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The clinical case of myocardial infarction after COVID-19 infection

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SARS-COV-2 has caused one of the largest pandemics by now. Cardiovascular complications occur in 30–45 % of cases and, along with respiratory failure, are the cause of death in 65 % of patients with unfavorable disease course. This article presents clinical case that demonstrates patient with comorbid diseases (coronary artery disease, arterial hypertension) after COVID-19 reinfection who developed myocardial infarction with cardiac arrest that was caused by cardiopulmonary insufficiency.

The patient was admitted with the diagnosis of acute myocardial infarction along with severe novel coronavirus infection. Complications of the main disease: bilateral diffuse COVID-associated pneumonitis, alveolitis. Concomitant diseases: arterial hypertension, stage III, left ventricular hypertrophy.

The case demonstrates the sequence of changes not only in vessels, but also in body organs with the development of acute myocardial infarction after SARS-COV-2 reinfection and emphasizes the need for long-term observation after the infection in patients with comorbidities such as coronary artery disease and arterial hypertension.

Key words: COVID-19, pulmonary fibrosis, myocardial infarction.

Conflict of interest: none declared.

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Introduction

The COVID-19 outbreak has become one of the main health issues worldwide [1]. In 50 % of cases, it is characterized by a severe and very severe disease course that includes the development of pneumonia, acute respiratory distress syndrome (ARDS), and multiple organ dysfunction [1–5]. Cardiovascular complications occur in 30–45 % of cases and, along with respiratory failure, are the cause of death in 65 % of patients with unfavorable disease course. Cardiac damage can have ischemic/non-ischemic causes and manifest by increased troponin and B-type na-

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triuretic peptide levels, which are associated with increased risk of ARDS, ventricular tachyarrhythmia, myocardial infarction, acute kidney injury and coagulopathy. These pathological processes are of special importance in patients with concomitant cardiovascular diseases, which increase the risk of both severe COVID-19 course and mortality in case of re-infection [3, 4]. However, literature data on morphological changes in body organs after the SARS-CoV-2 infection and re-infection are limited.

Clinical case

Patient T.A., woman of 65 years old, was admitted to the Republican Clinical Hospital of the Ministry of Health of the Chuvash Republic with the diagnosis of acute myocardial infarction. Severe novel coronavirus infection was confirmed by the presence of Immunoglobulins G (IgG) and M (IgM) to coronavirus infection (SARS C0V-2): IgG positive, positive ratio (PR) — 15.7 and IgM positive, PR — 7.4). Complications of the main disease included bilateral diffuse COVID-associated pneumonitis and alveolitis. Concomitant diseases were: arterial hypertension, stage III, left ventricular hypertrophy. Target BP — 130–139/80 mmHg.

According to medical records, patient has been suffering from elevated blood pressure for about 15 years and received antihypertensive medications. She has been treated for coronary artery disease (CAD) in the outpatient setting. Two months ago, she was diagnosed with COVID-associated pneumonitis with diffuse bilateral infiltrates and alveolitis. She took antibiotics and expectorants by herself that did not improve her health state. When she had come to the hospital: chest computed tomography (CT) revealed bilateral diffuse viral COVID-associated pneumonitis, alveolitis, with about 75 % damage of the lung tissue, compaction of the lung tissue in the form of "ground glass opacity", polymerase chain reaction (PCR) for COVID-19 was positive. Patient received treatment at the Department of Infectious Diseases where her health state significantly improved. She was discharged after two consecutive negative nasopharyngeal swab tests had been received followed by outpatient treatment.

Approximately one week ago, she complained of cough with sputum that was difficult to separate and fatigue. She called an ambulance due to chest pain, spreading through the chest surface. Electrocardiogram (ECG) showed: sinus rhythm, ST segment elevation in AV–I, V2–5 leads, reciprocal depression of the ST segment in II, III, AVF leads.

Medical examination at admission: general health state is severe. Skin: pale with cyanotic tint, lower extremity edema. Body temperature — 37.1 °C.

Neurological examination. Conscious, verbal contact is limited due to cognitive impairment. Limb movements are limited. Sensitivity is not broken. Pupils: d=s=4 mm, the pupillary light reflex is preserved. Body type is hypersthenic. Weight=84 kg.

Respiratory system. Chest palpation is painless, auscultation revealed harsh symmetrical lung sounds, breath sounds are diminished on the right side and in the lower lobes, no wheezing, respiratory rate — 66/min, SaO2–47 %.

