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Case Report

Fountain of Youth or the Trojan Horse: A Case Report of Pulmonary Thromboembolism After Anticoagulant Administration in a COVID-19 Patient

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Abstract

Background: The COVID-19 pandemic has raised a serious challenge for health care systems, a challenge which requires taking effective and intensive measures to provide patient care. COVID-19 can cause damages to various organs, including heart, through causing various changes in the inflammatory and coagulation systems. Some cases of cardiac injury can display mistakable signs of myocardial infarction (MI). Cardiac injury can mimic acute conditions such as MI.

Case Presentation: In this study, a case of a 33-year-old man with an initial diagnosis of MI by ST-elevation was investigated. He later developed pulmonary thromboembolism (PTE) after being treated with fibrinolytic and anticoagulants; after further investigations, however, he was found not afflicted with primary MI. Our findings may have proven useful in demonstrating the unexpected effects of anticoagulants on COVID-19.

Conclusion: Miss-diagnosing these cases as well as administrating effective treatment for COVID-19 patients may have posed real risks to the patients and made it difficult to manage them due to the high risk of death and the lack of differential diagnosis of the given patients.

Keywords: COVID-19, Pulmonary embolism, Acute myocardial infarction /STEMI, Antithrombotic treatment

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Background

The COVID-19 pandemic has created a serious challenge for health care systems, which requires taking effective and intensive measures to provide patient care (1). In addition to the risks of causing respiratory distress, the cardiovascular damage caused by this disease threatens the health of COVID-19 patients in various ways (2). Moreover, some cases of cardiac injury can display mistakable signs of myocardial infarction (MI) (3). Diagnosing these cases and providing effective treatment for these patients may pose risks to the patients and make it difficult to manage them due to the high risk of death and the lack of differential diagnosis of the given patients.

In this study, a patient afflicted with COVID-19 and pseudo-MI who also developed pulmonary thromboembolism (PTE) during treatment was investigated.

Case Presentation

A 33-year-old man with no underlying disease and no history of familial cardiovascular disease was admitted to the hospital suffering from shortness of breath, fever, myalgia, as well as bone pain and, then, was hospitalized due to presenting with decreased O2 saturation and COVID-19 symptoms. After carrying out further examinations, his PCR test results were found to be positive for COVID-19. He was treated in the hospital for COVID-19 with Kaletra (lopinavir/ritonavir) and hydroxychloroquine. Two days after admission, he developed atypical chest pain and the electrocardiogram (ECG) results initially suggested a normal health status for him but, after a few hours, showed inferior ST-elevation MI (Figure 1). In less than 30 minutes (door-to-needle time), therefore, the patient was treated with fibrinolytic reteplase 10 IU by intravenous injection for 2 minutes and the treatment was repeated half an hour later. The changes occurred in ECG after injecting Reteplase suggested an appropriate response to treatment. However, he developed pleuritic chest pain with dyspnea one day later and was referred to our center (Shahid Mohammadi Hospital, Bandar Abbas, Iran) for undergoing CT angiography (CTA). CTA results revealed patchy ground-glass opacity and consolidation resulting from COVID-19 pneumonia, left side pleural effusion, and filling defect in segmental

and subsegmental branches of RLL (right lower lobe) and LLL (left lower lobe), also suggested patchy consolidation and ground-glass opacity in RLL that reported pulmonary haemorrhage by radiologist. CTA results also showed acute PTE in pulmonary artery examination. (Figure 2, Supplementary file 1). Despite a normal ECG, elevated specific cardiac Troponin I (11926.4 pg/mL) and 45-50% ejection fraction with definite regional wall motion abnormality in the echocardiographic study indicating inferoseptal hypokinesia and mild hypokinesia in right ventricle suggested evidence of pseudo-MI. Finally, he was given the prescribed medications, including Kaletra, hydroxychloroquine, enoxaparin, aspirin, and atorvastatin in the intensive care unit (Table 1). Then, coronary angiography was performed seven days after admission (on 2 July), and his coronary arteries conditions were determined (i.e., normal epicardial coronary artery). Echocardiography carried out at discharge time showed normal ejection fraction for him.

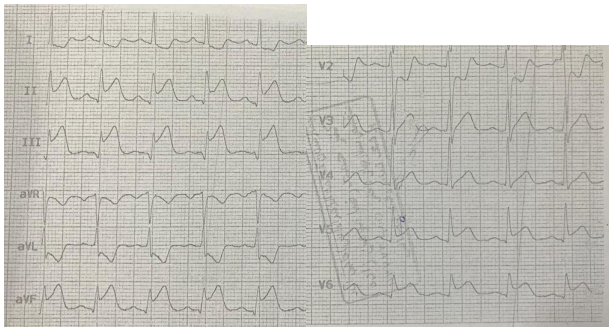

Figure 1. Patient ECG: Normal sinus rhythm, Rate:100/min, Normal Axis, St Elevation in II/III/Avf, St Depression in I and Avl and T Inversion in I and Avl.

