The Role of Routine Echocardiogram in Patients with COVID-19

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Introduction

In December 2019, the first cases of Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2), a virus that causes the Coronavirus Disease 2019 (COVID-19), were reported in Wuhan, Hubei Province, China. The disease has quickly escalated into a global pandemic, with more than 21 million cases in the United States as of January 2020. While most common clinical manifestations include fever, cough, fatigue, and/or myalgia, the range of symptoms can include mild upper respiratory tract illness to severe pneumonia with acute respiratory distress syndrome (ARDS) requiring intensive care with mechanical ventilation.

Evidence has suggested involvement of extra-pulmonary organs including the gastrointestinal tract, liver, as well as the heart. Myocardial involvement is seen in more than 20% of patients and is associated with higher risk of mortality. While patients with underlying cardiovascular illnesses are likely to have severe manifestations of the disease, studies on the role of SARS-CoV-2 in declining systolic functionality of the heart after recovery are limited. Because patients may exhibit no noticeable symptoms of myocarditis, high clinical suspicion can lead to efficient diagnosis and treatment. Herein, we present a case of myocarditis in a patient with mild coronary vascular disease who had tested positive for COVID-19 three weeks prior to returning to the hospital, at which time a significant systolic dysfunction was found on 2D echocardiogram.

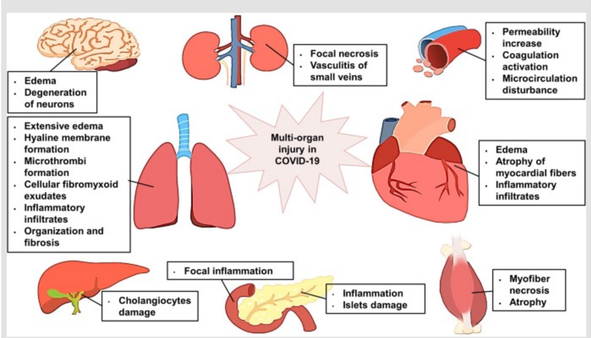

Figure 1. Multiorgan involvement in COVID-19 infection (6)

Case Presentation

A 58-year-old male with recent history of COVID-19 and no history of cardiovascular disease or any other significant medical history presented to the emergency room. Patient was diagnosed with COVID-19 three weeks prior and at this visit tested negative via PCR. His presenting complaint was non-bloody diarrhea associated with epigastric pain and chills, for which CT abdomen without contrast showed mesenteric and pericolonic stranding, suspicious for infectious or inflammatory enterocolitis. The patient denied chest pain and dyspnea upon arrival. Vitals revealed body temperature of 100.9°F and leukocytosis. He was also found to be hypotensive with blood pressure of 100/63, had a pulse rate of 88 BPM, and pulse oximetry showed 94% SaO2 on room air. Electrocardiogram showed sinus rhythm with slight upsloping ST-segment elevations in leads V3-V6, without criteria for STEMI or obvious ischemic changes. Initial troponin was 12 and repeat troponin trended down to 5.8. Patient's clinical presentation and elevated troponins were suspicious for acute myocarditis.

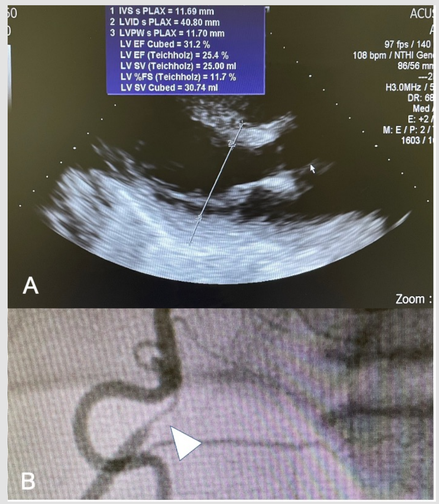

Figure 2. (A) 2D Echocardiogram showing LV EF 25.4%. (B) Coronary angiography showing lesion in mid-RCA (white arrowhead).

