Acute High Lateral Myocardial Infarction in a Patient with Covid-19 Pneumonia with Normal Coronary Arteries

Oguz Kilic*
Department of Cardiology, Karaman Research and Training Hospital, Turkey

Abstract
ST-Segment Elevation Myocardial Infarction (STEMI) may rarely be a clinical presentation of COVID-19. We present a 70-year-old male patient with no known cardiovascular disease and no conventional risk factors, who applied with STEMI and was diagnosed with COVID-19 infection during follow-up.

Introduction
The coronavirus disease-2019 (COVID-19) was first identified in China. The World Health Organization has described it as a pandemic [1]. It was found to cause diffuse thrombus in both arterial and venous circulation. Together with the evidence showing widespread intracoronary thrombus burden in COVID-19 patients presenting with ST-Segment Elevation Myocardial Infarction (STEMI), it suggests possible pathophysiological links between COVID-19 and acute coronary syndromes [2]. We aimed to present a COVID-19 patient with high lateral STEMI and had normal coronary arteries.

Case Report
A 70-year-old male patient presented to the emergency department with chest pain and shortness of breath lasting for 2 hours. At his admission, he was consciously oriented and cooperative. Arterial blood pressure was 120-80 mmHg, heart rate was 80 bpm and oxygen saturation was 97% on room air. He had no fever. He had no history of cardiovascular disease and conventional risk factors (smoking, hypercholesterolemia, diabetes mellitus, hypertension, or familial history of premature coronary artery disease).

*Corresponding author: Oguz Kilic, Department of Cardiology, Karaman Research and Training Hospital, Turkey. Tel: +90 5300493033; Email: dr.kilicoguz@gmail.com


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Thorax Computed Tomography (CT) and brain CT were requested to the patient. Ground-glass opacities covering approximately 50% of the lung parenchyma were observed in thorax CT. Pulmonary angiography was negative for pulmonary embolism (Figure 3). Brain CT was normal. The nasopharyngeal swab was positive for SARS-CoV-2 by real-time reverse-transcriptase-polymerase-chain-reaction assay. He was transferred to the pandemic service.

Discussion

Although COVID-19 often occurs with respiratory distress, studies have shown a significant increase in the risk of thrombosis. These thrombotic conditions are pulmonary embolism, deep vein thrombosis, cerebral infarction [3]. They may also apply with the STEMI clinic. This may be the only symptom of COVID-19. Especially, patients with comorbidities (diabetes mellitus, hypertension, hyperlipidemia and smoking history) are at risk. Our case is unique in the literature with the absence of conventional risk factors, the detection of normal coronary arteries, and the negative results of tests for other thrombotic disorders.

The mechanism that causes thrombus in COVID-19 patients has not been clarified yet. However, it is thought to be endothelial dysfunction caused by the cytokine storm caused by the increased production of pro-inflammatory cytokines (TNF-alpha, IL-1, IL-6) [4]. Thrombin together with endothelial dysfunction leads to coagulation activation. Therefore, we think that endothelial dysfunction and thrombin activation in the background of fibrous plaque erosion are the most likely mechanisms of STEMI [5]. Myocardial infarction with Non-Obstructive Coronary Artery (MINOCA) is clinically defined by the presence of the universal Acute Myocardial Infarction (AMI) criteria, absence of obstructive coronary artery disease (≥50% stenosis) and no overt cause for clinical presentation at angiography (eg, classic features for takotsubo cardiomyopathy) [6]. In our patient, the coronary arteries were also completely open. No major thrombus was observed. We are considering MINOCA due to COVID-19.

Conclusion

COVID-19 may present with STEMI with increased thrombosis. Although anticoagulant therapy may be beneficial in these patients, more studies are needed to elucidate the mechanism and make the treatment more comprehensive.

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References
