Case Report

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Specific Clinical Case in the Course of Covid-19 Infection

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ABSTRACT

Description of a specific clinical case that contributes to the elucidation of COVID-19 INFECTION effects on patients and shows some very unusual phenomena. This clinical case demonstrates the unique prothrombotic properties of SARS-Cov-2 that are due to different mechanisms, and which can be complicated by thromboembolic events.

Keywords: Pandemic; Hypercoagulation; Acute Coronary Syndrome; Ischemic Stroke

Introduction

COVID-19 exercised a global impact on public health and the provision of health care. Due to the unexpected necessity of a sufficient number of hospital beds for intensive care with opportunities for respiratory support and mechanical ventilation, a temporary redistribution and reorganization of the hospitals was required with the ensuing consequences for all medical specialties. The health care system was strongly affected by the COVID-19 pandemic, and the larger part of resources was diverted toward the management of that particular disease. A great number of hospital wards were reorganized for patients with COVID-19, which restricted the access to health care of patients with chronic conditions, including with acute coronary syndrome (ACS) [1-5]. SARS-Cov-2 affects seriously not only the respiratory system with a tendency of developing viral pneumonia and Respiratory Failure (RF), but also the cardiovascular one. Patients with risk factors for Cardiovascular Disease (CVD), such as male gender, advanced age, hypertension, and obesity, have been identified as a particularly vulnerable population with increased incidence and mortality rates when infected with SARS-Cov-2. SARS-Cov-2 penetrates the cells by attaching to the spike-protein of the receptor of the angiotensin-converting enzyme 2 (ACE2) on their surface. The excessive production of pro-inflammatory cytokines, the so-called cytokine storm, leads to multiple organ failure. SARS-Cov-2 causes disorders of the coagulation cascade leading to thromboembolic complications.

Description of the Clinical Case

We present the clinical case of a 53-year-old man who came to the emergency care unit with manifested toxico-infectious syndrome: chills, fever up to 39o C, and adynamy, with cough without expectoration. Prior to being hospitalized at our department, the patient was treated in out-patient settings with an immunomodulator, an antibiotic, and vitamins. On the fourth day after the onset of symptoms, the patient experienced chest pain at rest, which had an intermittent nature and lasted for several hours. On the 6th day, dyspnea occurred, the patient's state deteriorated progressively, and he sought urgent medical help. The patient did not report having any other previous conditions, family history of cardiovascular disease, or risk factors for cardiovascular diseases. Upon physical examination, the patient presented in a deteriorated overall state, intoxicated and sweating, febrile, with pronounced tachypnea and dyspnea, and a respiratory rate of 26/min. The oxygen saturation was 86% at atmospheric air, the breathing was vesicular with fine moist crackles and groups of crackles in both lungs, the heartbeat was rhythmic and with normal frequency (70 beats/min), with absence of pathological heart murmurs and sounds, the arterial pressure was 140/95 mm Hg. The rest of the organs and systems presented normal findings upon physical examination. The 12-lead ECG registered sinus rhythm, heart frequency of 70 beats/min., left type of electrical axis, incomplete

RBBB with ischemic T-waves in the precordial leads, as well as Q-spikes from V2 to V5, PR 192 ms, QRS 72 ms, QTc 489 ms (Figure 1).

