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A CLINICAL CASE OF CONCURRENT COURSE OF THE NOVEL CORONAVIRUS INFECTION COVID-19 AND ACUTE MYOCARDIAL INFARCTION

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COVID-19 is a severe acute respiratory infection caused by the SARS-CoV-2 coronavirus primarily affecting the respiratory system and leading to pathological changes in the cardiovascular system. The aim of the research is to analyze the clinical course of the new coronavirus infection in patients with comorbid conditions and the development of heart damage associated with COVID-19.

Materials and Methods: A clinical case of a patient belonging to a high-risk group is described, having comorbidities (such as chronic heart failure, type 2 diabetes mellitus, arterial hypertension), an age of 77 years, and female gender. Despite the patient's late presentation for medical assistance, resulting in worsening of myocardial infarction, which occurred in combination with severe acute respiratory distress syndrome, timely identification of the risk of cardiovascular complications was carried out during the hospitalization stage, and specific targeted therapy was initiated. However, 11 months after discharge, the patient experienced a second myocardial infarction, followed by a third one after 3.5 months (both with favorable outcomes).

Results of the Study: The presented clinical case confirms the existence of pathogenetic interaction between cardiovascular diseases and COVID-19, wherein the SARS-CoV-2 virus penetrates lung cells, heart, and other organs, affecting the endothelium of blood vessels, leading to partial destruction and damage to cardiac tissue.

Keywords: COVID-19, clinical case, arterial hypertension, myocardial infarction, risks of cardiovascular complications.

Background. COVID-19 is a severe acute respiratory infection caused by the SARS-CoV-2 coronavirus, not only affecting the respiratory system but also causing pathological changes in the cardiovascular system. The presence of concomitant cardiovascular diseases (CVD) determines a wide variability in the clinical course of COVID-19, rapid development of severe complications, and risks of a fatal outcome.

Research Objective: Analyzing the clinical course of the new coronavirus infection in patients with comorbid conditions and the development of heart damage associated with COVID-19.

Materials and Methods. A retrospective analysis of a patient's medical history was conducted who was hospitalized in May 2021 at the Yakutsk Republican Clinical Hospital (YRCH) with a confirmed diagnosis of "new coronavirus infection". A clinical example of a severe course of COVID-19 with the development of cardiovascular complications at different stages of the patient's illness was demonstrated. The patient belonged to a high-risk group: having 3 comorbid conditions (chronic heart failure, type 2 diabetes mellitus, arterial hypertension), being over 65 years old, and female.

Clinical Case. The patient U., 77 years old, was admitted complaining of pronounced shortness of breath, nausea, and dull chest pains. She considered herself ill since May 13, 2021, experiencing general weakness and shortness of breath during regular physical exertion. She did not seek medical help and did not take any medications. On May 18, 2021, due to worsening shortness of breath, increasing weakness, and the onset of chest pains, she consulted her local therapist. Her medical history included type 2 diabetes treated with insulin, ischemic heart disease (IHD), a previous myocardial infarction in 2019, and hypertension. On the same day, U. was hospitalized at the district Central Regional Hospital with suspected severe acute respiratory distress syndrome (SARDS) and community-acquired pneumonia. On May 19, 2021, a CT scan of the chest revealed bilateral lower lobe bronchopneumonia, and the treatment prescribed included levofloxacin, dexamethasone, interferon, heparin, Lasix, and Actrapid.

On May 21, 2021, 8 days after the onset of the illness, based on a telemedicine consultation, she was airlifted for inpatient treatment at YRCH, to the pulmonary department for COVID-19 patients, with the diagnosis: "Primary diagnosis: New coronavirus infection COVID-19, virus identified, moderate-severe form. Community-acquired bilateral multisegmental pneumonia, severe course."

From the epidemiological history, it is known that the patient had not previously contracted COVID-19, lives alone, received a flu vaccine in October 2020, and had not been vaccinated against pneumococcal infection or COVID-19.

Upon admission, her condition was assessed as severe. Her body temperature was 36.5°C. She had pronounced shortness of breath, a respiratory rate of 24-26 breaths per minute, SpO2 at 96% with the administration of supplementary oxygen at a rate of 15 liters per minute (without oxygen, saturation decreased to 80%). She had elevated blood pressure at 180/90 mmHg, a heart rate of 81 beats per minute. She was admitted to the intensive care unit (ICU) where she received oxygen therapy through nasal cannulas at a rate of 15 liters per minute, prophylaxis against thromboembolic complications, correction of acid-base balance and fluid-electrolyte balance, antibiotic therapy, hormonal preventive therapy, and adjustment of hypoglycemic and antihypertensive medications.

