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Case Report

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COVID-19 Presenting as an Acute Myopericarditis: A Case Report Study

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ABSTRACT

Coronavirus disease 2019 (COVID-19) is a global challenge for the health care community. It typically presents with fever, generalized fatigue, dyspnea and rarely with chest pain due to involvement of heart. We report a 26-year-old young man with no significant risk factor for coronary artery disease who presented with chest pain and elevated troponin due to myopericarditis. Cardiac ischemic work up was negative for acute coronary artery disease. However, his PCR came positive for SARS-CoV-2 pneumonia. To decrease morbidity and mortality in young adults, clinicians should be aware of atypical presentation of COVID-19 pneumonia to diagnose in an early stage and initiate prompt treatment management.

Learning Points

- Identify myopericarditis as an atypical presentation of COVID-19
- Early recognition and prompt treatment initiation can prevent cardiac complications and mortality

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Introduction

Coronavirus 2019 (COVID- 19), caused by severe acute respiratory syndrome coronavirus 2 SARS-CoV-2), was first detected in Wuhan, China, and it has caused a heavy toll on healthcare [1]. The most common symptoms are fever, myalgia, fatigue, and respiratory symptoms. Chest pain in the absence of preexisting cardiovascular disease in COVID-19 patients is a less frequent manifestation, leading to severe COVID-19 complications such as myopericarditis. A study was done between period of March 2020 to January 2021 has shown 0.146% risk of myocarditis among COVID-19 patients [2]. We report a case of myopericarditis occurring as a complication of COVID-19 infection in a young man.

Case Report

A 26-year-old man with a past medical history of tick bite presented to the emergency department with sudden-onset, sharp, left-sided chest pain ongoing last 4 hours. He denied a history of active smoking, a family history of diabetes, or coronary artery disease. He had recent flu-like symptoms (mild fever, arthralgia, generalized fatigue) managed with rehydration and Tylenol at home. The patient denied fever, cough, myalgia, shortness of breath, nausea, or vomiting. On cardiovascular examination, the lungs were clear without wheezing, crackles, and normal S1 and S2 of heart sound without murmurs. He was afebrile, saturating 96% on ambient air, and hemodynamically stable. Other examination findings were within normal limits. Chest X-ray showed no acute pulmonary pathology (fig1). The initial rapid COVID test was negative. Electrocardiogram (EKG) showed sinus rhythm with first-degree atrioventricular block, without acute ischemic changes (fig2). His initial troponin level was elevated to 0.19 ng/dL (normal: below 0.03 ng/dL) and subsequently peaked at 0.96 ng/dL. Echocardiography showed a normal left ventricular chamber size and estimated ejection fraction above 55% with no pericardial effusion. The next day nasopharyngeal swab tested positive for COVID-19 by polymerase chain reaction (PCR). The rest of the respiratory viral panel was negative, including Adenovirus, Rhinovirus, Influenza A & B, Parainfluenza, and respiratory syncytial virus (RSV) (Table 1). Lyme diagnostic workup was negative for active Lyme disease. Cardiology deferred emergent coronary angiography, and he underwent cardiac computed tomography angiography, which ruled out coronary artery disease (fig3 &4). A diagnosis of myopericarditis was made based on typical chest pain, ECG presentation, and echocardiogram findings. During the hospital course, he was treated with oral colchicine and ibuprofen. He recovered with inpatient treatment and was discharged on day 4.

