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COVID-19: Simultaneous Thrombosis of Two Coronary Arteries

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Abstract

Since the first documented outbreak of a novel severe acute respiratory syndrome inducing Coronavirus in China at the end of 2019, the virus has spread to all continents. Multiple authors have reported an increased rate of thrombo-embolic events in affected patients. 31-year-old male, without any cardiovascular risk factor admitted to hospital with an elevation of Troponin I after seven days of coronavirus diagnosis. Cardiac magnet resonance imaging revealed the presence two infarcts and coronary angiography showed thrombus in right coronary and left anterior descending arteries. We present a serious acute cardiac event in the context of a paucisymptomatic Coronavirus infection.

Keywords: COVID-19 • Acute coronary syndrome • Thrombosis

Introduction

In February 2020 the World Health Organization officially termed coronavirus disease 2019 (COVID-19). Since the first documented outbreak of a novel severe acute respiratory syndrome inducing Coronavirus in China at the end of 2019, the virus has spread to all continents, becoming a pandemic and causing the most serious world health emergency [1].

Previous studies have linked acute infections with an increased risk for acute myocardial infarction [2]. The overall incidence of acute cardiac injury has been variable but roughly 8-12% of the positive cases are known to develop significant elevation of Troponin [3]. We present a serious acute cardiac event in the context of a paucisymptomatic Coronavirus infection.

Methods

We describe the case of a 31-year-old, non-smoker, male on the seventh day after diagnosis of COVID-19 collecting nasopharyngeal swabs for polymerase chain reaction.

Results

The patient was admitted with symptoms of myalgia, weakness, chest pain unrelated to exercise and sweating. No fever. No cough. The patient had no comorbidities. On arrival, electrocardiogram was normal. On admission, vital signs were blood pressure, 119/60 mmHg; heart rate, 103 bpm; weight, 82kg; height, 180cm, and body mass index, 25,30. Baseline oxygen saturation was 97%. Laboratory tests showed high troponin (1,05), as well as high ck-mb (12,7) and normal C-reactive protein(0,03),D-dimer(400),brain natriuretic peptide(43). The diagnosis of myocarditis of viral etiology by COVID-19 was considered, and a cardiac magnet resonance imaging requested for confirmation one day after hospital admission.

The cardiac magnet resonance revealed the presence two infarcts: mid-ventricular anterior wall and basal inferior wall (Figure 1). Further diagnostics with cardiac magnet resonance, the cardiac catheterization was arranged and showed: thrombotic content in the right coronary artery and left anterior descending arteries, with no impaired blood flow (Figures 2 and 3). We prescribed dual anti-platelet therapy and an angiotensin-converting-enzyme inhibitor. After two negative COVID-19 swab results in over 24 h and absence of typical symptoms, the patient was no longer considered infectious and could be assigned to a cardiac rehabilitation program.

COVID-19 is caused by a novel enveloped RNA beta- coronavirus. Seven species of these beta-coronaviruses are known to cause human infections, with four mainly causing mild flulike symptoms and the remaining three resulting in potentially fatal illnesses. Although respiratory tract is the primary target for COVID-19, cardiovascular system may get involved in several different ways. Following are the common mechanisms responsible for CV complications in COVID-19:

Direct myocardial injury-COVID-19 enters human cells by binding to angiotensin-converting enzyme 2 (ACE2), a membrane bound amino peptidase which is highly expressed in heart and lungs. ACE2 plays an important role in neuro humoralregulation of CV system in normal health as well as in various disease conditions. The binding of COVID-19 to ACE2 can result in alteration of ACE2 signaling pathways, leading to acute myocardial and lung injury [4].

Systemic inflammation - More severe forms of COVID-19 are characterized by acute systemic inflammatory response and cytokine storm, which can result in injury to multiple organs leading to multiorgan failure. Studies have shown high circulatory levels of proinflammatory cytokines in patients with severe/critical COVID-19 [5].

Altered myocardial demand-supply ratio- Increased cardiometabolic demand associated with the systemic infection coupled with hypoxia caused by acute respiratory illness can impair myocardial oxygen demand-supply relationship and lead to acute myocardial injury.

Plaque rupture and coronary thrombosis- Systemic inflammation as well as increased shear stress due to increased coronary blood flow can precipitate plaque rupture resulting in acute myocardial infarction. Prothrombotic milieu created by systemic inflammation further increases the risk.

Acute myocardial injury is the most commonly described CV complication in COVID-19. Abnormal coagulation parameters also correlate with disease severity [4]. The pro-thrombotic effects of COVID-19 are currently under investigation and could be enhanced by immune system hyperactivity with cytokine storm and pronounced local and systemic inflammatory reactions [6].

In a systematic review including 374 patients, cardiac troponin I levels were significantly higher in those with severe COVID-19 infection compared with those non-severe disease [7]. Cardiac injury has been reported from 8% to 12% [3].