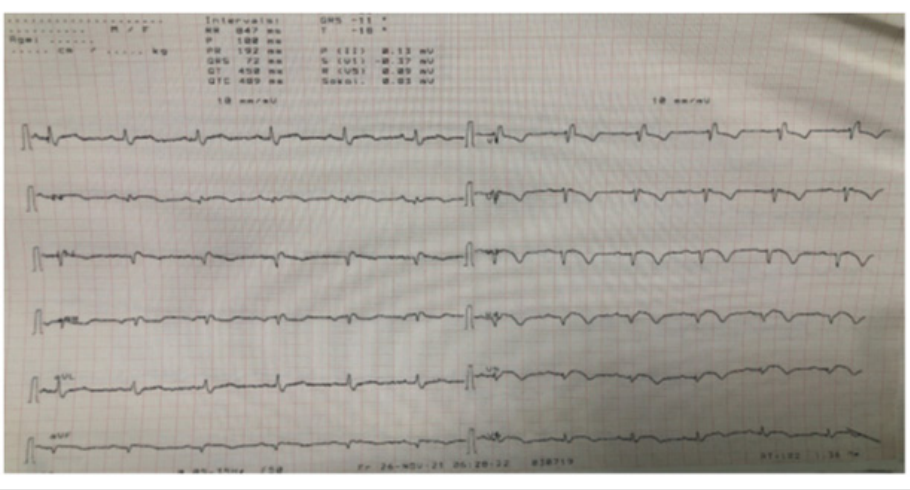

Figure 1: 12-lead ECG. Sinus rhythm, heart frequency 70 beats/min., left type, incomplete RBBB with ischemic T-waves in the precordial leads, as well as pathological Q-spikes from V2 to V5, PR 192 ms, QRS 72 ms, QTc 489 ms.

Paraclinical Tests: WBC: 12 G/L; Neu: 10.9, Ly #: 0.5 G/L; Ly (%): 4%; Hgb: 138 g/l; RBC: 4.69 T/L; Hct: 40.9%; PLT: 588 G/L; CRP: 105 mg/L; Creatinine: 71 mmol/l; D-dimer: 1,067 ng/m; CK: 1,964 U/l; CK-MB: 257 U/l; Tn 1 > 26.6 ng/ml; Cholesterol: 3.6 mmol/l; LDL: 2.75 mmol/l; HDL: 0.63 mmol/l. The rapid antigen test for SARS-Cov-2 was negative.

Diagnostic Imagery: The CT scan of the lungs revealed evidence of bilateral interstitial pneumonia associated with a SARS-Cov-2 infection with a moderate degree and at a late stage of development with formation of fibrous strands (Figure 2). Because of the highly elevated enzymes indicative of myocardial necrosis, and against the backdrop of angina symptoms and the ECG changes, the patient was hospitalized at a cardiology intensive care unit. He was placed an oxygen mask with a concentration of 10 l/min, which improved the O2 saturation to 95%, and was treated with a saturation dose antiaggregants, a statin, and a bolus of unfractionated heparin (UFH) based on the patient's weight. The echocardiography revealed slightly reduced systolic LV function with evidence of apical thrombosis 16/16 mm in size, preserved diastolic function, light-degree mitral insufficiency, preserved longitudinal RV systolic function without signs of pulmonary arterial hypertension (Figure 3). In the course of the hospitalization, the patient had recurrent angina symptoms, fever, and signs of respiratory failure. A RT-PCR test was conducted, which turned out positive.

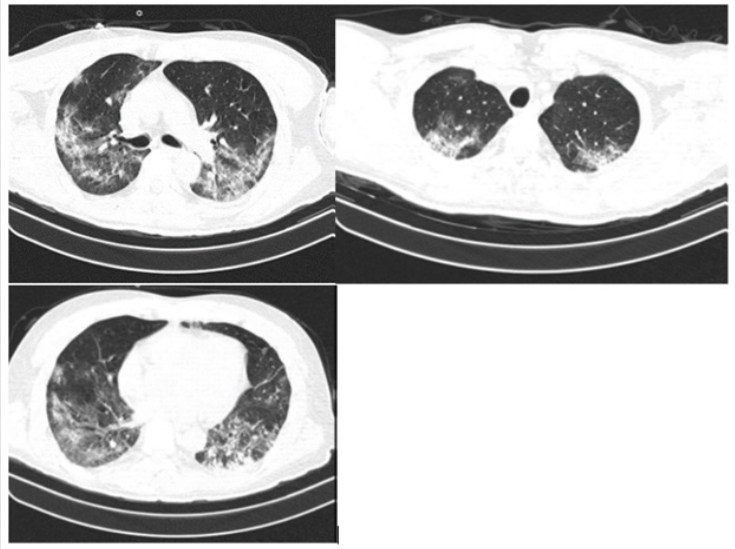

Figure 2: CT scan of lungs.

