



Case Report

ST-elevation myocardial infarction in a 39-year-old patient with “normal” coronary arteries as a thrombotic complication of COVID-19



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ABSTRACT

We report the case of a 39-year-old male without traditional risk factors for coronary artery disease (CAD), i.e. smoking, hypercholesterolemia, hypertension, diabetes mellitus, familial history of premature CAD, admitted with anterior ST-segment elevation myocardial infarction and concurrent coronavirus disease-2019 infection. Coronary angiography showed high intracoronary thrombus burden and thrombotic occlusion of the proximal segment of left anterior descending artery, while optical coherence tomography revealed intact endothelium after thromboaspiration.

<Learning objective: Coronavirus disease-2019 (COVID-19) may predispose to thrombotic complications in both the venous and the arterial circulation. ST-segment elevation myocardial infarction (STEMI), rarely, may be the main clinical presentation of COVID-19. STEMI in patients with concurrent COVID-19 may be caused by thrombotic coronary occlusion even in the setting of “normal” coronary arteries.

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Introduction

Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) causes coronavirus disease-2019 (COVID-19), was first recognized in China and has been characterized as a pandemic by the World Health Organization. Among the various consequences of COVID-19, coagulation abnormalities are common in both the venous and the arterial circulation [1]. Moreover, current evidence suggests possible pathophysiological links between SARS-CoV-2 and acute coronary syndromes, with evidence indicating higher intracoronary thrombus burden in COVID-19 patients presenting with ST-segment elevation myocardial infarction (STEMI) [2]. We herein report the case of a 39-year-old male admitted with anterior STEMI and concurrent COVID-19 infection, with high intracoronary thrombus burden and thrombotic occlusion of the proximal segment of left anterior descending (LAD) coronary artery, in whom optical coherence tomography (OCT) revealed intact endothelium after thromboaspiration.

Case report

A 39-year-old Caucasian male confirmed positive with COVID-19 for the past week and being asymptomatic at home, attended

the emergency department complaining of retrosternal pain for the past hour. On presentation his blood pressure was 135/80 mmHg, heart rate was 90 bpm, oxygen saturation was 98% on room air, while he had no fever. There was nothing remarkable from the physical examination.

The patient had no conventional risk factors for coronary artery disease (CAD), i.e. smoking, hypercholesterolemia, diabetes mellitus, hypertension, or familial history of premature CAD. He did not use any recreational drugs or substances.

A 12-lead electrocardiogram revealed ST-segment elevation in leads V4–V6, indicative of an anterior STEMI (Fig. 1). An echocardiogram showed anteroapical hypokinesis with moderately reduced left ventricular systolic function (ejection fraction ~45%). The patient was immediately transferred to the catheterization laboratory with the use of full personal protective equipment.

Coronary angiography (CAG) showed a contrast filling defect suggestive of thrombus formation and subsequent occlusion of the proximal segment of LAD (Fig. 2a). Right coronary artery and left circumflex artery were normal. An aspiration catheter was subsequently used in LAD, and a high burden of red thrombus was aspirated with achieved Thrombolysis in Myocardial Infarction (TIMI) flow 3, without stent implantation (Fig. 2b). No residual filling defects were observed.

The patient was transferred to the coronary care unit in a single chamber designated for COVID-19 patients. Laboratory tests demonstrated peak high-sensitivity troponin T levels 1664 pg/mL, total cholesterol 102 mg/dL, low-density lipoprotein-cholesterol 62 mg/dL, high-density lipoprotein-cholesterol 27 mg/dL, triglyc-

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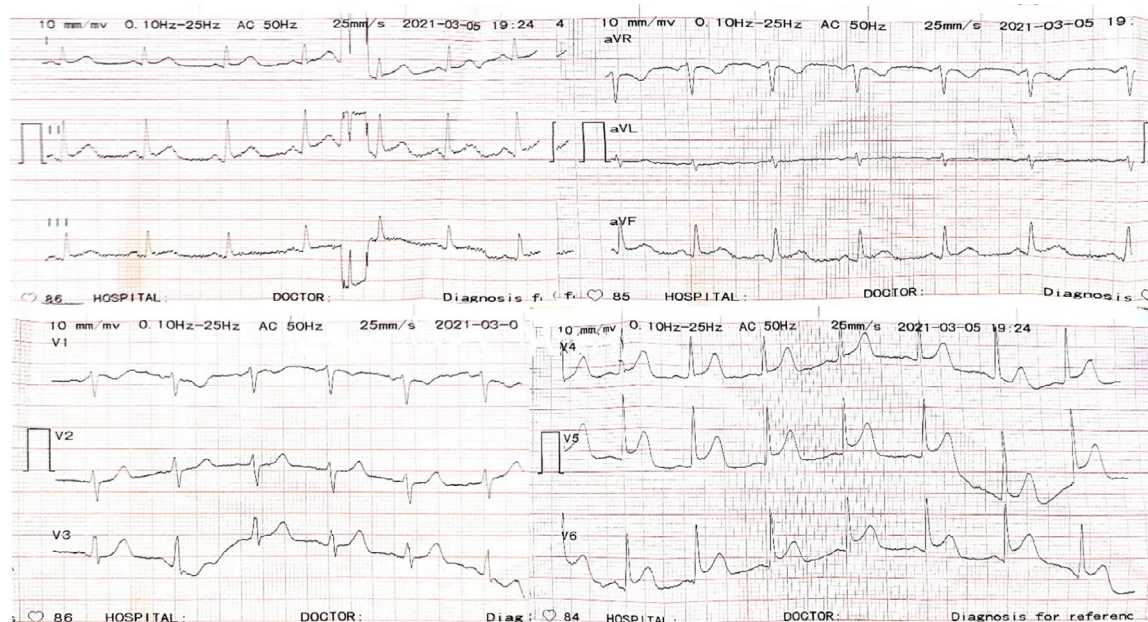


