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A SEVERE CLINICAL CASE REPORT OF A NOVEL CORONAVIRUS INFECTION (COVID-19) OF A PATIENT WITH DIABETES MELLITUS

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The article describes a clinical case of a severe course of a novel coronavirus infection (COVID-19) in a woman with comorbid pathology from the practice of the infection department of the Yakutsk Republican Clinical Hospital (YRCB). Type 2 diabetes mellitus, obesity, and arterial hypertension in an elderly woman were predictors of the severe course of a new coronavirus infection, which led to the development of ARDS and caused the death of this patient.

Keywords: COVID-19, viral pneumonia, diabetes mellitus, obesity, "cytokine storm", Yakutia.

Introduction. According to March 14, 2022, the total number of COVID-19 cases worldwide is more than 458 million, with more than 6 million deaths.

People with serious comorbidities are at a higher risk of severe disease caused by COVID-19 [1]. For example, an analysis of 1,099 diabetic patients hospitalized with COVID-19 showed that the severe course of infection (16.2%) was almost 3 fold more common than the mild course (5.7%) [2].

Regarding mortality, according to the results of the Chinese Center for Disease Control and Prevention, among 44,672 patients diagnosed with COVID-19, the mortality rate among people with diabetes was also significantly higher (7.3%) than in the general population (2.3%) [3].

In type 2 diabetes mellitus (T2DM), both humoral and cellular immunity are affected as a result of primary immune impairment. Poor glycemic control, in turn, impairs the immune response to viral infection and potential bacterial infection in the lungs. In addition, T2DM is associated with obesity, a risk factor for severe infection through systemic inflammation, bronchopulmonary pathology, and sleep apnea. Moreover, in DM the frequency of comorbidity and the presence of vascular pathology is high: cardiovascular disease, chronic heart failure (CHF), chronic kidney disease (CKD), diabetic foot, etc. [4, 5, 6].

Discussion: The article presents a clinical case of a novel coronavirus infection in the presence of many risk factors of a severe form of the disease, which resulted in a fatal outcome.

Patient P., 73 years old, first symptoms appeared on December 4, 2021 - fever up to 38.5°C, weakness, chills, pains in joints, body aches and pains. On December 6, 2021, she called a physician, and on December 7, 2021, a PCR test was taken for SARS CoV-2 RNA. During the outpatient phase of treatment, arbidol and antipyretic drugs was prescribed.

The patient had concomitant diseases: type 2 diabetes mellitus, grade 3 arterial hypertension, grade 1 obesity

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(BMI 30.11 kg/m2), chronic pyelonephritis, COPD. For BP stabilization she took Lorista 50 mg and Capotene 25 mg on an irregular basis. She suffered from diabetes mellitus since 2015, and took oral antidiabetic drugs irregularly. Periodically she received courses of Metformin (850 mg 2 times a day), Galvus (50 mg 2 times a day). She was not vaccinated against influenza, novel coronavirus and pneumococcal infections.

On the 4th day of the disease, i.e., on 08.12.21, the patient was hospitalized with a confirmed diagnosis in the infectious diseases department of the YRCB.

Objective status at admission: Condition of moderate severity, body temperature +36.2 °C. Alert. Appetite and sleep were preserved. The position was active. The skin and visible mucous membranes were clean, of normal color, no rashes. The pharynx had no catarrhal phenomena, and the tongue was clean and moist. Peripheral lymph nodes were not enlarged, painless. Respiration was independent, free, auscultatory in all fields, respiratory rate 18 per minute, SPO2 96% without supplementation with humidified oxygen. Heart tones are rhythmic, muffled. Hemodynamics are stable, BP 120/80 mmHg, heart rate 100 beats/min. The abdomen was soft, not swollen, painless on palpation, the liver was not enlarged. Urination was independent and free. Diuresis was adequate. Stool was normal. There were no edemas.

On admission the blood tests showed moderately elevated CRP 13.6 mg/l, ferritin - 106.7 mcg/l, LDH - 281 units/l, in the general blood test the leukocytes were 7.1 x 109/l, segmented neutrophils - 44%, stab neutrophils - 7.0%, lymphocytes - 36%, platelets - 226 x 109/l, ESR 19 mm/h, as well as there was hyperglycemia 16.6 mmol/l, moderate increase of transaminases ALT - 45.3 units/l, AST - 45.4 units/l. Computed tomography of the chest organs diagnosed bilateral polysegmental pneumonia, volume of lung lesion less than 25%, CT - 1.