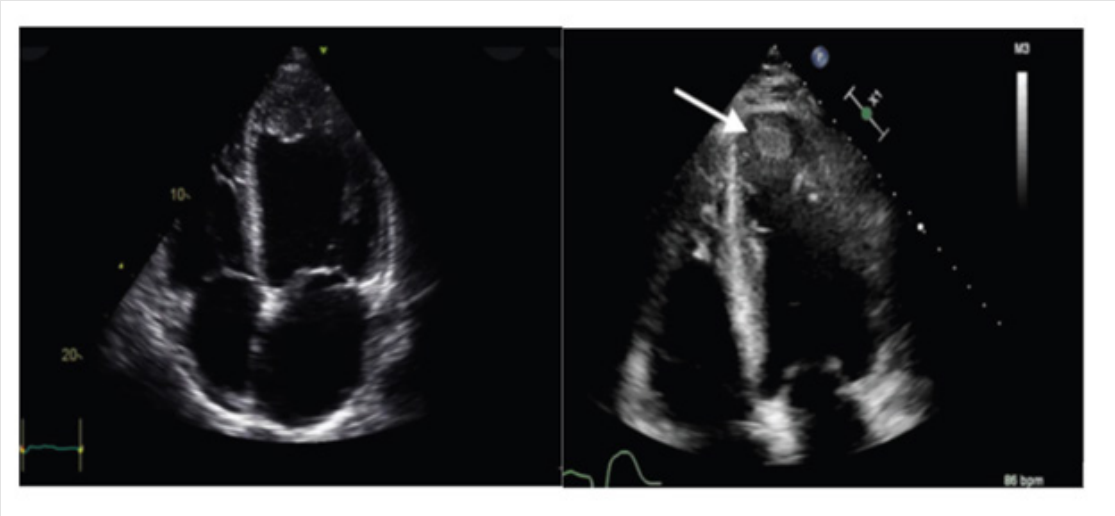

Figure 3: Transthoracic 2D echocardiography in apical 4-cavity section: there is evidence of apical thrombosis 16/16 mm **in size.**

