COVID-19 and myocardial infarction with myomalation. A clinical case report

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This article presents a case of the development of antero-inferior myocardial infarction, myomalation, apex wall rupture and hemopericardium following SARS-CoV2 infection.
The clinical case. Patient T.E., 36 years old, was admitted to hospital on 15.05.2023 with the diagnosis: "New coronavirus infection of severe degree. Acute myocardial infarction". She had no complaints on admission. From the medical history: she became ill three weeks before the hospitalisation, when the weakness appeared, body temperature increased to 37.3 °C. She took non-steroidal anti-inflammatory drugs with a temporary improvement. For several days the body temperature reached up to 38.4 °C. In the evening of 14.05.2023 the patient noted a transient substernal discomfort at rest. 15.05.2023 — the patient's condition worsened, pressing substernal pain had appeared, that led to an ambulance call. Electrocardiogram (ECG) data: abnormal Q-wave in leads II, III, aVF and V2-V6. In the same leads there were the ST segment elevation and the inversion of the T-wave. Blood pressure (BP) — 105/76 mmHg. The NEWS2 score is 9 points. PCR test for coronavirus is positive. Chest computed tomography (CT) scan: CT evidence of viral interstitial pneumonia — CT-3 (73 % of lung tissue lesions). Despite the initiated treatment, the patient died. The autopsy revealed signs of viral pneumonia. Karyolysis and the accumulation of blood between myocytes were found in the heart. The myocardium was circularly flaccid; there was a slit-shaped irregular defect with the disruption of myocardial integrity in the area of the inferior and anterolateral wall of the left ventricle (LV).
Conclusion. In the case presented, a young patient without comorbidities developed an anterior-inferior MI after SARS-CoV-2 infection. Severe complications occurred — myomalation, inferior and anterolateral LV wall ruptures and hemopericardium.

Keywords: COVID-19, thrombosis, coronary arteries, myocardial wall rupture, hemopericardium.

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Introduction

The most prominent clinical manifestation of COVID 19 is pulmonary damage. However, after infection with **SARS-CoV-2**, the hematopoietic system is primarily affected and coagulopathy occurs, which plays an important role in the pathogenesis and clinical manifestations of the disease [1–3]. When secondary activation of the coagulation system occurs during severe infection, endogenous anticoagulant mechanisms cannot be controlled and acute generalized inflammatory reaction leads to endothelial vascular dysfunction, resulting in generalized thrombosis and tissue ischemia.

The incidence of cardiovascular complications after **SARS-CoV-2** infection ranges from 5 to 38% in hospitalized patients [3–5]. These include the development of acute heart failure due to acute coronary syndrome, myocardial infarction (MI), myocarditis, arrhythmias [2, 3]. COVID-19 infection affects important pathways of biochemical regulation of the heart, such as ACE2 signal transduction pathway, fibrinogen pathway, redox homeostasis, leads to destabilization and rupture of atherosclerotic plaques, exacerbates myocardial damage and dysfunction [6, 7]. Myocardial damage without direct plaque rupture can also occur due to cytokine storm, hypoxic state, coronary spasm, endothelial or vascular dysfunction [8–10]. Despite the emerging trend of decreasing cases of COVID-19, the problem continues to persist, MI in COVID-19 cases remains one of the debated issues in the medical scientific community. Due to the relevance of the problem, we present a case of development of circular myocardial infarction, myomalation, rupture of the inferior anterolateral LV wall and hemopericardium after SARS-CoV2 infection.

Clinical case

Patient T.E., 36 years old, on 15.05.2023 was hospitalized for several hours at the Republican Cardiology Dispensary of the Ministry of Health of Chuvashia in the ICU. She was admitted for the diagnosing and treatment of COVID-19 coronavirus infection and its complications. She had no complaints on admission. From the medical history: she became ill three weeks before the hospitalisation, when the weakness appeared, body temperature increased to 37.3 °C. She took non-steroidal anti-inflammatory drugs (NSAID) and analgetics with a temporary improvement. For several days the body temperature reached up to 38.4 °C, the patient continued the intake of antipyretics with mild improvement. In the evening (14.05.2023) she noticed transient discomfort behind the sternum at rest. In the morning (15.05.2023) the patient's condition worsened, the substernal pressing pain appeared, she took NSAIDs — with no effect. In the evening (15.05.2023), due to persisting symptoms, called an ambulance and was admitted to the Republican Cardiology Dispensary.

Known diseases: according to the outpatient card — no chronic diseases of internal organs. The patient rarely sought medical help.

Physical examination: Medical state is critical. Consciousness is clear. Emotionally stable. Normosthenic physique. Superficial and deep sensitivity are preserved. Skin: cyanotic color. Visible mucous membranes are pale pink. Peripheral lymph nodes are not enlarged. Cardiovascular system: Blood pressure — 105/76 mmHg. Heart sounds are muffled. Respiratory system: respiratory rate — 24 per minute. Breathing is noisy, auscultation — small bubbling rales in the lower parts of the lungs. Urination and excretion are normal. The NEWS2 score is 9 points.

