk factors for complications and develop new optimal approaches to their treatment and prevention.

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