Based on complaints, medical history, objective examination, the clinical diagnosis was established: U07.1 Novel coronavirus infection COVID-19 (confirmed on December 7, 2021), moderate form. Bilateral polysegmental viral pneumonia of CT-1 (09.12.2021). DN-0. Diabetes mellitus type 2. Hypertension 1 st., arterial hypertension 3 st., risk of CCO 3. Obesity 2 st.

The patient was treated as an inpatient in the infectious disease department for 4 days and received: Favipiravir 800 mg 2 times a day, with anticoagulant purpose Heparin 7.5 thousand units 3 times a day by p/k, With proactive anti-inflammatory purpose Metipred 60 mg every 6 hours (at 06:00, at 12:00, at 18:00, at 00:00). and also taking into account exacerbation of chronic bronchitis with presence of purulent sputum, Ceftriaxone 2 g once a day was appointed. Besides, the patient was prescribed short-acting insulin Apidra and long-acting insulin Tresiba 18 units at 21:00, Amlodipine 5 mg once a day, Omeprazole 20 mg twice a day, Bromhexin 8 mg three times a day with hypotensive purpose.

Despite the ongoing therapy, respiratory failure increased, hyperglycemia was not stabilized: glucose averaged 16.4 mmol/l, glycated hemoglobin - 11.7%. Given the negative dynamics, chest X-ray was performed repeatedly, where the progression of lung lesions up to 50% was detected, CT 2-3. Blood tests showed CRP 67.7 mg/L, LDH 364 units/L, creatinine 87 mmol/L, urea 8.4 mmol/L, general urinalysis showed moderate leukocyturia 4-6 per field of view, yeast-like fungi.

On 12/12/21, due to the severity of the disease, persisting temperature up to +37.2 °C, increased degree of lung damage to CT-3, decreased saturation to 89% when inhaled with atmospheric air, 94% with humidified oxygen, with the threat of ARDS, interleukin-6 inhibitor was prescribed with anti-inflammatory proactive purpose. With negative dynamics, on the 9th day of the disease the patient was transferred to the pulmonology department with ICU support.

An endocrinologist was consulted in the pulmonology department in connection with hyperglycemia up to 32 mmol/l, and hypoglycemic therapy was readjusted. The inflammatory process could not be controlled against the background of the therapy. Blood tests on December 13, 21 showed a significant increase in CRP level to 127.6 mg/L, ferritin 301.9, LDH 325 units/L, neutrophilic leukocytosis - leukocytes 10.1 x 10 9 9/L, neutrophils 81.9%, as well as elevated sedimentation to 33 mm/h. Given the increase in CRP level from 67.7 mg/L to 127.6 mg/L, high ferritin level of 301 mcg/L, low saturation, 94-95% with humidified oxygen supply, presence of severe KT-3 pneumonia with proactive anti-inflammatory purpose, intravenous levilimab 648 mg was re-injected.

On the 12th day of the disease, patient P. with deterioration was transferred to the emergency room with complaints of dyspnea, marked weakness, desaturation up to 85%. On December 15, 21, the volume of pulmonary lesions on CT scan. The volume of pulmonary involvement was 72-76%, CT 3/4, Lab tests showed white blood cells 32.4 x 109/L, neutrophils - 91.3%, sed rate 1.0 mm/h, platelets 231, glycated hemoglobin 12.5%, glucose 10.2 mmol/L, CRP 3.4 mg/L, ferritin 320 µg/L, LDH 730 units/L, creatinine 97.0 µmol/L, urea 7.6 mmol/L. Her condition progressively worsened, her respiratory and cardiovascular signs grew, her blood count increased to 27.1*109/l, neutrophils 85, Lymphocytosis up to 27.1*109/l, neutrophils - 85%, ALT - 67.4 units/l, AST - 153.7 units/l, urea - 23.7 mmol/l, creatinine - 801 µmol/l, ferritin - 406 µg/l, LDH - 1171 units/l, CRP - 7.2 mg/l, glucose - 10 mmol/l. According to the results of computed tomography on December 24, 21, compared to CT investigation on December 19, 2021, there was negative trend, the lesion volume was more than 75%, CT-4. Effusion in the left pleural cavity.