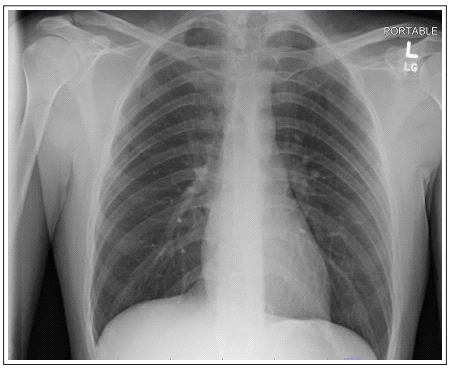

Figure 1: Chest X-ray showed no lung pathology

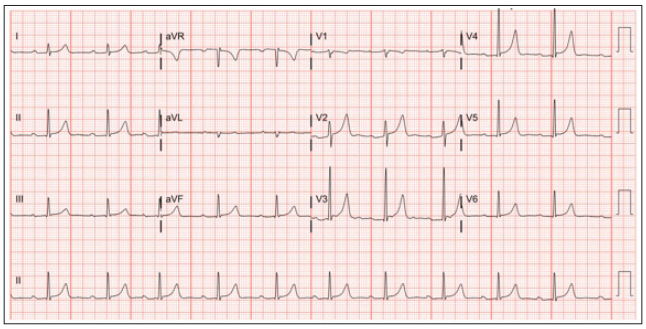

Figure 2: EKG showed no acute ischemic changes with 1st degree AV heart block

Respiratory viral panel	Results
Adenovirus	Not Detected
Bordetella parapertussis IS1001	Not Detected
Bordetella pertussis (ptxP)	Not Detected
Chlamydia pneumoniae	Not Detected
Coronavirus 229E	Not Detected
Coronavirus HKU1	Not Detected
Coronavirus NL63	Not Detected
Coronavirus OC43	Not Detected
SARS CoV-2 Cepheid	Positive
Human Rhinovirus/ Enterovirus	Not Detected
Influenza A & B	Not Detected
RSV Cepheid	Negative
Mycoplasma pneumoniae	Not Detected
Parainfluenza Virus 1,2,3,4	Not Detected
Respiratory Syncytial Virus	Not Detected
Human Metapneumovirus	Not Detected
FLU A & B Cepheid	Negative

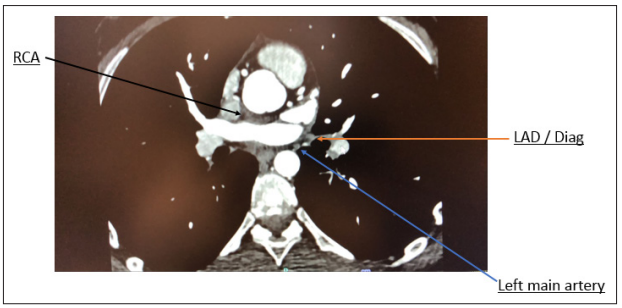

Figure 3: Normal CTA Coronary Artery. Left anterior descending artery (LAD), Right coronary artery (RCA).

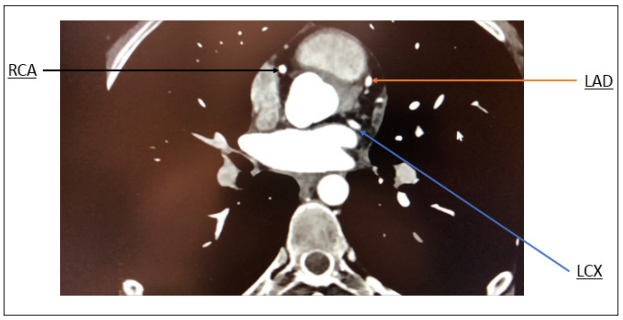

Figure 4: Normal CTA Coronary Artery. Left anterior descending artery (LAD), Right coronary artery (RCA), Left Circumflex Artery (LCX)

Discussion

Certain viral infections can cause myopericarditis, especially enteroviruses. Myopericarditis has also been observed in infection with SARS-CoV-2 [3]. The proposed mechanism is the presence of membrane-bound peptidase angiotensin-converting enzyme 2 (ACE2) receptors, which SARS-CoV-2 uses to bind to cells, expressed in high heart and lung tissue concentrations. Viral entry then results in acute tissue injury and cardiovascular complications [4]. The degree of myocardial involvement varies, leading to variable clinical manifestations such as chest pain, palpitations, arrhythmias, or elevated troponin levels. Therefore, the clinician must identify early cardiac symptoms in patients with COVID-19 infection to prevent cardiac complications and mortality.

Serial measurement of cardiac troponin-I levels can help identify patients developing a severe form of the disease. The pathophysiology of elevated troponin in the setting of COVID-19 is attributable to non-ischemic myocardial injury induced by sepsis, systemic inflammation, hypoxia, cytokine storm, and stress cardiomyopathy [5]. Sinus tachycardia and non-specific ST changes, such as diffuse ST-segment elevation, are the most common EKG abnormalities seen in myopericarditis [6]. Other EKG patterns that might suggest myopericarditis include PR segment depression, particularly in leads with ST-segment elevation, QT prolongation, and atrioventricular block, the latter of which was seen in our patient. In addition, echocardiography can be indispensable in assessing the structure and function of cardiac chambers and wall motion abnormalities. Cardiac Magnetic Resonance Imaging can also be used to evaluate for inflammatory and pericardial disease. Nonsteroidal anti-inflammatory drugs (NSAIDs) combined with oral colchicine are the mainstay treatment option for viral myopericarditis [7].

Conclusion

COVID-19 infection can present with atypical symptoms of acute myopericarditis with minimal pulmonary symptoms. Studies have shown 16 times higher risk of myocarditis in COVID-19 patients. Clinicians should consider testing COVID-19 patients presenting with chest pain for elevated cardiac biomarkers, which might be suggestive of myopericarditis.

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Disclosure Statement

The authors have no conflicts of interest to declare. Statement of Ethics Informed consent was obtained from the patient for any materials used in the manuscript. This case report was conducted ethically in accordance with the World Medical Association Declaration of Helsinki.

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