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Acute Myocardial Infarction Complicating Coronavirus Infection (Case Report)

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Острый инфаркт миокарда как осложнение коронавирусной инфекции (клиническое наблюдение)

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Summary

Coronavirus infection caused by the SARS-CoV-2 virus is a multifaceted disease due to generalized vascular endothelial damage. Endothelial damage also underlies COVID-associated coagulopathy.

The paper presents a case of coagulopathy causing myocardial infarction in a 43-year-old patient with no history of coronary disease. We have reviewed the available literature for the pathophysiological rationale of the assumed possibility of coronary thrombosis resulting from coagulopathy with the intact intima of the coronary arteries.

Conclusion. The present observation of coronary thrombosis with radiographically intact coronary artery intima confirms the important role of coronavirus infection in triggering endothelial dysfunction. Currently, the most effective strategy for this type of coronary lesions is the use of anticoagulants and antiplatelet agents along with ECG, echocardiography and troponin level monitoring.

Keywords: COVID-19; covid-associated coagulopathy; myocardial infarction

Conflict of interest. The authors declare no conflict of interest.

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Introduction

Coronavirus infection is primarily a respiratory disease, therefore the new coronavirus was named SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) [1]. However, unlike «classic» community-acquired pneumonia, COVID-19 has many other targets, including cardiovascular system [2]. In particular, endothelial dysfunction and coagulation disorders are considered among the most frequent complications of coronavirus infection [3].

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In patients with COVID-19, severe manifestations such as viral pneumonia and systemic inflammation often coexist with coagulation disorders [4–6].

Proteins, glycoproteins and proteoglycans on the surface of host cells, including serine transmembrane protein 2 (TMPRSS2) and heparan sulfate proteoglycans (HSPG), are important for the initial interaction between viruses and cells [7–13]. Other proteins acting as viral receptors, such as sialic acid receptors [14, 15], matrix metalloproteinase inducer CD147 [16] and angiotensin-converting enzyme ACE2, mediate viral entry into the host cell [17]. ACE2, which is part of the renin-angiotensin-aldosterone system [18, 19], is currently the most studied receptor in the context of SARS-CoV-2 [19] and is considered one of the crucial cellular target proteins for viral infection [20]. There is evidence that the virus interacts with ACE2 through its transmembrane spike glycoprotein, which is essential for determining host cell tropism and viral diversification [5, 17, 18, 21]. The HSPG binding has also been demonstrated to cause significant conformational changes in the spike protein structure, whereas the receptor-binding domain of the spike subunit contains an HSPG binding site [22, 23]. The HSPG is a co-receptor of the cell surface proteoglycan with the ACE2 protein for recognition of the spike protein of SARS-CoV-2 [24–26]. The SARS-CoV-2 spike protein has been experimentally found to have high affinity for human ACE2 [9, 27]. The density of ACE2 in each tissue can correlate with the severity of tissue damage [28–32].

Regardless of the specific ACE2 expression loci, SARS-CoV-2 binds to the corresponding ACE2 sites wherever there is an endothelium, as it is the endothelial cells that express ACE2 [33]. Endothelial cells are fundamental to vascular endothelial function and regulate aggregation, thrombosis, fibrinolysis, and vasodilation [5, 17, 34].

ACE2 has the most extensive expression pattern in the heart, lungs, gastrointestinal system, and kidneys [32, 35]. In addition, ACE2 plays an important role in the neurohumoral regulation of the cardiovascular system. The expression of ACE2 in the brain has been suggested to contribute to the development of neurogenic hypertension [36, 37]. Binding of SARS-CoV-2 to ACE2 causes acute myocardial and lung damage by disrupting alternating ACE2 signaling pathways [35]. On the one hand, elevated ACE2 receptor density increases the viral load, but on the other hand, it can reduce the extent of cardiac damage because ACE2-induced conversion of angiotensin II to angiotensin (1–7) is a protective factor for the heart against the effects of the renin-angiotensin-aldosterone system [38]. Viral entry into the cell causes suppression of ACE2 regulation and increases systemic angiotensin II levels, resulting in increased cardiac damage [39]. Infection affects important pathways of biochemical regulation of the heart, such as ACE2 signaling pathway, fibrinogen pathway, redox homeostasis, causes stent-related plaque rupture, and finally worsens myocardial damage and dysfunction [40, 41]. Myocardial damage without direct plaque rupture can also occur due to cytokine storm, hypoxic damage, coronary spasm and endothelial or vascular damage [42, 43].