Cardiovascular system. Heart sounds are muffled. Heart rate — 111 beats/minute. The pulse on the radial artery is determined with normal filling pulse voltage. BP — 160/80 mmHg.

Digestive system. The tongue is dry with "dirty" coating at the root. Abdomen was symmetrical, soft and painless during palpation. Bowel sounds are determined via auscultation. The genitourinary examination has no pathology, diuresis values are within normal limits.

Treatment included antiarrhythmic medications, beta blockers, thrombolytics, nitrates. The coronary angiography revealed atherosclerotic lesion of three coronary vessels including the lesion of the trunk of the left coronary artery.

Lung CT scan revealed pneumonia, signs of lung congestion, compaction of the lung tissue that manifested as "ground glass opacity", pulmonary consolidation in combination with reticular opacities, approximately 75 %-damage of lung tissue.

ECG dynamics: ST segment elevation in V2-V4 leads, followed by spread to the inferior wall, ST segment elevation in III, II, AVF S1–4 leads — circular lesion. Troponin level — 14 ng/ml.

Laboratory investigations: signs of inflammation progressed in blood tests (leukocytosis — 35.5×109/l, segmented neutrophils — 84.6 %), thrombotic microangiopathy; almost two-fold increase of lactate dehydrogenase (LDH). The level of C-reactive protein was elevated up to 95 mg/l, ferritin and presepsin levels corresponded to an average risk of systemic inflammation development (487 pg/ml); increased risk for micro thrombosis (increased D-dimer, LDH).

Acid-base analysis revealed hypoperfusion — metabolic (6 mmol/l) lactic acidosis (pH — 7.20, BE (base excess) — 16 mmol/l); — progression of respiratory failure — the decrease of the SatO2 to 46 % (measured with pulse oximeter) during atmospheric air breathing, tachypnoea up to 36/min.

Despite ongoing treatment, the patient's condition had progressively worsened, and death was declared.

Pathological examination revealed bilateral polysegmental viral pneumonia with areas of carnification. The examination determined uneven compaction of pulmonary tissue predominantly in the posterior sections; heavy, airless, pieces of lung tissue that sank in water; reddish incisions with increased amount of reddish liquid that flows down from the surface of the when pressed. Histological examination revealed extensive areas of pneumosclerosis with many full-blooded vessels with perivascular sclerosis (Figure 1). The interalveolar septa are unevenly thickened with round cell infiltration and plethora of the capillaries. Alveoli are in the state of dystelectasis with the desquamation of alveolocytes into alveolar lumen and macrophages into the alveolar wall, homogeneous pink masses in the form of "crescents" similar to hyaline membranes, blood clot in the vessel (Figures 2, 3). Bronchial walls were sclerotized and thickened to varying degree, loosely infiltrated with lymphoid elements, with fibrin into the bronchial lumen.

Acute transmural myocardial infarction (MI) of the posterior LV wall was detected. Macroscopical examination revealed dark red area up to 4.7×4.5×1.7 cm in

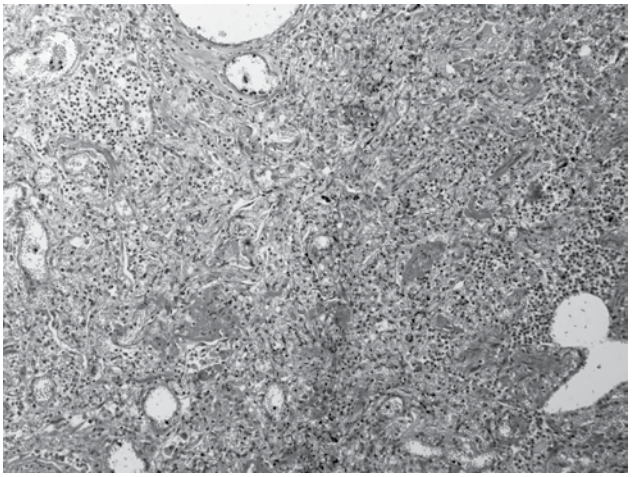


Figure 1. Microscopic picture of pneumofibrosis (1) with vessels plethora. Hematoxylin and eosin (H&E) staining, ×900

