

Managing Myocardial Infarction in the COVID-19 Epidemic: A Case Report

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Abstract

Coronaviruses can cause viral pneumonia with extrapulmonary manifestations and complications. Many patients have either underlying cardiovascular disease or cardiac risk factors. Acute heart attacks are also frequent in severe cases of coronavirus disease 2019 (COVID-19), which is associated with high mortality.

In this paper, we describe a patient with COVID-19 who presented with myocardial infarction (MI) symptoms but lacked the initial symptoms of the infection such as fever and cough. COVID-19 and myocardial infarction were diagnosed. The patient underwent thrombolytic treatment and fully recovered.

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Introduction

Since December 2019, coronavirus disease 2019 (COVID-19) has caused significant mortality and morbidity in more than 30 countries with its acute respiratory syndrome. Ample evidence of cardiac injury has recently been observed in patients with COVID-19.¹

Zhou et al² stated that cardiac complications, including heart failure, new or worsening arrhythmia, and new or worsening myocardial infarction (MI), were prevalent in patients with pneumonia. Cardiac arrest has been reported in 3% of hospitalized patients with pneumonia.³ Contrary to these studies, however, the American College of Cardiology (ACC) clinical bulletin emphasized that the association

between COVID-19 and cardiac injury and mortality risk is unclear.

In this study, we describe a man who presented with cardiac injury but had no obvious clinical symptoms of viral infection, although his COVID-19 test was subsequently positive. We also discuss the management of COVID-19 patients with coronary heart disease in a real-world context in a developing country.

Case Report

A 65-year-old man with chest pain spreading to the left shoulder, along with weakness, paleness, and diaphoresis,

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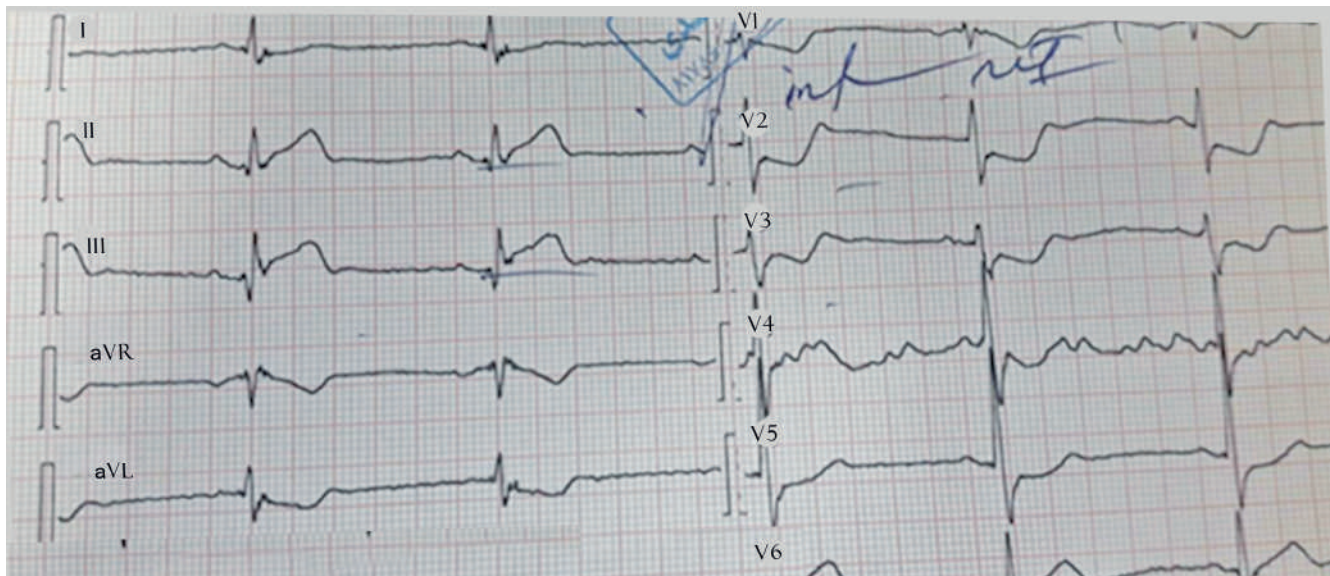


Figure 1. The patient's electrocardiogram shows inferoposterior myocardial infarction.

went to the emergency department of a private hospital. The patient's pain had begun an hour earlier, and his companion declared that the patient had gastroenteritis the preceding week.

Lab tests demonstrated a high troponin T level, and an electrocardiogram (ECG) showed a concave ST-segment elevation in inferior leads II, III, and aVF, in conjunction with ST depression in leads V₁ to V₃ with an R/S ratio above 1, indicating inferoposterior MI (Figure 1).