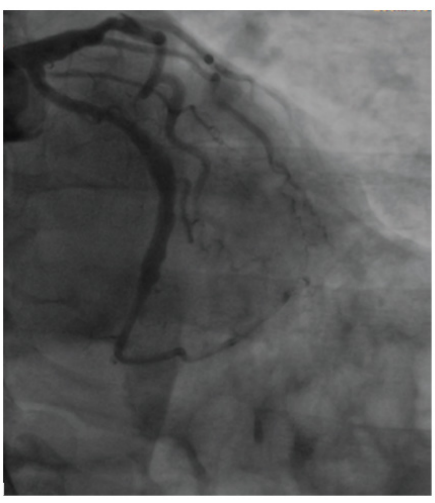

Figure 4: Unstable plaque in the proximal segment of LAD**.**

A selective coronary angiography was performed with strict respecting of all antiepidemic measures. The SCAG revealed evidence of right type coronary circulation, LM without stenoses, LAD with unstable plaque in the proximal segment with overlapping massive non-obstructive thrombosis engaging the bifurcation of D1 (medina 1-1-0), LCx with aneurysmally altered middle third of the artery without significant stenoses, RCA –ectasic vessel without significant stenoses. The ostium of LCA was cannulated with GC EBU 3.5/6 Fr, a 0.014 guide was placed in the vessel's periphery, and a pPCI was performed on LAD with direct implantation of two drug-eluting stents (4.0/30 mm and 4.5/13 mm) with subsequent post-dilation with a balloon from the second stent at the place of overlapping. An optimal final angiographic result was achieved (Figures 4-7). Against the backdrop of initiated infusion therapy with UFH based on APTT, coupled with a vitamin K antagonist, and dual antiaggregant and statin therapy, anti-inflammatory treatment was also started with a corticosteroid based on the patient's weight: colchicine 2 x 0.5 mg, infusions of water-saline solutions, low-dose diuretic therapy with monitoring of the central venous pressure. Early treatment with a beta-blocker titrated to maximum doses, and an ACE-inhibitor was initiated. After stabilizing the patient's state, on the 4th post-procedural day, he was discharged due to refusal to continue the treatment at a specialized unit for treatment of COVID-19 patients.

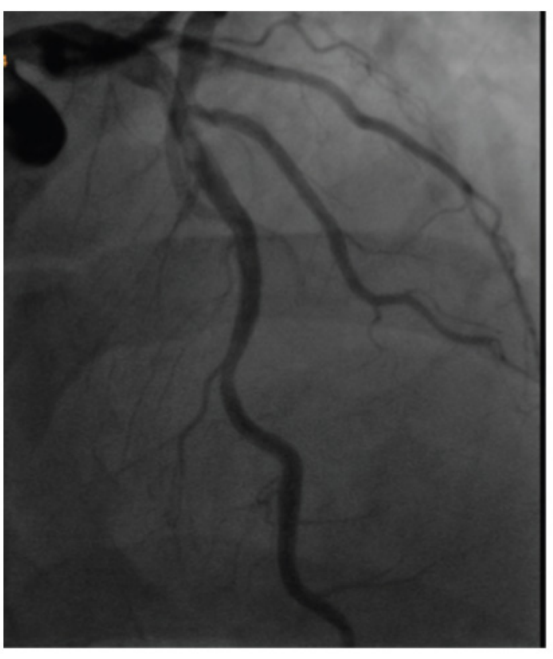

Figure 5: Massive non-obstructive thrombosis**.**

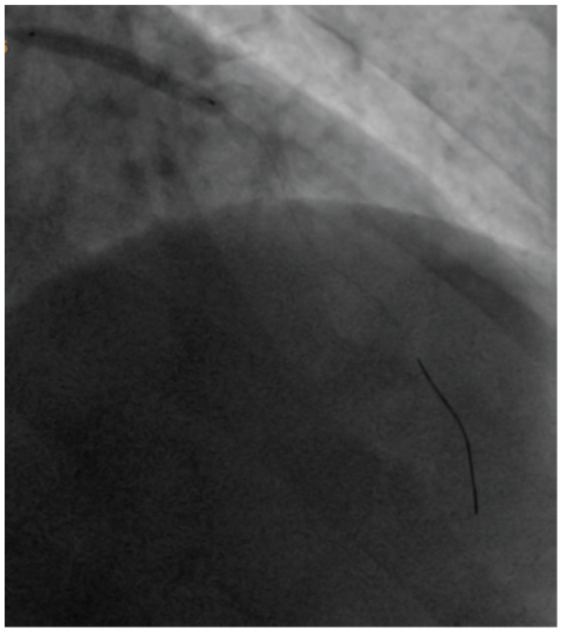

Figure 6: pPCI on LAD with direct implantation of two drug-eluting stents: 4.0/30 mm and 4.5/13 mm and subsequent post-dilation with a balloon from the second stent.

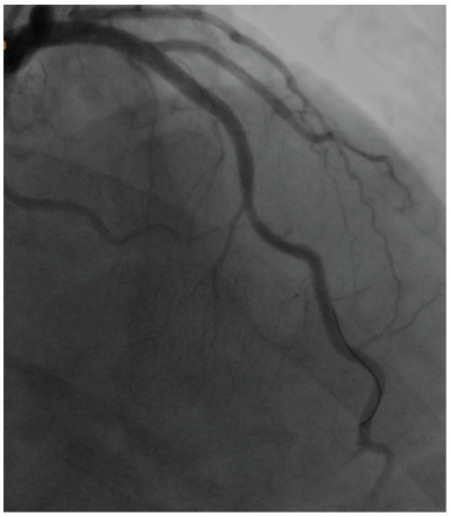

Figure 7: Optimal final angiographic result.