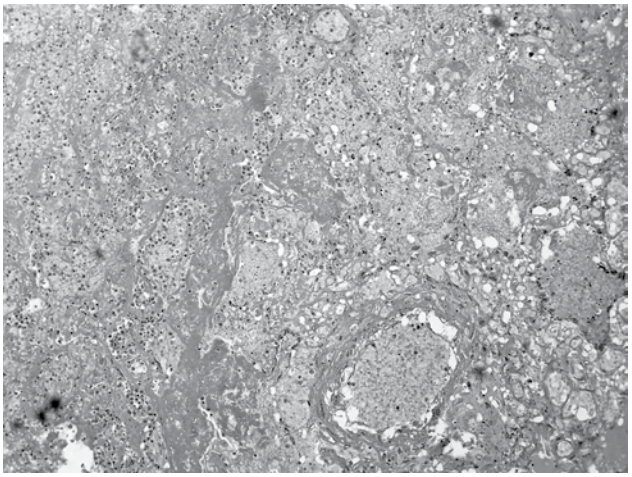
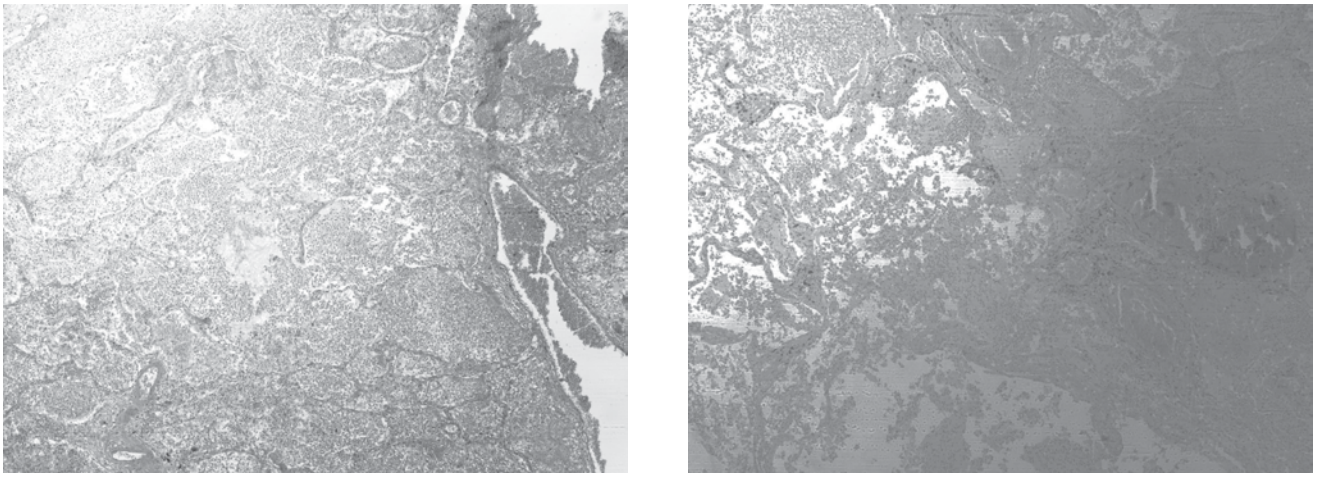


Figure 2. Microscopic picture of the thrombus into the vessel, hyaline membranes, areas of fibrosis. Hematoxylin and eosin (h&e) staining, x900



a b

Figure 3. Microscopic picture of viral pneumonia: a) inflammatory exudate, dilated and plethora vessels, interalveolar septa and hyaline membranes; b) leukocyte infiltration into the lumen of the alveoli with hemorrhagic component, blood vessels with sludge. Hematoxylin and eosin (H&E) staining, ×900

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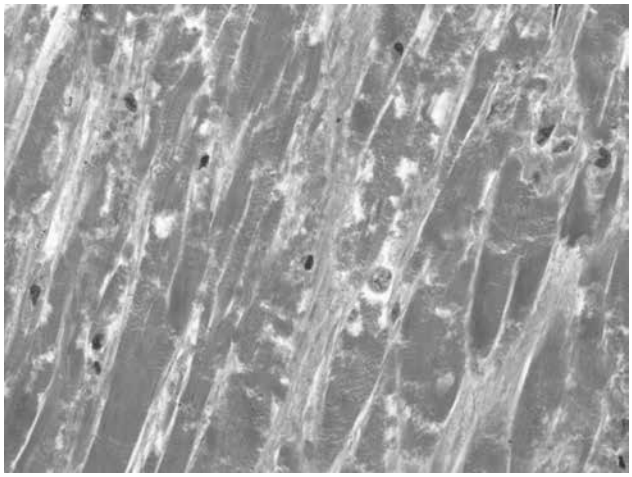

Figure 4. Microscopic picture of necrosis in cardiomyocytes (nuclear karyolysis). Hematoxylin and eosin (H&E) staining, ×900