Upon examination at admission: PCR for COVID-19 was positive, the blood test showed anemia (hemoglobin 83.10 g/L), marked leukocytosis 23.5010⁹/L, thrombocytosis 599.010⁹/L, accelerated ESR 53.0 mm/h. Biochemical blood analysis revealed elevated liver transaminases activity, lactate dehydrogenase, hyperglycemia, and inflammatory response (glucose - 15.3 mmol/L; ALT- 75.9 IU/L; AST- 81.4 IU/L; lactate dehydrogenase- 399.1 IU/L, C-reactive protein-110.6 mg/L).

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On the chest CT scan from May 21, 2021: signs of bilateral multisegmental interstitial pneumonia. High probability of COVID-19. Severity of lung involvement on CT scan is more than 75%. CT-4. Differential diagnosis includes pulmonary edema. Basal segments of lower lobes on both sides show compressive atelectasis. CT signs of mediastinal lymphadenopathy (Figure 1).

ECG from May 21, 2021: Sinus rhythm, HR=64 bpm. In leads II, III, AVF, V5-V6 (inferior, lateral wall), there is observed ST segment depression with a positive T wave (Figure 2). A high-sensitivity troponin was measured: elevated result of Troponin I up to 1.340 ng/mL.

According to the echocardiogram (May 22, 2021): "Areas of local contractility impairment: hypokinesis in the basal anterior-septal, mid posterior-septal, basal posterior-septal regions.

Conclusion: Global left ventricular contractility reduced EF - 34% (C). Aortic wall and valve leaflets thickening. Mitral valve regurgitation - 1-2 degrees, tricuspid valve - 1 degree, aortic valve - 2 degrees. Left atrium and pulmonary trunk dilatation. Pulmonary hypertension - 1 degree." Based on the examination results, the clinical diagnosis was established: Primary diagnosis: U07.1 - New coronavirus infection COVID-19, virus identified, moderate-severe form. Community-acquired severe-grade bilateral multisegmental pneumonia, CT-4.

Background: Type 2 diabetes, on insulin. Diabetic microangiopathy. Diabetic macroangiopathy.

Concomitant conditions: Ischemic heart disease. Post-infarction cardiosclerosis (2019). Grade 3 hypertension. Stage 2 arterial hypertension. Left ventricular hypertrophy. Cardiovascular risk - 4. Secondary atrial enlargement. Relative tricuspid valve insufficiency - 2 degrees, mitral valve - 1-2 degrees. Pulmonary hypertension - 1 degree. Bilateral hydrothorax. Chronic heart failure with low ejection fraction (34%) 2B stage. Moderate anemia. Chronic cholecystitis, not in exacerbation.

Considering the suspicion of acute coronary syndrome, a cardiologist was consulted, and coronary angiography with PCI-1 stent in RCA No.2-CIMP was performed. Anti-anginal, antiplatelet, diuretic, antiarrhythmic therapy was prescribed, along with recommendations for monitoring, clinical analysis, and ECG control. In the subsequent course, anginal pain, dyspnea, and biochemical blood parameters increased: ALT increased to 85 IU/L, AST to 100.5 IU/L, creatine phosphokinase. Total creatine kinase: 262.7 IU/L (26-192); Troponin I up to 1.650 ng/mL; also LDH: 528.0 IU/L (135-214). Amid intensive therapy, C-reactive protein decreased to 62.7 mg/L, in CBC: leukocytosis slightly decreased to 20.1210^9/L (4-10); low hemoglobin persisted (HGB): 85.80 g/L (110-160); erythrocytes - 3.510^{12}/L, platelets - $616*10^{9}$/L. ECG recorded negative dynamics with the appearance of ischemic changes (Figure 3).