The patient died on December 30, 21, the cause of death was respiratory failure, progressive pulmonary and cardiac failure, multiple organ failure syndrome. Диагноз клинический посмертный:

Main disease: U07.1 - COVID-19, virus identified (07.12.2021), severe form.

Complication: community-acquired bilateral viral-bacterial polysegmental pneumonia of severe grade, CT-3-4. DN 2-3. On December 25, 21, he was placed on a ventilator. Syndrome of systemic inflammatory reaction. Syndrome of multiple organ failure. Pulmonary edema, cerebral edema.

Background disease: Diabetes mellitus type 2, decompensation stage. Target level of glycated hemoglobin less than 7.5%. Diabetic micro-, macroangiopathy. Diabetic nephropathy. Chronic kidney disease, stage 5, GFR 4 ml/min as of 12/29/2021. Mixed genesis dyscirculatory encephalopathy (hypoxic, diabetic).

Associated diseases: J12.8 - Other viral pneumonia. Atherosclerosis of the aorta, aortic and mitral valves. Minor pericarditis. Hypertensive disease, 3rd stage. Grade II arterial hypertension, risk of CCO4. Bilateral hydrothorax. Chronic heart failure, ejection fraction 64%, stage 2a. Chronic bronchitis, exacerbation. Grade I obesity. Adipose hepatosis. Urinary tract infection.

Conclusion: In the presented clinical case the patient had many factors aggravating the course of the infection process, such as type 2 diabetes mellitus, obesity, the presence of a focus of chronic infection (chronic bronchitis), arterial hypertension, diabetic nephropathy

and advanced age, which contributed to the unfavorable course of the new coronavirus infection.

Patients with diabetes mellitus should monitor glucose levels more closely throughout the day and continue taking the blood glucose-lowering drugs recommended by their physician. The presence of diabetes mellitus is a significant risk factor for the rapid progression and poor prognosis of novel coronavirus infection (COVID-19). This group of patients needs priority vaccination against COVID-19 and pneumococcal infection, which can significantly reduce the risk of developing viral-bacterial pneumonia.

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MULTISYSTEM INFLAMMATORY SYNDROME IN CHILDREN (CLINICAL OBSERVATION)

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Multisystem inflammatory syndrome is the most severe manifestation of a new coronavirus infection in children. The article presents the clinical case of the multisystem inflammatory syndrome features associated with COVID-19 in a teenager. The purpose of the work is to provide information on this topical clinical problem.

Keywords: multisystem inflammatory syndrome (MIS-D), fever, toxic erythema.

Introduction. Since April 2020, in some European countries and in the USA, children have been observed with signs of Kawasaki disease and toxic shock syndrome, causing inflammation of the whole body [6,8]. The new disease was given the name - children's multisystem inflammatory syndrome (MIS-D), associated with COVID-19. This disease occurs after infection with COVID-19 and affects mainly school-age children [4,5,6].

MVS-D is an inflammatory response of the body that occurs approximately 3-4 weeks after infection with a coronavirus infection. The initial symptoms of the disease are often manifested as fever, rash all over the body, redness of the eyes, abdominal pain, diarrhea and vomiting. The heart, blood vessels, and central nervous system are also affected, which requires emergency care [2,5].

According to the literature, almost 100% of patients with MVS-D have a fever, in one retrospective study of 21 patients it was reported that all had gastrointestinal symptoms, which usually occurred in the early stages of the disease [7]. Respiratory symptoms such as cough and rhinorrhea were relatively rare. Half of the patients had cardiogenic shock [9,10]. Another study reported that 56% of patients had macrophage activation syndrome (MAS), and Kawasaki-like symptoms were in 16-25% of patients [11].

The pathophysiology of multisystem inflammatory syndrome remains largely unclear. Apparently, it is based on a virus-induced hyperimmune reaction [3,8,5]. The most important role in the pathogenesis is played by the activation of T-lymphocytes, hyperproduction of pro-inflammatory cytokines (TNF-α, interleukins 1, 2, 6, 8, 10, granulocyte-macrophage colony-stimulating factor), deposition of immune complexes in the vascular wall. These mechanisms determine the development of a multisystem inflammatory response and explain most of the clinical and laboratory signs of the syndrome, such as fever, hyperferritinemia, coagulopathy, and an increase in inflammation markers [1,9].

Treatment. In accordance with the CDC (Centers for disease control and prevention) algorithm, therapy depends on the clinical manifestations and severity of the disease. Antibiotics are selected