FEATURES OF COVID-19 IN ENDOCRINE DISEASES

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ABSTRACT:
The SARS-CoV-2 turned out to be aggressive, and the number of diseases around the world continues to grow to the present. According to observational studies, people with chronic diseases are more a risk for severe COVID-19 disease. Endocrinologists are faced with new tasks–to inform persons with endocrinopathies as quickly as possible about the risks that their health could potentially cause, a pandemic; on infection prevention methods and management tactics for people with endocrinopathies in the face of a virus-caused disease.

Key words: SARS-CoV-2, COVID-19, Obesity, Diabetes, Oxidative Stress, Endocrinology.

I. INTRODUCTION
To date, three of the most dangerous coronaviruses are known– these are SARS-CoV, MERS-CoV, and SARS-CoV-2. The new coronavirus SARS-CoV-2 is a single-stranded RNA-containing virus, belongs to the family Coronaviridae, belongs to the Beta-CoV B lineage. It is assigned to the II group of pathogenicity, like some other representatives of this family; SARS-CoV2 is presumably a recombinant virus between the bat coronavirus and an unknown coronavirus by origin [1].

In the pathogenesis of ARDS due to COVID-19 infection, the main role is played by an excessive response of the immune system with a rapidly developing fatal cytokine storm. Studies have shown that mortality in COVID-19 is associated, among other things, with an increase in the level of interleukin-6 (IL6) [1,2].

Coronaviruses can provoke damage to the respiratory system, gastrointestinal tract, and nervous system. The incubation period lasts for 2-14 days, more often 3-7 days (there are no exact data at the moment) [3].

The second most common group of people with endocrinopathies is people with thyroid diseases. In this group, there are no additional risks or special rules for prevention or management in case of infection. For people with hypothyroidism, it is important to remember that compliance with the rules of thyroxine replacement therapy increases their resistance to any infection, and poor compensation for hypothyroidism can artificially reduce body temperature, preventing a realistic assessment of the severity of an infectious disease in a pandemic and thereby reducing the likelihood of adequate care. Interruption of thyroxine replacement is unacceptable and should continue in the hospital and intensive care unit (ICU). Patients who have recently started therapy with thyrostatics should not forget that these drugs have side effects (sore throat and diarrhea) with an increase in temperature and these phenomena formally resemble infection. Persons treated for highly differentiated thyroid cancer should also comply with all necessary preventive measures. Such patients may experience anxiety due to delays in the planned necessary dynamic examination, it is important to warn them that a delay of several months will not harm their health and is quite acceptable [2].

Persons with a violation of the parathyroid glands-hypoparathyroidism should carefully follow the recommended treatment regimen, and if there are complaints typical for decompensation of the disease, minimally increase the therapy taken. If symptoms persist, discuss further management by phone with your doctor. People with
hyperparathyroidism who have postponed the planned surgical treatment should reduce the intake of calcium with food, increase fluid intake. Under these conditions, postponing the operation for 1-2 months will not cause harm to health. If nausea occurs, it is possible to use herbal-based adaptogens [5].

In the event that patients have diabetes insipidus, the outpatient should carefully monitor the intake of drugs and the liquid consumed, and in case of severe course in the hospital, pay special attention to electrolyte metabolism and compensation for water-electrolyte losses due to fever and impaired consciousness, as well as monitor the diet [6-12].

Patients with primary or secondary adrenal insufficiency, when infection occurs, follow the same rules as in other situations with intercurrent diseases – increase the dose of glucocorticoids and always have injectable drugs in stock, which should be administered at the threat of a crisis (pressure drop, nausea, vomiting) [13,14].