Thus, COVID-19 increases the risk of heart disease in patients with cardiovascular comorbidities [44].

Clinical case report

Patient K., 43 years old, having obesity and hypertension, was urgently admitted to Moscow City Clinical Hospital No. 52 on November 20, 2021, with a preliminary diagnosis of COVID-associated pneumonia and clinical presentation of acute coronary syndrome. On November 6, 2021, he developed a fever of up to 38°C and an impaired sense of smell. COVID-19 PCR (+) dated November 10, 2021, computer tomography (CT) of the chest dated November 20, 2021 (Fig. 1) has shown CT grade 1 pneumonia, before hospitalization he took Eliquis 2.5 mg once a day, Ibuclin, Dexamethasone, and antiviral med-

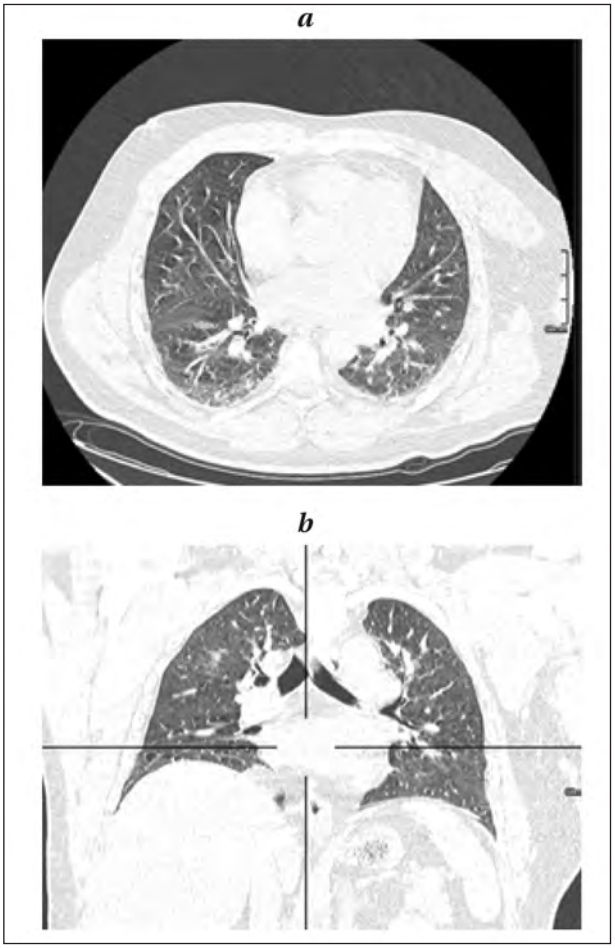

Fig. 1. Chest computed tomography dated November 20, 2021.

ications. On November 19, 2021, in the evening, the patient felt transient discomfort behind the sternum at rest, in the morning of November 20, 2021, his condition worsened, he had squeezing central chest pain and tried oral non-steroidal anti-inflammatory drugs with no effect.

On the evening of the same day, amidst persisting symptoms, he called an ambulance. The electrocardiogram (ECG) (Fig. 2, *a*) showed sinus rhythm, ST elevation in I, AVL, V2-V6, QS in V3-V6. He was diagnosed with ST-elevation acute coronary syndrome, COVID-19 infection confirmed by PCR. On admission, troponin I was 107.00 ng/L.

The patient was admitted to the intensive care unit for coronary angiography (CAG). On CAG done November 20, 2021 (Fig. 3, *a*), parietal thrombosis of left anterior descending artery (LAD) with reduced coronary blood flow was found.