Laboratory and instrumental diagnostics

Complete blood count: Leukocytes: 25.3×10^9/l, Lymphocytes: 2%, Monocytes: 5%. Coagulation testing: D-dimer: 19.95 mcg/mL, APTT: 64.1 sec, fibrinogen: 17.9 g/L, troponin T: 2.1 ng/mL. PCR test for coronavirus infection was positive.

ECG data: abnormal Q-wave in leads II, III, aVF and V2-V6. In the same leads there were the ST segment elevation and the inversion of the T-wave.

Chest CT scan: CT evidence of viral interstitial pneumonia — CT-3 (73% of lung tissue lesions).

Despite the initiated treatment (IV nitrates, antibiotics, antiplateletes, loop diuretics, ventilator), preparation for CAG and PCI, sudden death occurred.

The immediate cause of death was pulmonary and cardiac failure due to COVID-associated pneumonitis, alveolitis and circulatory MI.

Final clinical diagnosis:

Main diagnosis: 1. New coronavirus infection, virus identified, severe degree. 2. Acute ST-Elevation Myocardial Infarction with Q-waves.

Complications: Viral interstitial pneumonia. Stage 3 respiratory failure. Acute cardiovascular failure.

Sectional examination of respiratory organs revealed the following changes: lungs with areas of

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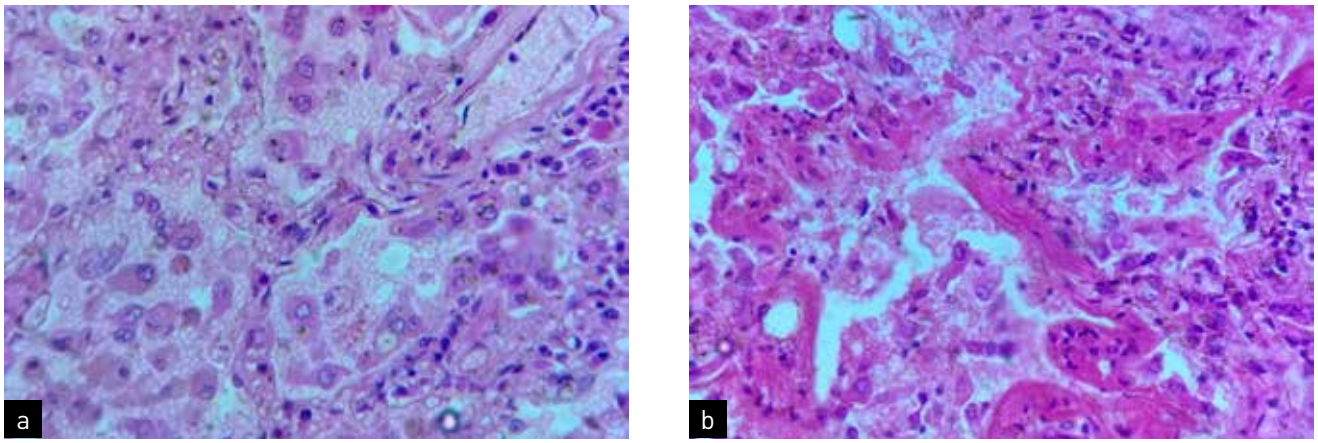

Fig. 1. Microscopic picture: a — inflammatory exudate in the lumen of alveoli, denudation of basal membranes; b — edematous fluid in the lumen of alveoli, hyaline membranes along the contour of alveoli. Hematoxylin and eosin staining, x900

uneven compaction, heavy, airless, red on section. Histological examination revealed dilated full blood vessels with perivascular sclerosis. Alveoli are unevenly distributed, their lumen is filled with serous exudate, in some places — with admixture of erythrocytes, sloughed alveolocytes and macrophages, on the walls of some of them there are deposits of homogeneous pink masses in the shape of "crescents" in the form of hyaline membranes (Fig. 1). Edematous fluid is present in the lumen of some alveoli. The interalveolar septa are thickened and sclerosed, irregularly hemorrhagic.

The following changes in the cardiovascular system were noted: in the area of the apex the myocardium is circularly flaccid with merging areas of red color, there is a slit-shaped irregularly defect with myocardial rupture in the area of the inferior and anterolateral wall of LV, myocardium at the edges of the slit is red. In the same zone there is an area of softening up to 2.6×2.7×1.2 cm. In the lumen of the anterior interventricular branch, thrombotic masses with dense adherence to the intima of the vessel are seen. Histological examination: epicardium with moderate amount of adipose tissue underneath. There is blood statis in capillaries, interstitial edema. Transverse striation of cardiomyocytes is lost, deformation and karyolysis are observed in them. Clusters of polymorphonuclear leukocytes are detected along the periphery of necrosis, forming a demarcation zone between necrotized and intact tissue (Fig. 2 a). Blood accumulation between myocytes was noted (Fig. 2 b). The result of virological examination of sectioned material (lung tissue) (Laboratory of Virological Research and Diagnostics Center of Hygiene and Epidemiology of the Chuvash Republic): SARS-CoV-2 coronavirus RNA was detected in lung, heart.

