CASE STUDY: COVID-19 INDUCED MYOCARDITIS

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Learning Objectives

Understanding the link between Covid and the development of myocarditis.

Case Description

- A 26-year-old male with a past medical history notable for hyperlipidemia, COVID (Mid-December), and recent travel to Mexico, was admitted for abdominal pain, diarrhea, vomiting, fever with concerns for colitis.
- During the hospitalization, he was found to have a troponin that peaked to 9 with concern for myocarditis, acute decompensated heart failure with EF 30% and moderate-severe MR. The patient was diuresed and cardiac MRI and right heart catheterization with biopsy showed mild myocarditis with high numbers of eosinophils.
- The patient was stabilized and scheduled with close follow-up for genetic testing and cardiology.

Pathophysiology of Viral Myocarditis

- Combination of direct cell injury and T-lymphocyte–mediated cytotoxicity
- Interleukin 6 (IL-6) orchestrates the proinflammatory responses from immune cells, including the T lymphocytes. This process causes T-lymphocyte activation and a further release of inflammatory cytokines, which stimulate more T lymphocytes, leading to a positive feedback loop of immune activation and myocardial damage.

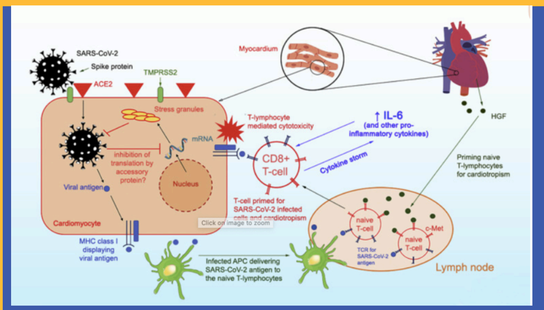

Figure 1. Mechanism by which COVID enters cells

- Angiotensin-converting enzyme 2 (ACE2) is an aminopeptidase that converts Angiotensin (Ang) II into Ang (1-7).
- Coronavirus uses ACE2 as a cellular receptor to invade target cells. In particular, the spike protein of SARS-CoV-2 (the beta-coronavirus responsible for COVID-19) is processed by transmembrane protease-serine 2 (TMPRSS2) and favors the binding of the spike protein to ACE2
- Ang II, acting on AT1 receptors, exerts powerful vasoconstrictor, pro-fibrotic, and pro-inflammatory effects. In contrast, Ang (1-7), acting on Mas receptors (MasR), is a potent vasodilator, anti-apoptotic, and anti-proliferative agent

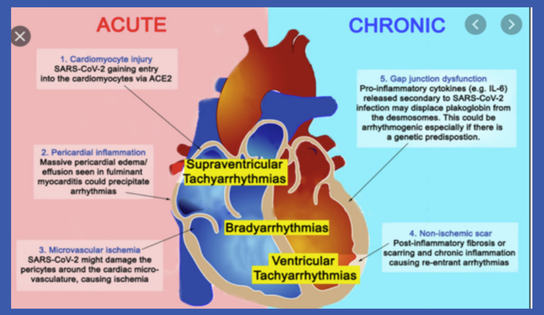

Figure 2. Acute vs Chronic Implications of COVID myocarditis

Discussion

Human coronavirus has been linked to myocarditis in patients of all age groups. There is thought that the pathophysiology of COVID-19–related myocarditis is a combination of direct viral injury and cardiac damage due to the cytotoxic host immune response. Clinical findings of COVID-19-related myocarditis include changes in electrocardiogram, cardiac biomarkers, poor cardiac function and symptomatic findings including chest pain, shortness of breath, fatigue, and arrhythmias.

COVID-19 induces myocarditis and an investigation of the long-term cardiac complications of COVID-19. Clinical presentation of SARS-CoV-2 myocarditis varies among cases. Some patients may present with relatively mild symptoms, such as fatigue and dyspnea whereas others report chest pain or chest tightness on exertion. Many patients show symptoms of tachycardia and acute-onset heart failure with cardiogenic shock. In these severe cases, patients may also present with signs of right-sided heart failure, including raised jugular venous pressure, peripheral edema, and right upper quadrant pain.

The viral RNAs of Middle East respiratory syndrome coronavirus (MERS-CoV) and SARS-CoV, close relatives of SARS-CoV-2, were found in the heart tissues of infected animals, suggesting coronaviruses do possess some form of cardiotropism. However, at this time, there are no findings which suggest that there is direct infection and replication of SARS-CoV-2 in heart cells.

The long-term impact of COVID-19 myocarditis remain unknown, but recent studies have suggested ongoing cardiac inflammation and other cardiac sequalae after COVID-19 that is independent of preexisting conditions. This indicates the need for better understanding of the pathophysiological mechanisms by which COVID-19 induces myocarditis and an investigation of the long-term cardiac complications of COVID-19.

References

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