Looking back at similar outbreaks of infections, during the H1N1 pandemic in 2009, obesity was recognized as an independent risk factor for influenza complications, so it is not surprising that obesity is also a potential independent risk factor for SARS-CoV-2. Obese patients are very prone to reduced airflow due to the limited expansion of the trunk, which makes it difficult to weaken the airflow (and increases susceptibility to poor breathing), oxygen consumption and respiratory potential can be seriously affected and predisposed to infection and the need for more oxygen support [15-17]. Finally, these patients present a serious problem for intubation (since additional adipose tissue on the larynx makes intubation more difficult) [18-20].

Severe acute respiratory syndrome coronavirus binds to the angiotensin converting enzyme 2 (ACE2) receptor for intracellular invasion, and it was found that the mechanism of acute lung damage during infection is mediated by activation of the renin-angiotensin system (RAS). RAS blockade has been proposed as a potential treatment for COVID-19. It is noteworthy that APF2 is expressed in human adipose tissue. The general activation of the RAS axis plays an important role in the pathophysiology of the risk of cardiovascular diseases associated with obesity and visceral obesity [21]. The interaction between the RAS system, adipose tissue and COVID-19 can only partially explain the higher risk of morbidity and mortality for obese COVID-19 patients [22-24].

Human dipeptidyl peptidase 4 (DPP4) has also been identified as a functional receptor for the spike protein of the Middle East respiratory syndrome MERS-CoV. It binds to the receptor-binding domain and interacts with T cells and nuclear factors involved in the pathogenesis of inflammatory diseases. DPP4, a transmembrane protein, has been identified in human adipose tissue and is associated with type 2 diabetes associated with obesity. Inhibition of DPP4 increases the secretion of glucagon-like peptide-1, which leads to an improvement in insulin sensitivity and glucose metabolism in adipocytes. Inhibition of DPP4 may also play a role in the immune response to COVID-19, reducing inflammation. Inhibition of the enzymatic activity of DPP4 suppresses the proliferation of T cells and the secretion of pro-inflammatory cytokines, such as interleukin IL-6 and IL-10 [25].

Recent reports have shown an increase in the inflammatory environment, which leads to a cytokine storm in patients with SARS-CoV-2 disease, mainly manifested in an increase in IL-2, IL-7, monocyte chemo-attractant protein 1 (MCP1), tumor necrosis factor-α (TNFa). Earlier studies of SARS-CoV infection have shown that it mediates its actions by suppressing the activity of NF-kB (kappa light chain nuclear factor-an enhancer of activated B cells), which leads to a decrease in the expression of cyclooxygenase-2, weakening inflammation [26].

Obesity is a state of mild inflammation with various inflammatory products directly secreted by adipose tissue. Hyperplastic and hypertrophied adipose tissue it releases inflammatory cytokines (TNFa, IL-1, IL-6, IL-10), adipokines (leptin, resistin, adiponectin), MCP-1, etc. Signs of inflammation are subsequent hypoxia and ischemia. Both hypoxia and ischemia lead to a state of oxidative stress, additionally stimulating the secretion of inflammatory proteins and active oxygen radicals that disrupt the functionality of mitochondria and DNA [27]. Thus, hypertrophied and at the same time hypoxic adipocytes change their normal protein synthesis and shift towards the production of cytokines and inflammatory proteins, which leads to insulin resistance, type 2 diabetes mellitus (DM2), metabolic syndrome, atherosclerosis and arterial hypertension [28].

Michalakisa & Iliasb suggested that leptin resistance may worsen the outcome of patients during the 2009 pandemic influenza A (H1N1), since leptin has a positive effect on the maturation, development and function of B cells, along with changes in lymphocytes and inhibition of CD8+ T-cell response and memory impairment of the T-cell response observed in obesity, which under normal circumstances will act against the virus [4].
II. CONCLUSIONS

A review of the literature made it possible to identify the main difficulties in managing patients with obesity and COVID-19: the presence of chronic concomitant diseases, an increased risk of ARDS, the risk of thromboembolic complications against the background of increased D-dimer levels and immune inflammation, the risk of bedsores, the risk of secondary infection, limited possibilities of medical manipulations and techniques.

REFERENCES