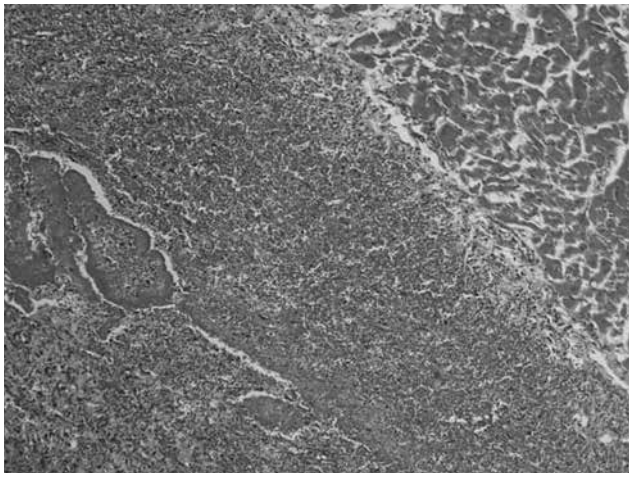

Figure 5. Microscopic picture of the demarcation line and myomalacia. Hematoxylin and eosin (H&E) staining, ×900

size at posterior LV wall. Cardiac tissue was dense, 13x10x8 cm in size, with mixed blood clots into heart chambers. The thickness of the right ventricular wall was 0.3 cm, of the left ventricle — 1.7 cm. Histological examination revealed interstitial edema, uneven fragmentation of muscle fibers, uneven hypertrophy of cardiomyocytes, connective tissue proliferation. There were areas of homogenization of muscle fibers with loss of nuclei and transverse striation with small hemorrhages and mild perifocal neutrophil infiltration (Figure 4), areas of autolysis into the necrotic zone (Figure 5). Coronary arteries had dense unevenly thickened walls and the lumen on transverse sections was narrowed up to 60–80 % due to atherosclerotic plaques. The lumen of pulmonary artery branches had liquid blood and blood clots. The intima of the aorta had pale yellow color and was covered with fatty streaks and atherosclerotic plaques at the stage of atheromatosis and calcification. In the intima of the mesenteric, iliac, and femoral arteries, there were also numerous atherosclerotic plaques with areas of calcification and atheromatosis.

The kidneys had dense tissue, bean-shape and size of 8×5×3 cm. The renal capsule was even, easily removed. The surface had small cicatricial retractions of brownish-gray color. The parenchyma was plethoric with numerous whitish-gray small veins. The kidneys anatomical pattern was easily distinguished — the boundary of cortex and medulla zones was clear. The pelvicalyceal system did not show any visible pathology, the mucosa was grey in color, smooth and clean. The urinary tract had no obstruction — the ureters had no constrictions or strictures, its mucous membrane was pale, gray in color, clean. Histological examination: the stroma was edematous; capillaries of the glomeruli were unevenly plethoric. Hyalinized glomeruli were identified. The epithelium of the kidney tubules had dystrophic and necrobiotic changes (Figure 6). The medulla had perivascular hemorrhages and areas of plethora (Figure 7). Arterial walls were circularly thickened and sclerotized.

There were signs of multiple organ failure that manifested as pulmonary and cerebral edema.

The following pathological diagnosis was established:

Main pathology: novel coronavirus infection (COVID-19), confirmed by the presence of Immunoglobulin G (IgG) and M (IgM) to coronavirus infection (SARS C0V-2): IgG positive, positive ratio (PR) — 15.7 and IgM positive, PR — 7.4): bilateral diffuse COVID-associated pneumonitis, alveolitis with areas of pneumosclerosis, acute transmural myocardial infarction of the posterior left ventricular wall.

Complications of the main pathology: acute respiratory distress syndrome, acute coronary thrombosis due to associated thrombotic lesion, acute cardiopulmonary failure, pulmonary edema, cerebral edema.

Concomitant diseases: arterial atherosclerosis at the stage of atheromatosis and calcification (60–80 % stenosis). Arterial hypertension: left ventricular hypertrophy (1.7 cm), focal glomerulosclerosis.