Due to the parietal thrombus in LAD with reduced coronary blood flow (TIMI-II), without coronary atherosclerosis but with evidence of embolism in the terminal portion of LAD in the apical area, myocardial damage was considered due to the type 2 myocardial infarction with the underlying coagulopathy and endothelial dysfunction. We suggested that the spontaneous fibrinolysis developed after

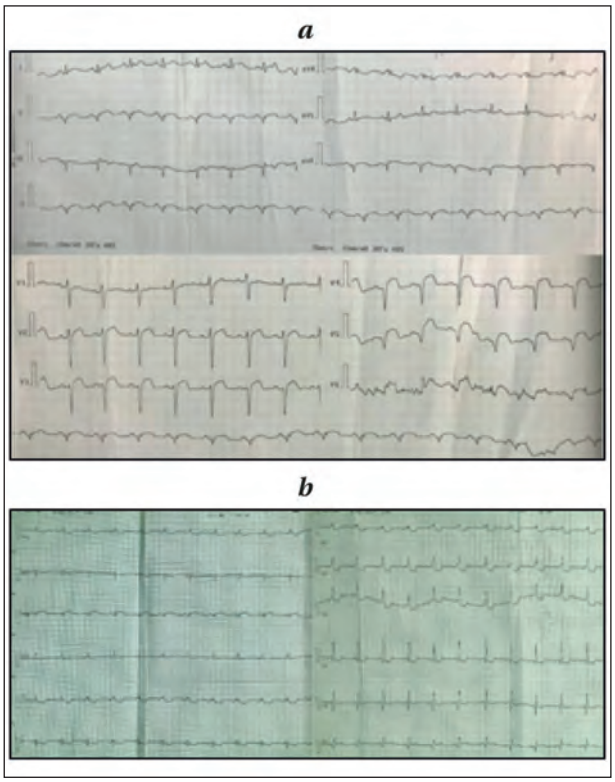

Fig. 2. ECG dated November 20, 2021 (*a*) and after rhythm restoration of November 23, 2021 (*b*).