Given the current prevalence of the coronavirus epidemic, the patient was also screened for COVID-19 infection. He had neither a cough nor a fever, but his SO₂ level was below 93%. No history of pulmonary disease was reported. A chest X-ray showed ground-glass opacity in the basal and peripheral portions of the left lung and the lower zone of the right lung. The findings suggested viral pneumonia (Figure 2).

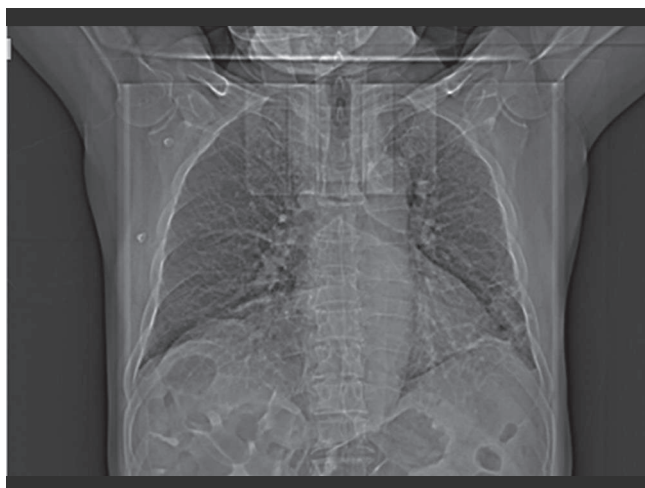


Figure 2. The chest X-ray in the posteroanterior view shows viral pneumonia.

The patient had a history of addiction and dyslipidemia. His laboratory tests showed positive cardiac troponin I, lymphocytopenia (WBC=12.62×10³/μL), a less-than-normal monocyte count (2.5%), and a normal C-reactive protein level.

The patient had chest pain lasting more than 1 hour, a thrombolysis-in-myocardial-infarction (TIMI) score below 5, and a primary angioplasty in myocardial infarction (PAMI) score of less than 6 hours. Considering the strong suspicion of COVID-19 infection, the unavailability of a standard negative-pressure catheterization laboratory system, and medical staff protection, a decision was made to commence thrombolytic treatment. He was prescribed 300 mg of aspirin, 300 mg of clopidogrel, and 80 mg of atorvastatin. Subsequently, tenecteplase (45 mg intravenous bolus) was prescribed in an isolated room to maintain personal protection while a heparin infusion was administered.

A high-resolution computed tomography scan of the lungs showed increased pleural thickness, especially on the left side; a patchy ground-glass image with a crazy-paving view in both lungs, particularly in the left lung and in the peripheral and basal areas; and a patchy consolidation image in the posterobasal segments of the left lung (Figure 3).

The patient's symptoms disappeared, and he was monitored in an isolated room. Afterward, he underwent echocardiography, which demonstrated near-to-normal left ventricular systolic function (ejection fraction ≈45%) without mechanical complications. (The ejection fraction was 35% before thrombolytic therapy.) Based on the ECG, normal echocardiography, and good clinical symptoms, the patient was transferred to a COVID-19–dedicated hospital, where he received a 2-drug treatment of hydroxychloroquine and ritonavir/lopinavir in addition to cardiac medications. He was discharged from the hospital at the end of the

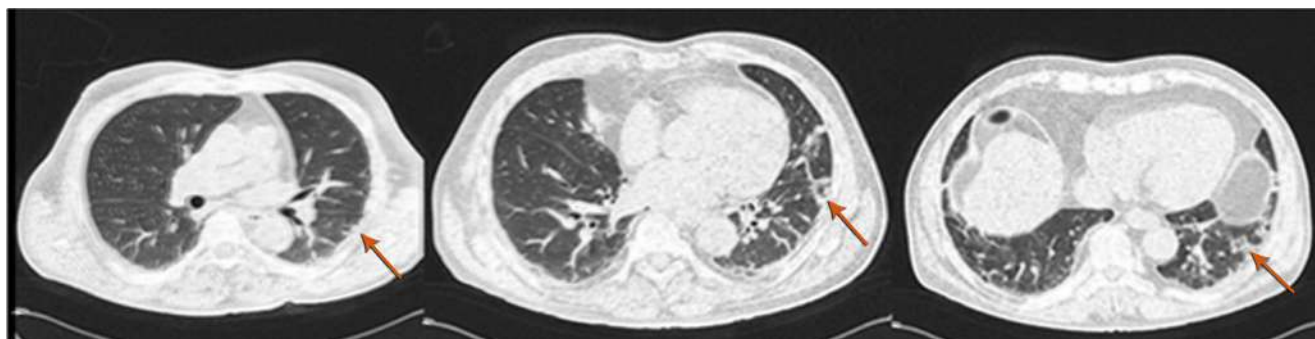


Figure 3. The high-resolution computed tomography scan of the lungs shows a patchy ground-glass image at different sections.

treatment period in good general condition after receiving comprehensive instructions on how to maintain his health and that of people around him.