Chest X-ray showed mild bilateral perihilar opacities, suggesting atelectasis or possibly early infiltrates. There were no focal consolidation or pleural effusions noted. A 2D echocardiogram was obtained which showed normal left ventricle size, global hypokinesis, and an estimated left ventricular ejection fraction of 25.4%, suggesting severely reduced systolic function. Patient underwent cardiac catheterization with DES x2 for significant blockage of the mid-RCA. While the elevated levels of troponin was concluded to be due to possible post-COVID-19 myocarditis vs. NSTEMI, the severe reduction in systolic function was determined out of proportion to, and thus less likely to be secondary to pre-existing coronary artery disease. The patient had no known history of cardiac conditions and denied symptoms of cardiovascular dysfunction.

Treatment in the intensive care unit (ICU) consisted of Ciprofloxacin 800mg/day, Flagyl 1500mg/day, Protonix 40mg/day, IV fluids, and Levophed drip. Once the patient was determined to be clinically stable, he was discharged on levofloxacin, flagyl, ticagrelor, aspirin, atorvastatin, brilinta, metoprolol, and pantoprazole

Discussion

We describe a patient presenting with suspected acute myocarditis, with electrocardiogram showing ST-segment upsloping elevations, elevated serial troponins, and evidence of systolic dysfunction on echocardiogram. The significant decline in systolic function in our patient, with no history of cardiovascular diseases and a recent history of COVID-19 infection, implies possible myocardial injury due to SARS-CoV-2. While cardiac catheterization revealed significant blockage of the RCA, the overall impression from the procedure was a mild CAD. This finding was determined to be out of proportion to a severe decline in systolic function as shown by a 25% ejection fraction on 2D echocardiogram. Therefore, higher suspicion was placed on viral myocarditis.

Common etiologies of significant myocardial inflammation include autoimmunity and viral infections. Myocardial injury resulting from SARS-CoV-2 is associated with a notable increase in risk of mortality. Studies have suggested involvement of the cardiovascular system in COVID-19, although the mechanism remains unclear. One possible explanation is direct myocardial injury due to extra-pulmonary migration of infected alveolar macrophages seen on endomyocardial biopsy by Tavazzi et. al, which was also observed in autopsy samples of patients with Middle East respiratory syndrome coronavirus (MERS-CoV). The myocardium can also be involved indirectly due to the cytokine storm that results from infection, evidenced by an increase in interleukins 6, 10, and 2R as well as tumor necrosis factor-alpha (TNF-a) which are associated with myocardial compromise. Another possible mechanism for extrapulmonary manifestations of COVID-19 is based upon the expression of the metallo-peptidase Angiotensin Converting Enzyme-2 (ACE-2) in a variety of tissues including epithelial cells in lung alveoli, small intestine, and vasculature, and smooth muscle cells. The spike-like capsid, from which the name "Coronavirus" is derived, binds and downregulates ACE-2, decreasing metabolism of angiotensin II to cardioprotective angiotensins 1-7.

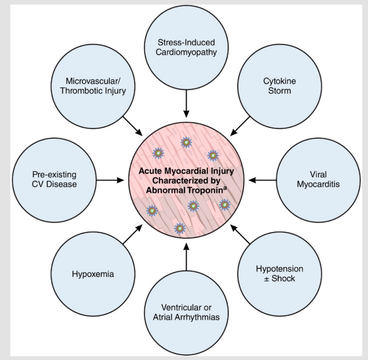

Figure 3. Possible mechanisms of myocardial injury in COVID-19 infection (7)

Conclusion

The importance of the above patient case lies in highlighting the significant decline in systolic function of the heart after recovery from COVID-19, and how incorporating 2D echocardiograms into the routine screening process for COVID-19 patients can help obtain an early diagnosis. Because patients may exhibit no noticeable symptoms of myocarditis, high clinical suspicion can lead to efficient diagnosis and treatment. Abnormalities found on 2D echocardiogram, as seen in our case, can be utilized to guide the next steps, including obtaining a cardiac MRI and endomyocardial biopsy to confirm the diagnosis.

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