Fig. 1. Electrocardiogram showing ST-segment elevation in precordial leads V4-V6.

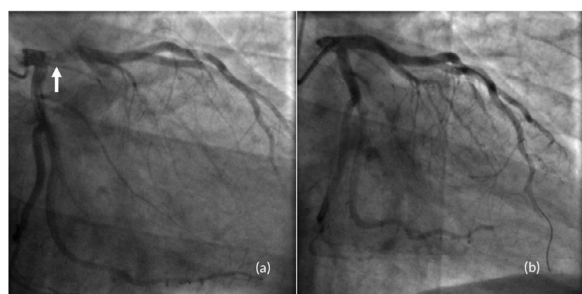


Fig. 2. Coronary angiography showing (a) a filling defect (white arrow) suggestive of large intracoronary thrombus in proximal left anterior descending coronary artery (b) complete flow restoration in left anterior descending coronary artery after thromboaspiration.

erides 176 mg/dL, lipoprotein(a) 10.4 mg/dL, homocysteine 9.4 μ mol/L, D-dimers 1473 ng/mL, C-reactive protein 62 mg/L, and interleukin-6 (IL-6) 17 pg/mL. Immunology for antiphospholipid antibodies and the lupus anticoagulant were negative and levels of protein C, protein S, and antithrombin III were also normal. The nasopharyngeal swab was positive for SARS-CoV-2 by real-time reverse-transcriptase-polymerase-chain-reaction assay.

Apart from dual antiplatelet therapy with aspirin and ticagrelor, our patient received intravenous IIb/IIIa inhibitor (tirofiban) for 24 h and subcutaneous low molecular weight heparin (LMWH) in therapeutic dose for 5 days, i.e. enoxaparin 80 mg twice daily. Three days after his admission a repeat CAG showed almost normal coronary arteries, with LAD TIMI flow 3 (Fig. 3a). OCT was performed showing an intact endothelium, with no evidence of erosion or rupture of an atherosclerotic plaque (Fig. 3b). However, there was a lipid-rich plaque from 11 o'clock to 5 o'clock overlaid by an intact fibrous cap.

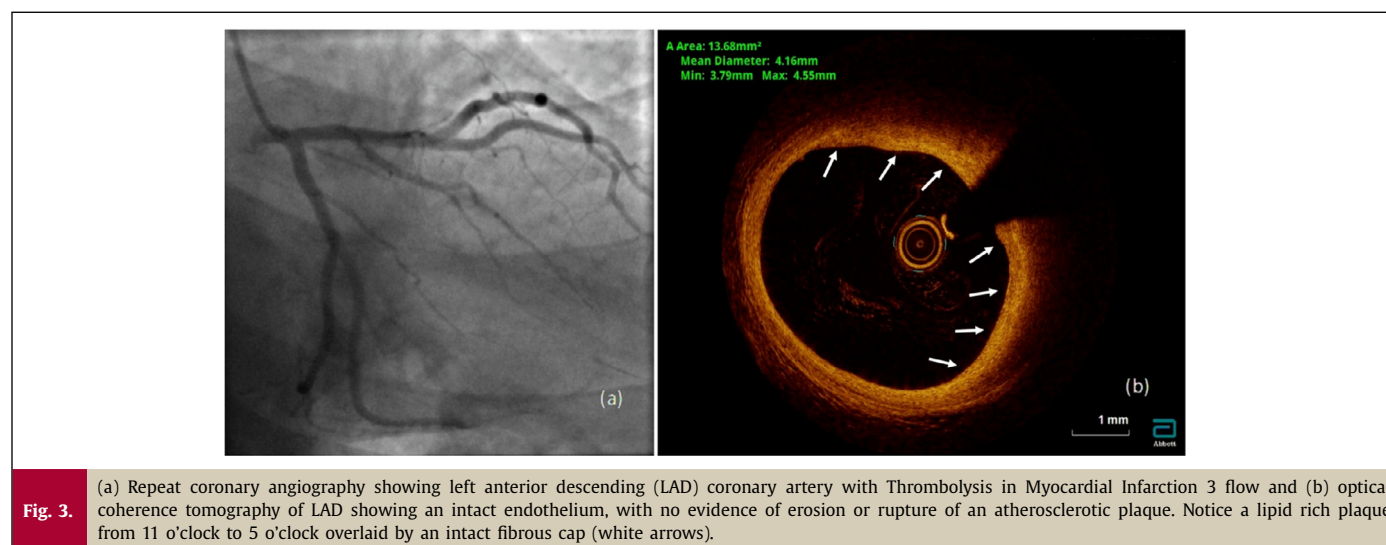
According to the instructions of infectious diseases specialists, the patient was also treated with remdesivir (100 mg IV daily) for 5 days. During his hospitalization, he underwent a chest computed