Discussion

Patients with long-term coronary heart disease and individuals with risk factors for atherosclerotic cardiovascular disease are at a higher risk of the development and progression of acute coronary syndrome during acute infections. During flu epidemics, previous investigations reported similar findings with respect to MI.^{4,5} Acute coronary events can be caused by a dramatic rise in oxygen demand as a result of infections, accelerating damage to the myocardium and leading to MI. Meanwhile, circulating cytokines are released as severe systemic inflammatory stress, which can lead to the instability and rupture of atherosclerotic plaques. Likewise, patients with heart failure are also prone to hemodynamic compensation during the stress of severe infectious diseases. Therefore, patients with cardiovascular problems may be at a greater risk of complications and death caused by severe inflammatory responses to COVID-19. In addition, acute myocarditis and heart failure were reported with SARS-CoV respiratory syndrome in the Middle East and can be expected to occur with COVID-19 due to the presence of a similar disease.

Our patient presented to the emergency department with MI, and subsequent tests revealed that he had COVID-19. Still, it is unclear why the patient exhibited no COVID-19 symptoms such as fever and cough, and whether his MI was a side effect of COVID-19. On a more general note, no robust data exist regarding whether cardiac risk factors such as dyslipidemia may lead to atherosclerotic plaque rupture with the exacerbation of infectious conditions and subsequently result in MI. Although such possibilities could be attributed to our patient's condition, we cannot precisely pinpoint the cause of his MI. Nonetheless, 2 papers published in *JAMA Cardiology* by 2 academic hospitals in Wuhan support these presumptions.^{6,7}

A recent pathological study found rare interstitial mononuclear inflammatory infiltrates in cardiac tissue

without substantial myocardial injury in patients with COVID-19,⁸ suggesting that the infection cannot directly damage the heart. All the above information confirms that the mechanism governing the effects of COVID-19 on cardiovascular events is still unclear.

Moreover, uncertainty surrounding the cause of our patient's MI rendered decision-making regarding how to manage his treatment complicated. Notably, the patient did not have the initial manifestations of COVID-19, which are cough and fever; consequently, initial screening for the coronavirus took low priority for the emergency department staff. Still, despite the patient's normal C-reactive protein level, we meticulously analyzed his symptoms, including gastroenteritis, in the preceding week, and performed radiography and computed tomography. Our findings confirmed that the patient was infected with the coronavirus.

The World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC) have attached great significance to the precise prevention and control of specific infections for the purposes of cardiovascular interventions. Catheterization laboratories should generally be configured to have negative-pressure ventilation, and precautions should be taken by wearing face masks, eye protection, special clothing, and gloves.⁹ Unfortunately, there is a paucity of catheterization laboratories with standard negative pressure for specific epidemic conditions, and the risk of disease transmission during medical interventions is very high for the staff.

In cases similar to ours, as noted by Claeys and Meester,¹⁰ if the patient's TIMI score is below 5, there is no difference between percutaneous coronary intervention and thrombolytic therapy. We opted for thrombolytic therapy and prescribed the drug of choice for thrombolytic treatment, tenecteplase, on the strength of its low risk of bleeding and higher efficacy.¹¹

Another critical issue to be considered in this epidemic condition is the drug interaction between antiplatelets and antivirals. Ritonavir/lopinavir can affect the activity of P2Y₁₂ inhibitors through the CYP3A4 inhibitor, which in turn reduces the serum concentrations of the active metabolites of clopidogrel and prasugrel and

increases the serum concentration of ticagrelor.¹² There is evidence to suggest that clopidogrel may not always confer sufficient platelet inhibition in conjunction with ritonavir/lopinavir. As a result, the most appropriate drug of choice is prasugrel.¹³ However, in a country like Iran that is under severe economic sanctions, it is difficult to access prasugrel and suchlike. Choosing the appropriate drug is, therefore, of vital importance as are robust risk stratification criteria for discriminating between high- and low-risk patients and consideration of contraindications to thrombolytic therapy.

Conclusion

Although the mechanism of cardiac injury during the COVID-19 pandemic needs further clarification, it is crucial to consider this disease in the management of COVID-19. To that end, medical staff should take heed of conditions such as patient hemodynamics, MI extension, emergency referral time, risk stratification criteria to discriminate high-risk patients from low-risk patients, and contraindications to thrombolytic therapy.

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