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Abstract Form

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Project Title:	A Case of Post-COVID Myocarditis in a 39 year old Female with PCOS and Incidental Finding of CAD

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction

Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) enters human cells by binding angiotensin-converting enzyme 2 (ACE2) receptor, expressed on both heart and lung cells. This case report aims to highlight the association between coronavirus disease of 2019 (COVID-19) infection and cardiovascular morbidity.

Case

39-year-old Hispanic female with history of PCOS, HLD, HTN, OCP-provoked DVT and PE, presented to emergency department with 4 days of new onset intermittent severe substernal chest pain radiating down her left arm. She had SARS-CoV-2 pneumonia the month prior to presentation significant for cough, anosmia, and myalgias, which resolved without hospitalization. She had history of DVT and PE in 2016, attributed to oral contraceptives, and treated with 6 months of Xarelto. Family and social history was non-contributory except for marijuana use.

On arrival, she was hypertensive, tachycardic, and afebrile. Coagulation panel was normal, troponin-I was elevated, and toxicology was negative for stimulants. She tested positive for SARS-CoV-2 but remained asymptomatic. Patient was started on dual anti-platelet therapy. On day 2 of hospitalization, she complained of chest pain, but ECG showed no new changes. A second episode of chest pain revealed lateral ST-elevations and Q-waves in inferior leads. Troponin continued to downtrend. Left heart catheterization was performed with incidental finding of 60% stenosis of the proximal LAD with a smooth plaque. Patient clinically improved by day 4 without further chest pain and was discharged.

Discussion

SARS-CoV-2 employs membrane-bound spike protein to bind ACE2 receptor, which causes viral uptake. ACE2 expression occurs in multiple organs, including lungs, heart, brain, kidneys, and intestines. These target tissues could implicate their clinical manifestations during COVID-19 infection. Reports of cardiovascular manifestations in the setting of COVID-19 have included arrythmia, pericarditis, heart failure, acute coronary syndrome, and myocarditis. Viral myocarditis arises from inflammation and necrosis of cardiomyocytes in absence of ischemia. Infection of cardiomyocytes triggers viral antigen presentation, causing cytotoxic T cell infiltration and induced cell lysis. In some cases, cardiac biomarkers can elevate, indicating myocardial injury. ECG may demonstrate ST- and Q-wave changes. Echocardiography findings classically show global hypokinesis. While endomyocardial biopsy remains the gold standard for diagnosis, clinically suspected myocarditis in low-risk patients can be established through presentation and non-invasive diagnostic findings.

In relation to this patient, bedside transthoracic echocardiogram revealed left ventricular (LV) ejection fraction of 45%, LV systolic function reduced with regional wall motion abnormalities, severely hypokinetic apical and distal segments. Heart catheterization demonstrated 60% stenosis of the proximal left anterior descending artery; however, this lesion was not suspected to be the culprit lesion causing myocardial injury. Etiology of injury was thought to be caused by global ischemia in the setting of post-COVID-19 infection.

Conclusion

Evaluation and tracking of clinically suspected myocarditis in the setting of COVID-19 infection may give insight into the pathophysiology of infection in cardiomyocytes due to SARS-CoV-2. This case report aims to illustrate the possible association between COVID-19 and myocarditis in the hopes of decreasing morbidity and mortality.