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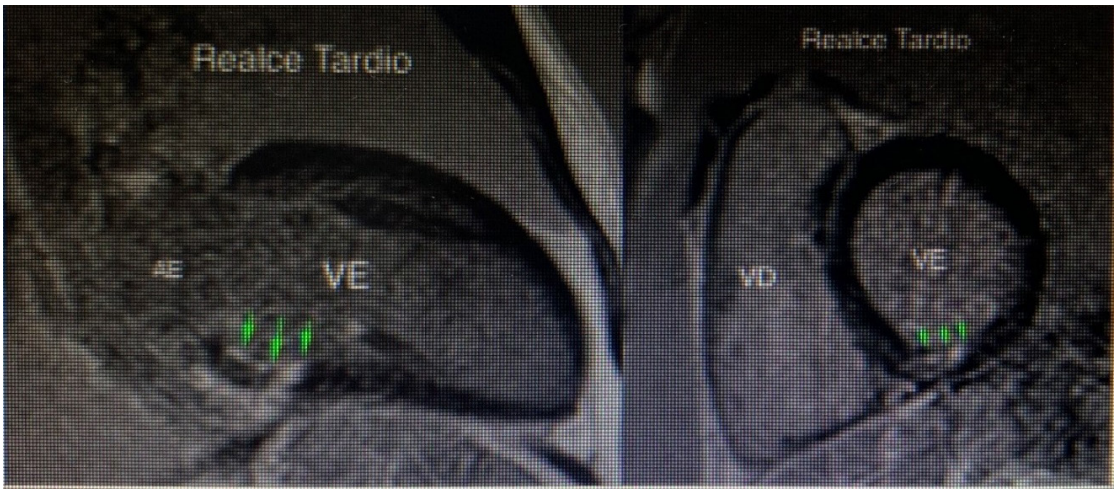

Figure 1. Post-contrast cardiac magnete resonance images in the long axis and long short planes, respectively; these images show delayed hyperenhancement of the mid-ventricular anterior wall and basal inferior wall wall compatible with infarct.

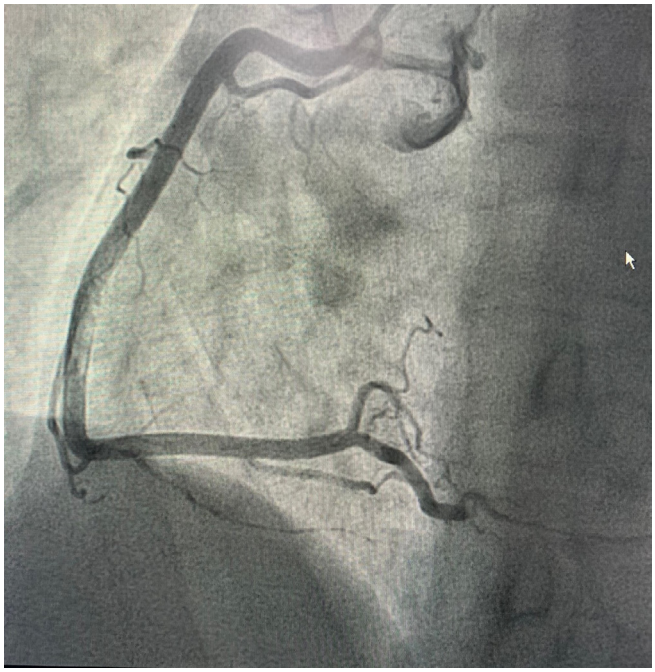

Figure 2. Coronary angiography image demonstrating right coronary artery with thrombus (solid blue arrow).

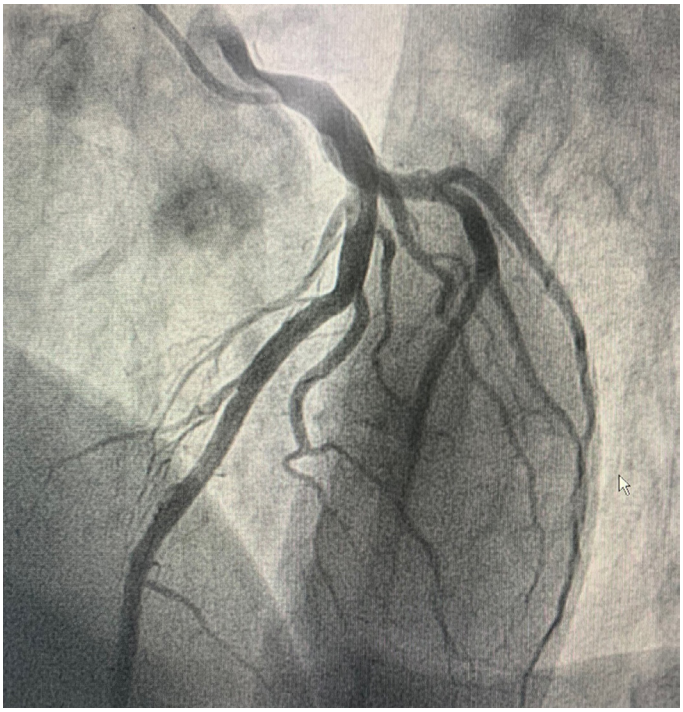

Figure 3. Coronary angiography image demonstrating left descending coronary artery with thrombus (solid blue arrow).

Few case reports of ST segment elevation myocardial infarction presenting in the context of COVID-19 have been published. Regardless of the actual incidence, acute cardiac injury has been consistently shown to be a strong negative prognostic marker in patients with COVID- 19 [8,9].

Recent articles have described impaired hemostasis and pro-thrombotic state associated with elevated multifocal thrombosis in patients hospitalized for COVID-19 [10,11].

Unfortunately, the presence of active atherosclerotic plaques could not be characterized by intracoronary imaging studies using intravascular ultrasound or optical coherence tomography, because limitations on material resources.

We believe that the pro-thrombotic or endothelitis-inducing effects of the COVID-19 infection may have caused either local coronary thrombosis or formation of a distal embolus and facilitated the coronary thrombosis in our patient.

Conclusion

In this case, we present a serious acute cardiac event in the context of an otherwise paucisymptomatic COVID-19 infection. Further analysis and explanation of the possible pathogenic mechanisms leading to MINOCA and other cardiovascular complications during this pandemic are urgently warranted.

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