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COURSE OF COVID-19 IN PATIENTS WITH DIABETES MELLITUS

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Abstract:
The article discusses the mechanisms of the relationship between coronavirus infection and DM, outlines the consequences of their mutual influence. The article also touches upon the issues of personalized choice of therapy in patients with various forms of COVID-19. Timely control and maintenance of glycemic levels within the target range defined for each individual patient underlie the successful prevention of COVID-19 and its treatment in case of infection. The severity of the disease determines the tactics of treatment and the choice of hypoglycemic therapy.

Keywords: COVID-19, diabetes, infection, coronavirus.

Introduction

The COVID-19 infectious epidemic caused by the novel coronavirus , the severe acute respiratory syndrome coronavirus 2 (Severity Acute Respiratory Syndrome Coronavirus 2, SARS-CoV-2), began its disastrous procession in December 2019 in Wuhan (China) and spread with lightning speed to almost all countries of the world. As of May 12, 2020, according to the World Health Organization, there were 4,058,252 confirmed cases of COVID-19, including 281,736 deaths.

The severity of this epidemic is largely due to the widespread (epidemic) prevalence of diabetes mellitus (DM) and obesity, the presence of which exacerbates the course of infection. Data from the first months of 2020 show that the majority of people with

severe COVID-19 have comorbidities, the most common of which are diabetes, obesity, cardiovascular disease, and hypertension.

In the context of today's pandemic , evidence has accumulated, on the one hand, indicating the association of obesity and diabetes with a more severe course of COVID-19 and death, on the other hand, that COVID-19 is associated with the development of hyperglycemia, especially in older people with diabetes. 2nd type.

To understand the relationship between this infection and DM, some structural features of the virus and its interaction with the cells of the human body are important. Coronavirus 2 refers to RNA-3-containing viruses that have an envelope, the structure of which contains several elements (glycoproteins) that ensure the structural integrity of the virion and its penetration into the target cell. The virus uses a special surface glycoprotein called a "spike" to bind to angiotensin converting enzyme 2 (ACE2) and enter the cell. ACE 2 as an element of the renin- angiotensin - aldosterone system (RAAS) was identified at the beginning of the 21st century. Its long and well-studied homologue, angiotensin converting enzyme, is a positive regulator of RAAS activity, providing the conversion of angiotensin I (Ang I) to angiotensin II (Ang II) by removing the dipeptide from the C-terminal end of the decapeptide EngI. Ang II is a profibrogenic, vasoconstrictor, pro- inflammatory peptide that binds and activates the angiotensin type 1 receptor (AT1R). ACE 2 has shown itself to be a negative regulator of RAAS activity, since, by cleaving the AngII residue elsewhere, it produces angiotensin, a heptapeptide that has a powerful vasodilating, anti-inflammatory, and antioxidant effect. It can also break down Ang I, producing angiotensin, which has similar properties.

In the experiment, genetic or pharmacological modeling of low expression of ACE 2 was accompanied by the development of impaired glucose tolerance and a decrease in the first phase of insulin secretion. Decreased expression of ACE 2 has been noted in animal models DM and in kidney biopsies of patients with DM and was associated with increased albuminuria and the presence of morphological signs of diabetic nephropathy in biopsies. It is believed that ACE 2 deficiency plays an important role both in the development of DM and in the progression of its renal and cardiac complications.

ACE 2 and changes in its expression play an important role in the mechanisms of interaction between DM and COVID-19. It is extremely important that the virus, by binding to ACE2, causes its down -regulation, which can give the following explanation for severe lung damage in DM and heart disease. vascular diseases — these patients initially have low ACE2 expression due to type 2 DM, and its further decrease under the influence of the virus leads to a severe deficiency of Ang1-7 and 1-

9 and a pronounced dominance of the effects of AngII in the lung tissue, which determines severe lung damage.

The lung is the most vulnerable target organ, firstly, because the vast surface area of the lungs makes them highly susceptible to inhaled viruses. 83% of ACE2- expressing cells were type II alveolar epithelial cells, and these cells can presumably serve as a reservoir for viral invasion. High expression of the ACE2 receptor, in addition to the lungs, has also been found in many extrapulmonary tissues, including the pancreas and kidneys already mentioned above, as well as the heart, endothelium, and intestines.

Acute hyperglycemia has been shown to increase the expression of ACE2 in cells, which may facilitate entry of viral cells. However, chronic hyperglycemia, as already noted, reduces the expression of ACE2, making cells vulnerable to the inflammatory and damaging effects of the virus. The interaction between COVID-19 and DM may be bidirectional.

These data suggest that infection may cause the development of DM or at least severe stress hyperglycemia. The fact that COVID-19 infection causes hyperglycemia in people without pre-existing diabetes has already been documented by some researchers. Hyperglycemia has been shown to persist for 3 years after recovery from SARS, indicating transient damage to beta cells. Today we cannot yet talk about the consequences of exposure to SARS-CoV-2, but in the near future the long-term consequences of this infection will certainly be assessed.

Particular attention should be paid to patients with type 1 diabetes with glycated hemoglobin levels above the target values due to the high risk of metabolic decompensation. Diabetic ketoacidosis (DKA) results from insulin deficiency and increased counter-regulatory responses that promote ketone production. In fact, any severe infection increases the risk of developing acute complications of diabetes with a high frequency. Infection is a causative factor in 30–60% of patients with a hyperglycemic hyperosmolar state and in 15–58% of patients with DKA.

Another group of drugs with probably positive effects on the course of COVID-19 are α - glycosidase inhibitors. These drugs attracted attention during the dengue fever epidemic, caused by a virus similar in structure to SARS-CoV-2 (DENV-1). These viruses, like SARS-CoV-2, have prM and E glycoproteins in their envelope structure. α - glucosidase 1 is an enzyme that plays a crucial role in viral maturation by initiating the formation of viral envelope glycoprotein oligosaccharides (pr M and E). α - Glycosidase inhibitors inhibited the activity of SARS-CoV-2-like viruses (DENV-1) by interfering with the configuration of the prM and E structural proteins, which provide a critical step in virion secretion. However, these drugs, while demonstrating a

significant reduction in viral load in animal experiments, had a rather weak effect in humans. This may be due to the local action of these drugs in the gastrointestinal tract. Patients with reduced appetite, gastrointestinal symptoms may require a reduction in the dosage of antidiabetic drugs or a switch from tablet preparations to insulin.

Patients who develop stress hyperglycemia during the course of COVID-19 should receive intensive treatment during hospitalization (maintaining glycemic levels of 6.1-7.8 mmol / l), and later, after discharge, they need periodic monitoring glycemic levels, as they have a high risk of developing diabetes.

In May 2021, the World Health Assembly adopted a resolution to strengthen diabetes prevention and control. It calls for action to be taken, including in the area of expanding access to insulin; increasing the level of harmonization and harmonization of regulatory requirements for insulin, as well as other medicines and medical products used to treat diabetes; and assessing the feasibility and feasibility of establishing a network tool for the exchange of information related to transparency in the markets for antidiabetic drugs and medical products.

There is a certain feeling that nature has decided to join the fight against pandemics of obesity and diabetes in such an extraordinary way as the development of COVID-19, putting our patients before a harsh choice - either normalization of body weight and tight control of glycemia, or a high probability of dying from a viral infection, demonstrating the vulnerability and impotence of such patients in the face of a new infection that will always lie in wait for humanity.

Literature

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