tomography that revealed bilateral ground glass opacities occupying approximately 15% of the pulmonary parenchyma, while computed tomography pulmonary angiography was negative for pulmonary embolism. The patient had an uneventful recovery and he was discharged after 5 days on dual antiplatelet therapy, LMWH on prophylactic dose for 2 weeks, metoprolol 25 mg bid, lisinopril 5 mg daily, and atorvastatin 40 mg daily.

Discussion

Although COVID-19 mostly presents as respiratory distress, research has demonstrated an increased thrombotic risk associated with poorer outcomes [1]. This pro-thrombotic state may present as pulmonary embolism [3], deep vein thrombosis, cerebral infarctions [4], or venous sinus thrombosis [5]. STEMI with intracoronary thrombus as the only or the first manifestation of COVID-19 is relatively rare and usually affects patients with diabetes mellitus, hypertension, and smoking history [2]. To our knowledge, our case is unique because it describes a patient with STEMI possibly due to thrombotic complication of COVID-19. This assumption is reinforced by the lack of traditional risk factors for CAD, the presence of almost normal coronary arteries, and the negative laboratory investigation for thrombotic disorders.

Mechanisms that cause high thrombus burden in COVID-19 patients are yet unknown. As with venous thromboembolism, arterial thrombus is thought to be due to endothelial dysfunction and platelet activation, as a result of the profound inflammatory response [6]. This pro-inflammatory state stems from the production of inflammatory cytokines, tumor necrosis factor- α , IL-6, and IL-1, resulting in a cytokine storm. This leads to activation of coagulation, with thrombin as the central component, predisposing to platelet activation and development of thrombosis. Therefore, we hypothesize that fibrous plaque erosion was the most likely pathophysiological substrate of the STEMI triggering an excessive thrombotic response in the setting of concomitant “thrombogenic” COVID-19 infection. Three days later, OCT showed an intact endothelium due to the healing process facilitated by the intensive antiplatelet therapy.



Furthermore, it is well known that the SARS-Cov-2 uses angiotensin-converting enzyme 2 (ACE-2) receptors as an entry point into human cells. ACE-2 receptors might have a role in endothelial cell activation and dysfunction since they are expressed not only on pneumocytes but also on endothelial cells [1].

Antiphospholipid antibodies have been also detected in COVID-19 patients, the presence of which may lead to thrombotic events [7]. These antibodies support the diagnosis of antiphospholipid syndrome; however, they also arise transiently in critical infections [8].

In small studies among STEMI patients with COVID-19, higher biological markers of inflammation (C-reactive protein), fibrinolysis (D-dimers), and antiphospholipid antibodies were observed, compared with COVID-19 negative patients [9]. Interestingly, median peak high-sensitive troponin may be greater in COVID-19 positive STEMI patients [2]. When it comes to procedural characteristics, there is a significantly greater use of aspiration thrombectomy and subsequent use of glycoprotein IIb/IIIa inhibitors in COVID-19 patients [2]. The combination of systemic inflammation and hypercoagulation in this population may be associated with poorer outcomes and increased mortality rate [9].

Conclusions

Evidence suggests that emerging COVID-19 infection is strongly associated with a pro-thrombotic state, while the presentation of STEMI might itself be a thrombotic complication of COVID. The use of anticoagulation may benefit this patient group, however further studies are required to establish the exact mechanism of coronary thrombosis in COVID-19 patients and the optimal duration of anticoagulation treatment. Our case indicates that COVID-19 can rarely predispose young patients with no comorbidities and almost normal coronary arteries to acute thrombotic coronary events while

having no respiratory distress symptoms. The impact of the infection on early and long-term outcomes in STEMI patients should be further investigated.

Declaration of competing interest

The authors declare that there is no conflict of interest.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at [doi:10.1016/j.jccase.2021.12.006](https://doi.org/10.1016/j.jccase.2021.12.006).

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