On the second hour upon the dehospitalization, his relatives noted that he was aphasic, and later on with right-hand side paralysis. They sought medical help at an emergency unit yet again. A CT scan of the brain was performed, which revealed evidence of an ischemic stroke in the basin of the left middle cerebral artery against the backdrop of optimum dosage of anticoagulant. The patient was hospitalized at a specialized intensive care unit for COVID-19 patients. Nootropic medications were added to the treatment. The patient was treated at a COVID-19 sector for intensive care for 26 days. He was discharged on the 27th day with residual right-hand side paralysis and was referred to a rehabilitation centre. The evidence of interstitial inflammatory changes in the follow-up CT scan of the lungs was in the stage of resorption and fibrosis; the patient was non-febrile and had no signs of respiratory failure or angina symptoms. On the third month upon his dehospitalization, the patient came for a follow-up visit. He was in visibly improved state without any residual neurological symptoms and angina complaints.

Discussion

Are the events of the SARS-Cov-2 infection and the thrombosis of a coronary vessel with subsequent intraventricular thrombosis a mere coincidence? Our clinical experience shows that the angiographic and echocardiographic studies reveal a different type of thrombogenesis that does not follow the pattern of the standard infarction and left-ventricular thromboses. We suppose, with a great degree of likelihood, that the COVID-19 infection provokes the occurrence of vascular events by its high pro-inflammatory and thrombogenic risk. Motivating the population to get vaccinated would lead to expected lower incidence, lower risk of complications, and lower mortality rates, respectively. The management of patients infected with SARS-Cov-2 by a multidisciplinary team comprising a pulmonologist, a cardiologist, an infectious diseases and an intensive care specialist at intensive care units equipped with the required resources in the event of complications would lead to a more favourable disease course and to reduction in the in-house mortality rates, as well as in the number of cases of cardiac arrest outside health care facilities. A US study based on the data of US' most comprehensive health care system (VHA) proves that COVID-19 survivors are at an increased risk of developing a number of cardiovascular and cerebrovascular complications. Despite the severity of the disease course, the risk of brain stroke in the following 12 months increases by 48% compared to the group of patients non-diagnosed with COVID-19. In these patients, the risk of developing rhythm disorders in the next 12 months increases by 66%. The risk of acute myocardial infarction in the following 12 months increases by 61%. The risk of developing heart failure increases by 71% in comparison with the people who have never been ill with COVID-19. The study reports that COVID-19 survivors are at a 92% increased risk of thromboembolic episodes in the next 12 months [6].

Conclusion

COVID-19 is a unique prothrombotic condition determined by various mechanisms, whose complications lead to thrombotic accidents. An increased rate of venous and arterial thromboses, including acute myocardial infarction, has been observed in COVID-19 patients. Caution is required for the development of cardiovascular events, such as acute coronary syndrome and thromboembolic events, when dealing with a SARS-Cov-2 infection, as well as a careful approach to risk assessment in people with existing cardiovascular disease. Patients with risk factors for CVD, such as advanced age,

diabetes mellitus, hypertension, and obesity, as well as patients with diagnosed cardiovascular disease and cerebrovascular disease, have been identified as particularly vulnerable populations with increased incidence and mortality rates when infected with COVID-19. During the acute phase of the SARS-Cov-2 infection, vital organs and systems are affected, i.e. the respiratory, cardiovascular, and cerebrovascular systems, with a high potential for developing long-lasting consequences that require long-term monitoring and therapeutic control.

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