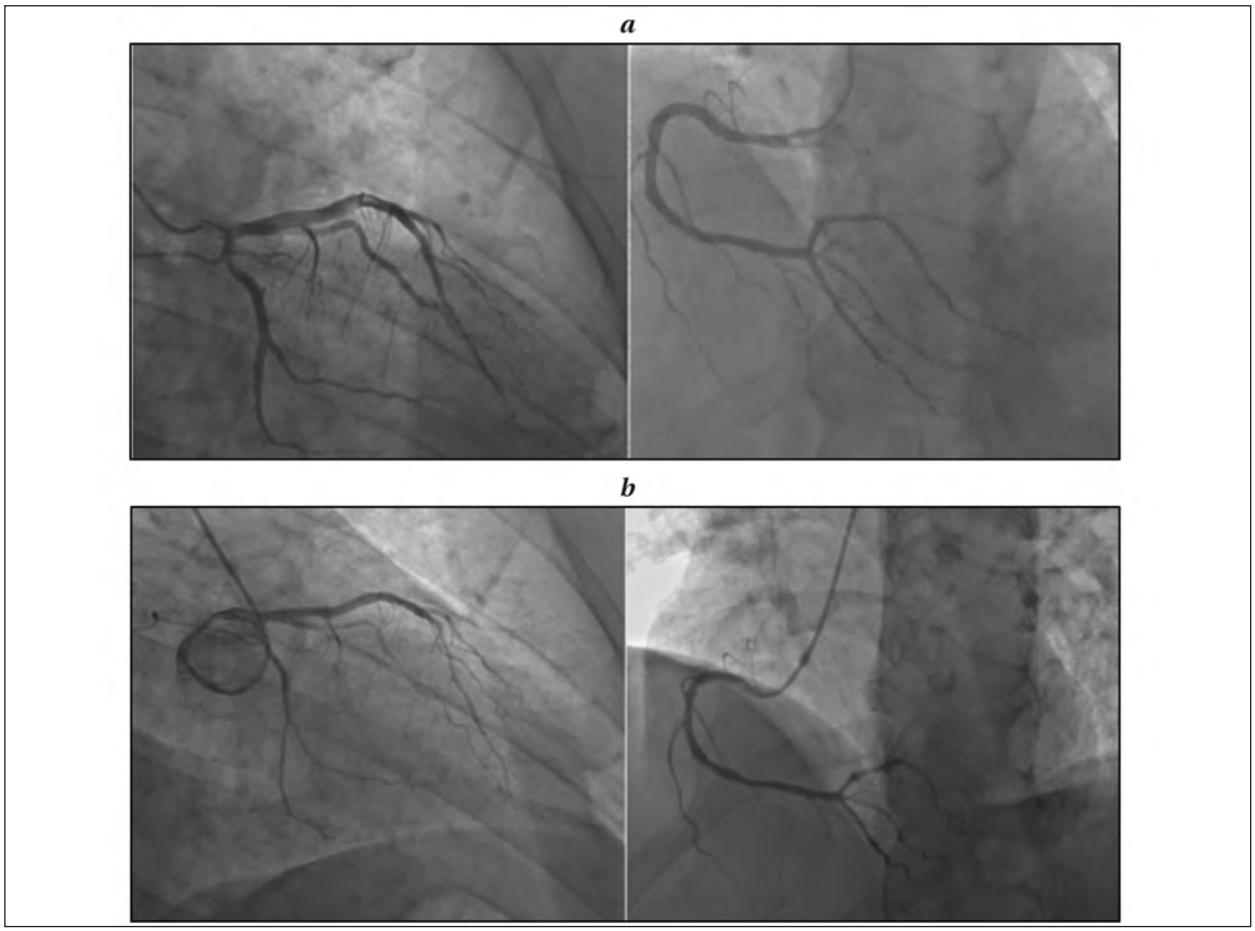

Fig. 3. Coronary angiography dated November 20, 2021 (*a*) and November 26, 2021 (*b*).

LAD thrombosis could promote embolic thrombosis with the major thrombus fragments in distal parts of LAD branches, while leaving the LAD itself relatively «intact» (angiographically seen as parietal thrombosis). This suggestion provided a rationale for administration of the IIb/IIIa receptor blocker Eptifibatide 0.75 mg/ml (100 ml) intravenously for 12 hours. In addition, dual antiplatelet therapy (acetylsalicylic acid 250 mg loading dose, then 100 mg + Ticagrelor 180 mg during percutaneous coronary intervention, Clopidogrel 600 mg loading dose, then 75 mg) was started. No coronary artery stenting was performed due to the absence of visible stenoses of the LAD. Thromboelastography (TEG) was also performed (Fig. 4) and showed normal plasma coagulation with normal clot density formation (R interval 12.6 min [reference range 9–27 min], MA 57.9 mm [reference range 44–64 mm], G 6.9 [reference range 3.6–8.5], CI 0.2 [reference range –3–+3]). The TEG results confirmed the suggested priority of endothelial dysfunction over coagulopathy per se in our case report.

The treatment was associates with improvement in patient's condition. No squeezing central chest pain was reported. Echocardiography dated November 21, 2021 has shown left ventricular ejection fraction (LVEF) ~60% with impaired local contractility of LV, circular akinesis of apex, hypo- and akinesis of middle and apical segments of septal wall, hypokinesis of basal and middle segments of lateral wall. ECG dated November 21, 2021 demonstrated ST elevation in I, II, V2-V6, abnormal Q wave in V3-V6 which was interpreted as acute myocardial infarction of anterior and lateral wall expanding to the LV apex. Troponin I of November 21, 2021 was 74.00 ng/L. 48 hours later, the ECG still showed ST elevation in I, AVL, V4-V6. Troponin I of November 22, 2021 was 36.00 ng/L.

On November 22, 2021, an atrial fibrillation paroxysm occurred, which was terminated by cardioversion within 48 hours of onset (Fig. 2, *b*). Further antiarrhythmic therapy with continuous intravenous amiodarone was administered. Antiviral and biological therapy was prescribed according to the Guidelines for prevention, diagnosis, and treatment of the novel coronavirus infection.