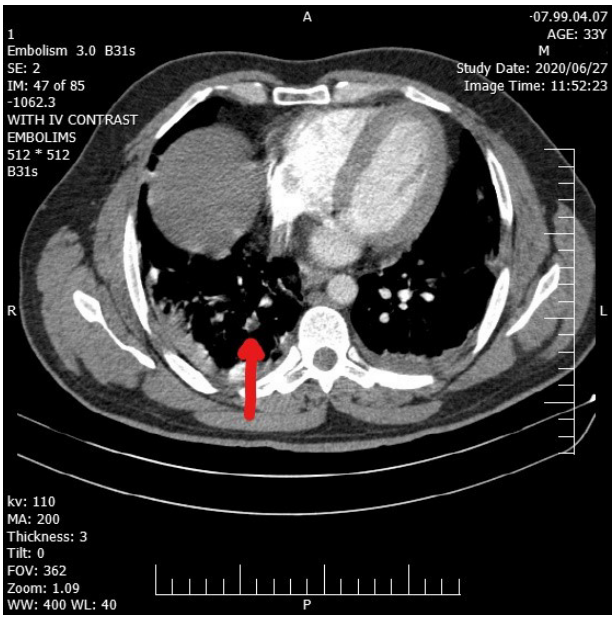

Figure 2. Computerized Tomography Image With Intravenous Contrast, Suggesting Pulmonary Thromboembolism.

Discussion

This case was most of all characterized by the occurrence of PTE after the treatment of pseudo-MI with fibrinolytic. However, the patient was expected to have immunity to PTE due to taking fibrinolytic and anticoagulants. Dealing with this patient was difficult due to his affliction with COVID-19 but, eventually, he was successfully treated by maintaining his treatment regimen through giving him the COVID-19 drug, anticoagulants, and antiplatelets.

Pathologically, there are several mechanisms for cardiovascular complications in patients with COVID-19. Acute myocardial injury resulting from a direct virus damage to myocardial cells is one of these mechanisms. A direct damage may be caused by the expression of angiotensin-converting enzyme 2 (ACE2) receptor in myocardial cells and the binding of SARS-CoV-2 virus to it, which can lead to an increase in cardiac enzymes (4). Systemic inflammation caused by cytokine storms can lead to damage to various organs such as the heart (5, 6). Several factors including impaired demand-supply balance and increased cardiac metabolism due to systemic inflammation, shear stress, rupture of sclerotic plaques and thrombosis, as well as systemic electrolyte imbalance due to systemic disease are responsible for causing cardiac injury (7).

In the study by Shi et al, it was demonstrated that about 19.7% of hospitalized COVID-19 patients suffered from cardiac injury (8). Increased troponin I, as a result of cardiac cell damage, may occur in cardiac injury. An increase in troponin among these patients is also directly related to the patient's deteriorating condition (5). There is an increased risk of death for COVID-19 patients with

Table 1. Serial Coagulation Tests and ECG Results of the Patient

Complication	Day				
	20 June (Admission for COVID-19)	25 June (STEMI and Treatment With Reteplase)	26 June (Pleuritic Chest Pain, Suspected to PTE)	27 June (Diagnosis of PTE After Referral of the Patient to Our Center)	29 June (Patient Recovery)
PT	12.0	12.5	12.4	15.8	13.1
INR	1.0	1.06	1.02	1.3	1.0
PTT	35	31	38	23	59
ECG result	Normal	STEMI	Normal	Normal	Normal

Abbreviations: PT, prothrombin time; PTT, partial thromboplastin time; INR, international normalized ratio.
The normal range for tests: (PT: 12-14, INR: 1.0, PTT: 25-45).

a cardiac injury (8).

The cytokine storm caused by COVID-19, moreover, can trigger ECG changes in patients by creating stress cardiomyopathy. ECG changes can also be triggered by hypoxia caused by acute respiratory syndrome in COVID-19 and by the mechanism of endomyocardial ischemia (9).

What made our case interesting was the occurrence of cardiac injury with changes in the patient's troponin, ECG, and echocardiography, all of which suggest the ST-elevation MI on different days. Normal angiography performed on patient also revealed evidence of pseudo-MI. Another interesting finding from our study that contradicted the normal results of coronary angiography was the occurrence of PTE in this patient. Although the treatment team adopted fibrinolytic as an emergency procedure due to suspected MI, it was expected that PTE was prevented by taking fibrinolytic in case when a clot was formed in the pulmonary arteries.

Despite the limited evidence, in the study of Llitjos et al, early use of anticoagulant or prophylaxis significantly increased venous thromboembolism in COVID-19 patients. PTE was also reported to occur in a number of these patients (10). Early use of anticoagulants may have led to PTE in the following days due to suspected MI in our patient patient. However, it was recommended that further studies be carried out to investigate coagulation in COVID-19 patients, procoagulant-anticoagulant balance, and the use of anticoagulants (6). In this case report, referring the patient from another center to our hospital simply implied that they were not able to provide all the evidence in detail; however, inadequacy of evidence was relatively overcome by reviewing the patient's file as well as by contacting the center that had initially admitted him.

Conclusion

It was recommended the patients with COVID-19 should receive further care in case when they suffered from cardiopulmonary complications as well as coagulopathy ones such as PTE.

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Author Contributions

SB and AG were involved in the supervision and treatment process. DH and MMS were involved in data collection, patient follow-up, and manuscript writing.

Availability of Data and Materials

Not applicable.

Conflict of Interests

Authors declared no conflict of interests.

Ethical Approval

This study provided the readers with no information revealing the patient's identity. Furthermore, the study was approved by the Ethics Committee of Hormozgan University of Medical Sciences (No.: IR.HUMS.REC.1399.314). The committee can be reached at https://ethics.research.ac.ir/IR.HUMS.REC.1399.314.

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Informed Consent

Written informed consent was obtained from the patient regarding publication of his details and the inclusion of images in this manuscript.

Supplementary Files

Supplementary file 1 contains a video file.

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