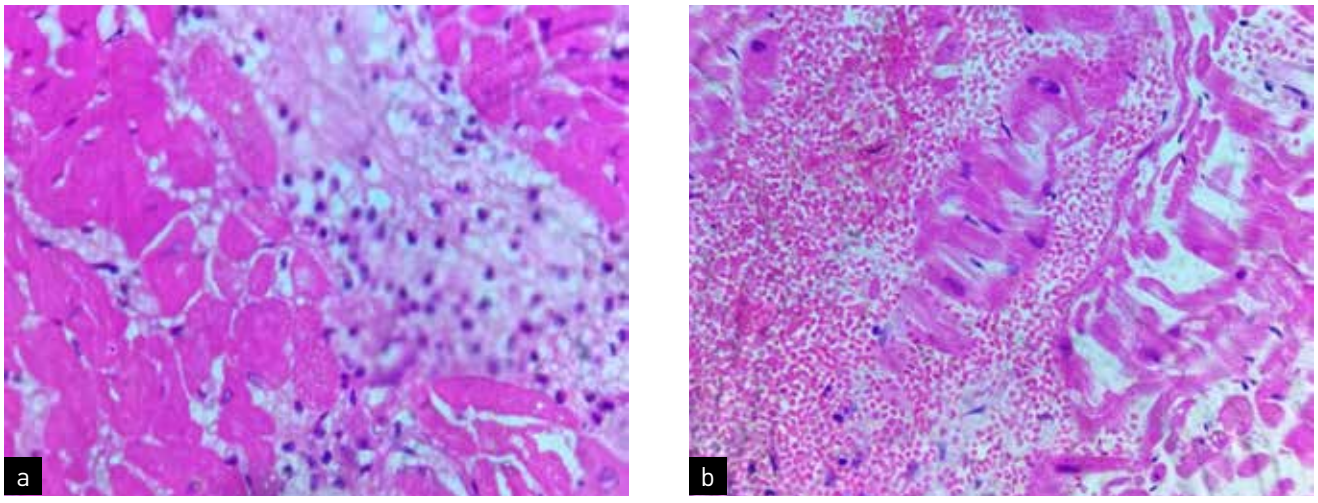

Fig. 2. Microscopic picture: a — areas of cardiomyocyte necrosis; b — accumulation of blood between myocytes. Hematoxylin and eosin staining, x900

Pathologic diagnosis (comorbid):

Main diagnosis (comorbid): 1. New coronavirus infection COVID-19, SARS-CoV-2 coronavirus RNA detected. 2. Acute circular myocardial infarction.

Complications: Viral interstitial pneumonia. Acute respiratory distress syndrome. Pulmonary edema. Rupture of the outer wall in the region of the apex. Myomalation. Hemopericardium.

Discussion

Cardiac damage after SARS-COV-2 infection is based on:

1) Vasoconstriction (due to increased angiotensin II concentration after angiotensin-converting enzyme II receptors are blocked by the virus).

2) Hypoxic state due to respiratory failure.

3) Myocardial infarction (type 1 and 2 — the ischemia due to increased myocardial oxygen demand or decreased coronary blood flow, e.g. coronary artery spasm, embolism, hypotension).

4) Due to acute viral myocarditis and "cytokine storm".

5) Thrombosis due to Covid-associated coagulopathy [9].

Thus, under the influence of SARS-COV-2, angiotensin II concentration is increased and angiotensin 1–7, which has cardioprotective properties, is decreased [8]. Angiotensin II, in turn, exerts vasoconstrictive and proatherosclerotic effects [7–9]. In addition, TNF-α expression is increased and the local and systemic inflammatory process is enhanced, leading to further myocardial damage [6–9]. Respiratory failure and generalized inflammatory process cause a mismatch between oxygen consumption and delivery to tissues with the development of hypoxia, which leads to excessive intracellular calcium accumulation and then to myocardial cell apoptosis and myocardial damage [6–8]. Direct intracellular penetration of SARS-COV-2 can also induce cardiomyocyte necrosis and myocardial destruction [6–9].

In the presented case, circular MI developed in a young patient without comorbid pathology after SARS-CoV-2 infection. There were severe complications — myomalation, ruptures of the inferior and anterolateral LV wall and hemopericardium.

Conclusion

Based on the pathological study, it was revealed that the patient's death was due to bicausal pathology represented by the main nosologies — COVID-associated pneumonitis, alveolitis with the development of acute respiratory distress syndrome and acute circular myocardial infarction. Severe complications occurred — myomalation, inferior and anterolateral LV wall ruptures and hemopericardium.

Conflict of interests: none declared.

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