After stabilization, the patient was admitted to the cardiology department on November 24, 2021. A follow-up coronary angiography (Fig. 3, *b*) was performed on November 26, 2021 due to the presence of coronary heart disease, myocardial infarction, contraindications for exercise testing and to assess the coronary artery patency and determine the management strategy. Positive changes were noted compared to the one of November 20, 2021: the left coronary artery (LCA) was intact, the right main coronary artery (RCA) had no hemodynamically significant stenosis, in the distal part (apical region) there was a slight delay in contrast agent passage; the left circumflex artery (LCA), obtuse marginal artery (OMA), and right coronary artery (RCA) had no hemodynamically significant stenosis.

The follow-up Holter ECG monitoring of November 27–28, 2021 has shown sinus rhythm with episodes of rapid atrial fibrillation and short runs of ventricular tachycardia. The follow-up chest CT scan of November 29, 2021 showed improvement compared to the one made on November 20, 2021.

The patient was discharged from the hospital in a stable condition (normal temperature, reduced markers of systemic inflammation) on day 11 after admission.

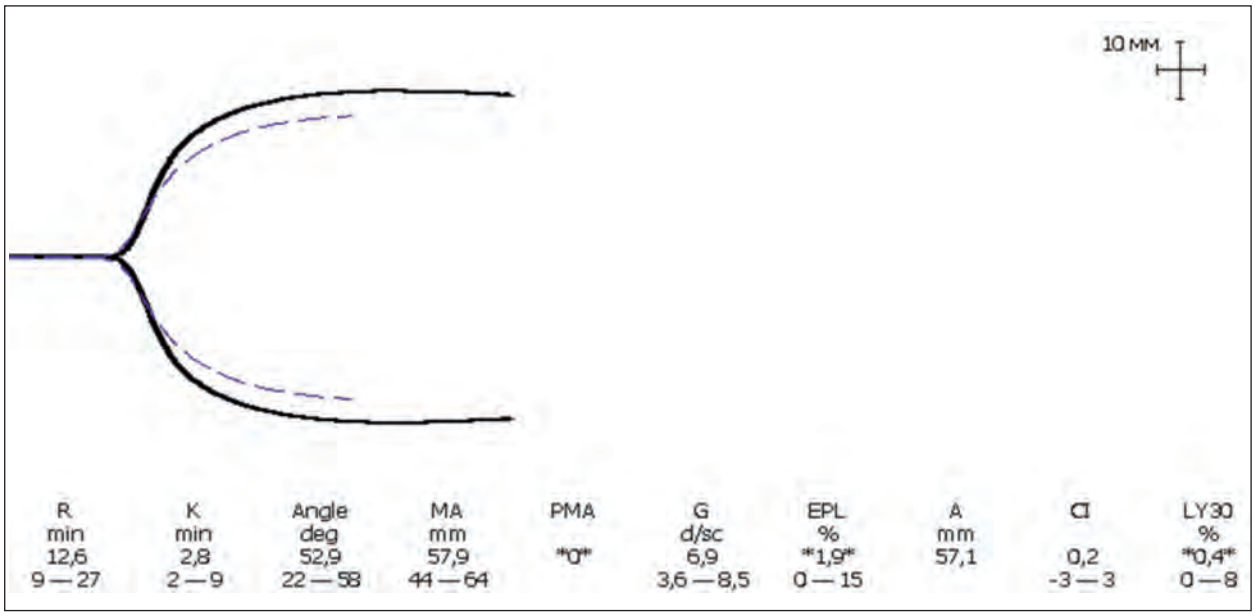

Fig. 4. Thromboelastography dated November 20, 2021.

Conclusion

This case report supports the important role of coronavirus infection in triggering endothelial dysfunction in coronary thrombosis with radiologically intact coronary artery intima. Currently, anticoagulant and antiplatelet therapy with ECG, echocardiographic and troponin level monitoring remain the most effective management strategy for this type of coronary lesion. Many issues of COVID-associated coagulation disorder and endothelial damage, which determine non-atherosclerotic coronary thrombosis, are still poorly understood. The phenomenon of spontaneous fibrinolysis with the underlying systemic COVID-associated hypercoagulation also remains unclear. These issues require further study.

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