Based on the results of measuring the Na-uretic peptide, an elevated BNP level of 1520 pg/mL was detected, indicating the presence of chronic heart failure in the patient. On May 22, 2021, a diagnosis of "acute myocardial infarction with Q-wave formation in the anterior-septal wall, presumably from May 21, 2021" was established by the cardiologist. The patient was transferred to Cardiac Center No.1 - Clinical Emergency Medical Center (CERC) on May 23, 2021, where a selective coronary angiography was performed: Atherosclerosis of the coronary arteries. Stenoses: Left main coronary artery (LMCA) 70%, proximal left anterior descending artery (LAD) 80%, proximal circumflex artery (Cx) 90%, distal Cx 50%, ostial right coronary artery (RCA) 50%, proximal posterior descending artery (PDA) 70%, distal PDA 80-70-80%, obtuse marginal artery (OM) 70%. Right dominance of myocardial blood supply. Due to complex multivessel coronary artery disease, stable hemodynamics, and a high Syntax score (53 points), a decision was made to refrain from percutaneous intervention. After two weeks, the patient was discharged in satisfactory

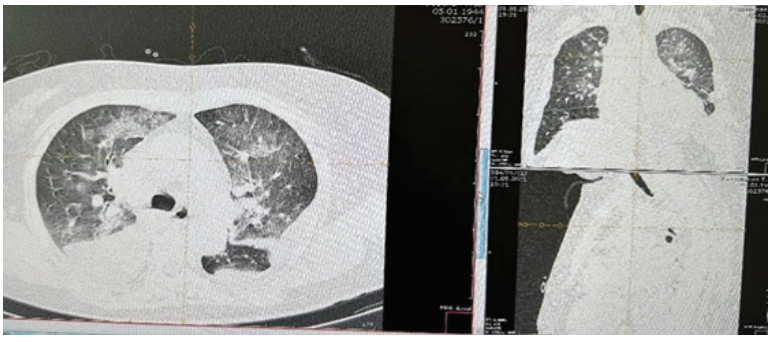

Fig. 1. Bilateral multisegmental interstitial pneumonia, 8th day from the onset of the first symptoms of the illness.

