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Case Report

A Middle-Aged Man with Dyspnea in the COVID-19 Era

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Abstract

Background: Dyspnea can be seen in COVID-19, and also other conditions including heart failure and this can make a diagnostic challenge.

Case Presentation: A 60-year-old man was presented with progressive dyspnea. Chest CT scan showed bilateral opacities, and he was admitted in COVID-specific ward. His dyspnea was worsened and oxygen saturation was reduced. Electrocardiogram showed T wave inversion in precordial leads, and troponin I was positive. Symptoms were improved with loop diuretic therapy and SARS-CoV-2 PCR was negative.

Conclusion: COVID-19 is not necessarily the underlying cause of dyspnea in all patients referring to the hospital with respiratory symptoms during this pandemic, and cardiac etiologies are still important.

Keywords: COVID; Myocardial infarction; Heart failure

Introduction

In December 2019, a new respiratory illness was reported from Wuhan, China, which was known later as COVID-19 [1]. Dyspnea and dry cough were among the main symptoms, and ground-glass opacities were detected in the chest imaging [1]. However, there are overlaps between COVID-19 symptoms, and other diseases including heart failure. This can lead to misdiagnosis due to high prevalence of COVID-19 presentation in the emergency department. We reported a case of a middle-aged man presenting with dyspnea, which can make a diagnostic challenge.

Case Presentation

A 60-year-old man was referred to the emergency department due to progressive dyspnea at rest. His symptom had begun from 5 days before admission, and was worsen with exertion. He had no history of chest pain, cough, or fever. The patient had a history of smoking 60 packs of cigarettes per year and was an opium user. His past medical history was otherwise unremarkable, and he was not on any medication. The patient denied any close contact with a sick person but admitted to his lax approach to social distancing recommendations. Physical examination showed a blood pressure of 125/83 mmHg, pulse rate of 96 beats per minute, respiratory rate of 24 per minute, temperature of 36.9 °C, and oxygen saturation of 96% in room air. Heart auscultation was normal and there was no lower limb edema. In lung auscultation, scattered crackles were heard. Other physical examination findings were normal.

Initial laboratory tests were requested, including a nasopharyngeal swab for SARS-CoV-2 Polymerase Chain Reaction (PCR). Due to the presence of respiratory symptoms, the patient underwent a chest Computed Tomography (CT) scan, which showed diffuse bilateral airspace filling (Figure 1). The initial laboratory test results showed a mild leukocytosis (white blood cell count was 12000×10^9/L). Because of his imaging findings, diagnosis of COVID-19 was considered, and the patient was admitted to the COVID-19-specific ward. In the following hours, the patient`s condition grew worse. He was suffering from a respiratory distress, and the oxygen saturation was fall to 80% in room air. A cardiology consultation was ordered, and an electrocardiogram was obtained which showed deep T wave inversion in V2-V5, along with Q wave formation and poor R wave progression in pericardial leads (Figure 2).

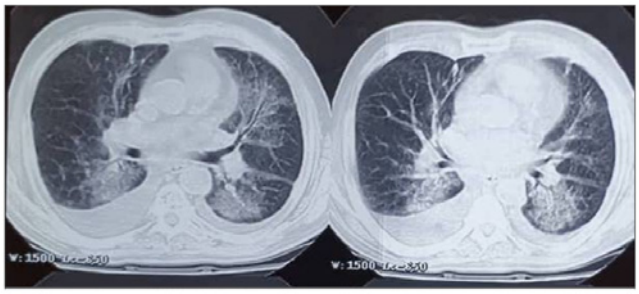

Figure 1: Chest CT scan showing bilateral alveolar involvement and ground-glass opacities. Note the pulmonary venous congestion and bilateral pleural effusion.

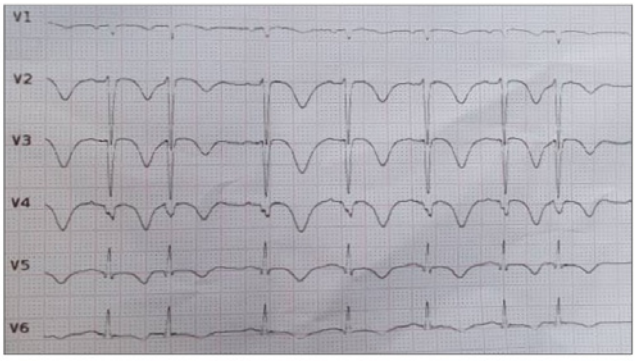

Figure 2: Electrocardiogram revealing T wave inversion in V2-V5, pathologic Q wave in V1, V3, and V4, and QT prolongation. Premature atrial complexes (PACs) are also evident.

The patient's electrocardiogram showed evidence of a neglected Myocardial Infarction (MI). Thus, cardiac troponin I was requested

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which was 1.2ng/L (upper normal limit was less than 0.11ng/L). Bedside echocardiographic was performed, which showed an ejection fraction of 25% with akinesia in the anterior and lateral walls. Indeed, the patient`s symptoms were due to an acute heart failure following a neglected MI. The patient underwent loop diuretic therapy, and was transferred to the Cardiac Care Unit (CCU). The patient's symptoms resolved dramatically following diuretic therapy and his oxygen saturation was improved. The SARS-CoV-2 PCR result was negative. The patient refused coronary angiography, and was discharged with guideline directed medical therapy few days later.

Discussion

We reported a case of neglected MI and the subsequent heart failure that was initially diagnosed and treated as COVID-19. While significantly fewer admissions for elective procedures are expectable during the COVID-19 pandemic, there are multiple reports of diminished numbers of urgent cases such as MI and decompensated heart failure [2,3]. One possible explanation is the fear of contamination on the part of patients, which keeps them from hospitals [3]. Nonetheless, we believe that at least a proportion of these patients still do refer to hospitals but are misdiagnosed with COVID-19. Multiple things related to the COVID-19 pandemic led to this misdiagnosis in this patient that should be noted. This is a likely scenario in areas with a limited testing capacity.

There is a substantial overlap between COVID-19-induced pulmonary involvement and pulmonary edema in chest imaging. It has been posited that a more predominant involvement of the central area, in tandem with concomitant cardiomegaly and pleural effusion (as our case), is more suggestive of pulmonary edema than COVID-19 [4]. Since it is not common practice to perform a CT-scan in pulmonary edema, most of the physicians are not familiar with its CT findings. On the other hand, performing CT-scan is highly recommended in the COVID-19 situation, so we think an increasing number of pulmonary edema cases are undergoing CT-scan these days.

Patients with diabetes mellitus or those consuming opioid-derived substances are prone to experience silent MI [5]. They might manage to ignore the pain or succeed in relieving it, but they are bound to succumb to dyspnea and pulmonary edema and seek medical help. This is the stage, unfortunately, where misdiagnoses are likely. Furthermore, while troponin may rise in COVID-19, in keeping with other instances of systemic inflammation, and be deemed to play a significant role in the prognosis of this infection [6], the principal cause of an elevated troponin level is MI and should, thus, be addressed first.

Conclusion

In conclusion, frontline physicians should pay heed to the fact that COVID-19 is not necessarily the underlying cause of dyspnea in all patients referring to the hospital with respiratory symptoms during this pandemic. Indeed, heart attack cases have not gone anywhere; we physicians need to have our wits about us to recognize them.

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