Medical records and autopsy results testify that the cause of death of 65-year-old woman was COVID-associated pneumonitis with diffuse bilateral infil-

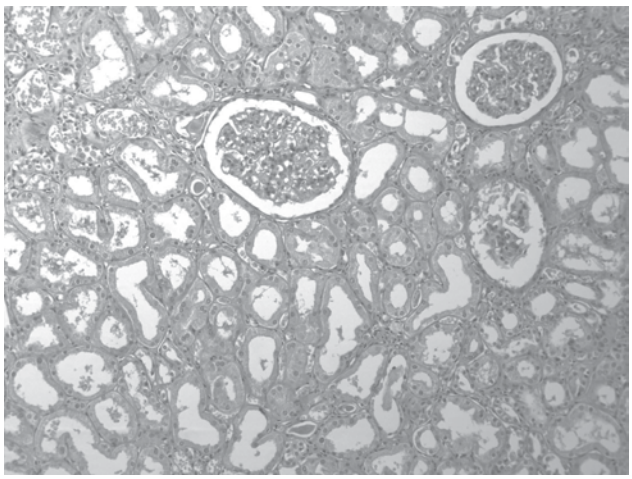

Figure 6. Microscopic picture of glomerular hyalinosis, signs of colloidal droplets into renal tubules similar to "thyroid kidney", degeneration of the tubule epithelium. Hematoxylin and eosin (H&E) staining, ×900

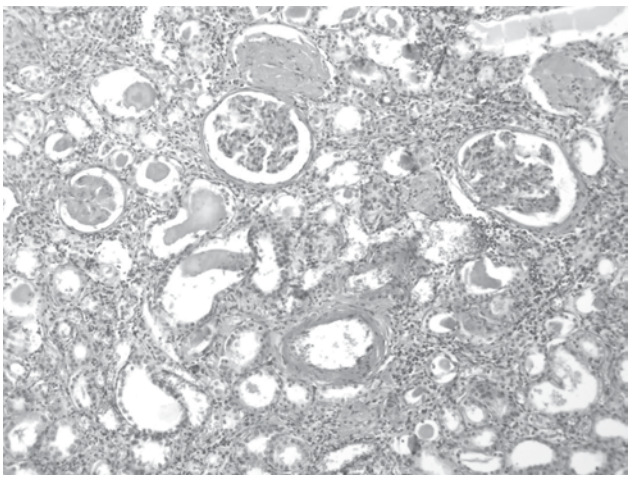

Figure 7. Microscopic picture of areas of necrosis of the kidney tubules epithelium with erythrocyte sludge into blood vessels. Hematoxylin and eosin (H&E) staining, ×900

trates and alveolitis complicated by the development of acute respiratory distress syndrome and acute MI that aggravated the course of the disease and affected the outcome by the cause of cardiopulmonary failure.

Discussion

It is known that cardiovascular complications are diagnosed in approximately 40% of patients who deceased from COVID-19 infection, AH and diabetes mellitus — in 30%. One study (n=41) revealed myocardial injury (high levels of high-sensitivity cardiac troponin I), another study (n=138) diagnosed acute cardiac injury (in 7.2% of cases), shock (8.7%) and arrhythmias (16.7%); most of the patients who developed these complications were admitted to intensive care unit. Previously published reports describe cases of acute heart failure, MI, myocarditis, and cardiac arrest. Patients with CAD had particularly high risk of complications due to atherosclerotic plaque rupture at the background of virus-induced inflammation.

Current clinical case describes changes in organs during recurrent COVID-19 infection that aggravated the course of existing cardiovascular diseases and led to the development of MI. The pathogenesis of cardiac damage is associated with vasoconstriction (due to the elevation of angiotensin II after the SARS-COV-2 blockage of angiotensin-converting enzyme II receptors); violation of oxygen delivery and consumption due to respiratory failure; the development of MI (types 1 and 2) associated with recurrent "cytokine storm" and thrombosis due to covid-associated coagulopathy. Thus, the development of acute MI led to the disease course aggravation that contributed to the occurrence of multiple organ failure and played the role of significant risk factor for an unfavorable outcome.

Cardiovascular complications in patients with COVID-19 are associated with the existence of cardiovascular diseases, especially with CAD, AH, and atherosclerosis. It is obvious that secondary damage is associated with chronic immune inflammation, direct damage of cardiomyocytes by SARS-CoV-2 that is confirmed by the increase of TnI along with inflammatory markers (interleukin-6, D-dimer, ferritin and LDH). According to F. Zhou et al. [8], the frequency of TnI increase was significantly higher among deceased patients, and can be used as inpatient death predictor (OR 80.07; 95% CI 10.34–620.36; p<0.0001) [5].

Conclusion

In the context of the current COVID-19 pandemic, patients with existing cardiovascular pathology represent special risk group for re-infection and have high risk of post-COVID complications and mortality. Cardiovascular complications significantly aggravate the course of COVID-19 and are associated with the development of multiple organ dysfunction syndrome, and are the main cause of poor outcome.

Conflict of Interest: none declared.

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