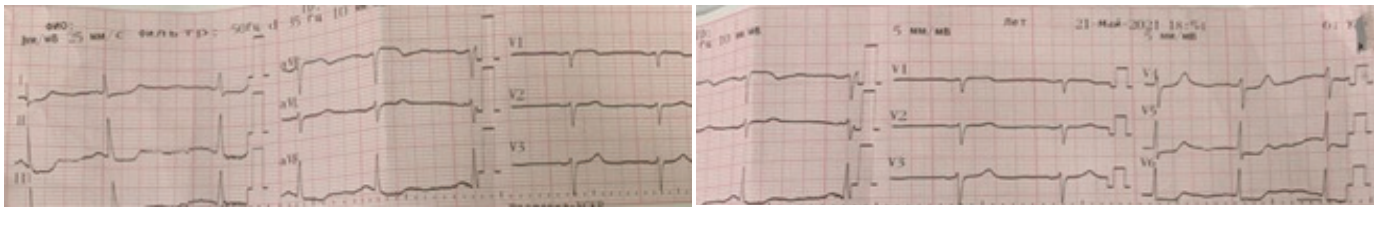

Fig. 2. ECG of patient U. upon admission

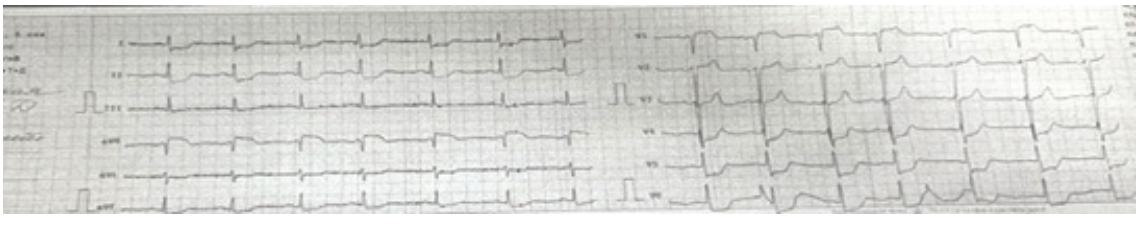

Fig. 3. ECG of patient U. one day after admission

condition, and a follow-up chest CT scan showed a reduction in the extent of damage to CT-2. However, after 11.5 months, in April 2022, the patient presented with clinical signs of a recurrent myocardial infarction and acute left ventricular failure at the Central District Hospital of the Momsky District. Diagnostic coronary angiography (April 20, 2022) revealed multivessel atherosclerotic coronary artery disease: Left main artery stenosis 70%, left anterior descending artery stenosis 70% at the ostium, 50% in the proximal third, 80% in the mid-third; diagonal branch stenosis 50%; circumflex artery 90%; obtuse marginal artery 50% at the ostium, 60% in the mid-third; right coronary artery stenosis 90% in the proximal third, 80% in the mid-third, 90% in the distal third; posterior descending artery 70%. No endovascular treatment was administered. Considering negative cardiac markers and stabilized hemodynamics, the patient continued with conservative therapy. Despite following all recommendations, the patient experienced acute decompensation of heart failure on August 1, 2022: worsening dyspnea and pronounced weakness, which the patient attributed to increased physical exertion the day before. On the same day, the patient was hospitalized in the therapeutic department of the district hospital. Examinations revealed a positive qualitative troponin test, consistent ECG changes: ST segment depression in the anterior-lateral and inferior walls of the left ventricle, ST segment elevation in aVR. Additionally, moderate anemia was diagnosed and corrected through blood transfusion. After coordination through telemedicine consultation, the patient was transferred for further treatment to a Regional Cardiac Surgical Center Oncology (RKCS ONC). After stabilization, the patient was transferred to a general ward. Subsequently, the patient underwent coronary angiography with stenting of the coronary artery (PCI with stenting of the posterior descending artery (PDA) using Resolute Integrity stents sized 3.030mm (distally p=16 atm.), 3.026mm (mid-section p=16 atm.), and 3.0*30mm (proximally p=18 atm.) on August 17, 2022. Conservative management was recommended, including adherence to antiplatelet therapy and anticoagulants. With improved condition, the patient was discharged for further treatment and follow-up at home, with a scheduled follow-up coronary angiography after 12 months.

Discussion. The presented example demonstrates that COVID-19 in comorbid patients can lead to the development of combined complications caused by the infection, in this case - non-hospital bilateral pneumonia and acute myocardial injury. The situation was complicated by the fact that in the district hospital, due to the lack of appropriate facilities, timely ECG, troponin test, and EchoCG were not performed, and timely anti-anginal and antiplatelet therapy for myocardial infarction was not administered. In addition to ECG and Echo-cardiography, the acute myocardial necrosis in the patient was indicated by elevated levels of troponin and cardio-enzymes (lactate dehydrogenase, liver transaminases, creatine phosphokinase). Considering the patient's heart failure, coronary artery disease, and stage 3 hypertension, it was reasonable to prescribe medications with anti-anginal, antiarrhythmic, antiplatelet, and antihypertensive effects. Severe COVID-19 progression can lead to cardiac pathology even after the patient's recovery - as in this case, leading to two myocardial infarctions occurring 11 months after discharge with a 3.5-month interval. The mechanism of cardiac involvement is primarily linked to the SARS-CoV-2 virus penetrating the ACE2 protein found in the endothelium of blood vessels and cells of the heart, lungs, and other organs, causing partial destruction and damage to the cardiac tissue. Studies [3, 5] have indicated involvement of the renin-angiotensin-aldosterone system among molecular and pathophysiological mechanisms since SARS-CoV-2 tropism is associated with angiotensin-converting enzyme 2, leading to altered neuromuscular response to SARS-CoV-2 tropism. This leads to abnormal functioning of the smallest blood vessels, endothelial inflammation, microthrombosis, vessel wall damage, and increased permeability. As a result, the risk of coronary artery disease leading to myocardial infarction increases. The presence of type 2 diabetes in the patient, which contributed to the recurrent myocardial infarctions, led to characteristic systemic inflammatory shifts, immune system function disturbances, metabolic changes, resulting in severe pneumonia with persistent oxygen deprivation, high hyperglycemia, systemic inflammatory disorders, and decreased tolerance for physical exertion. The onset and development of acute cardiac damage in COVID-19 are likely associated with the virus's spread from the respiratory tract through the bloodstream or the lymphatic system, as suggested by Italian researchers [6]. There is currently no confirmed evidence of detecting SARS-CoV-2 RNA in the heart. However, there are observations of the direct impact of SARS-CoV-2 on the cardiovascular system, eliciting an excessive inflammatory response that damages the myocardium [1]. The comorbid conditions of anemia and severe hypoxia due to SARS-CoV-2-induced pneumonia contributed to oxidative stress, myocardial damage, and irreversible destructive processes due to increased myocardial oxygen demand.

Conclusion. Thus, the presence of cardiovascular diseases may be associated with an increased risk of severe complications and outcomes of COVID-19. This is due to the characteristics of the modern population with cardiovascular diseases, dominated by the elderly and comorbid conditions. Conversely, COVID-19 can have an acute and chronic damaging effect on the cardiovascular system. It is anticipated that the COVID-19 pandemic will trigger a new wave of non-infectious diseases, primarily cardiovascular diseases, as their common pathogenesis is the chronic pro-inflammatory status [2].

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