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Editorial

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Covid-19 Effects on the Heart Diseases

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Introduction

Coronavirus disease 2019 (COVID-19), caused by a strain of coronavirus known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has become a global pandemic that has affected the lives of billions of individuals. Extensive studies have revealed that SARS-CoV-2 shares many biological features with SARS-CoV-2, the zoonotic virus that caused outbreak of severe acute respiratory syndrome, including the system of cell entry, which is triggered by binding of the viral spike protein to angiotensin-converting enzyme. Clinical studies have also reported an association between COVID-19 and cardiovascular disease. Pre-existing cardiovascular disease seems to be linked with worse outcomes and increased risk of death in patients with COVID-19, whereas COVID-19 itself can also induce myocardial injury, arrhythmia, acute coronary syndrome and venous thromboembolism.

Potential drug-disease interactions affecting patients with COVID-19 and comorbid cardiovascular diseases are also becoming a serious concern. In this Review, we summarize the current understanding of COVID-19 from basic mechanisms to clinical perspectives, focusing on the interaction between COVID-19 and the cardiovascular system. By combining our knowledge of the biological features of the virus with clinical findings, we can improve our understanding of the potential mechanisms underlying COVID-19, paving the way towards the development of preventative and therapeutic solutions. Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) has spread in nearly 200 countries in less than 4 months since its first identification; accordingly, the coronavirus disease 2019 (COVID 2019) has affirmed itself as a clinical challenge. The prevalence of pre-existing cardiovascular diseases in patients with COVID19 is high and this dreadful combination dictates poor prognosis along with the higher risk of intensive care mortality.

In the setting of chronic heart failure, SARS-CoV-2 can be responsible for myocardial injury and acute decompensating through various mechanisms. Given the clinical and epidemiological complexity of COVID-19, patients with heart failure may require particular care since the viral infection has been identified, considering an adequate re-evaluation of medical therapy and a careful monitoring during ventilation. Early phase symptomatic patients suffer from mild respiratory symptoms and may require supportive care like supplemental oxygen followed by an adaptive immunity stage with falling titers of the virus and resolution of symptoms. The second stage includes several mechanisms leading to pulmonary tissue injury, vasodilation, endothelial permeability and leukocyte recruitment that cause further pulmonary damage, hypoxemia, and cardiovascular stress. Ten percent of patients in the second stage may experience further exacerbation of immune response (hyper inflammation stage) become critically-ill, and they may suffer from Acute Respiratory Distress Syndrome (ARDS), acute cardiac injury, multi-organ failure, secondary bacterial infections, sepsis and require intensive care Accumulated evidence suggests that cardiac involvement is common, particularly in a patient hospitalized with COVID-19 disease. While if the same is true for all infected, symptomatic and asymptomatic people are unclear due to lack of vigilance on this cohort given the violent outburst of the disease forcing concentration of resources on hundreds if not thousands of hospitalized patients. Most of the meager available resources were also geared towards that cohort due to the sudden rise of cases world over with severe sickness and deaths that were noted. Acute myocarditis presents across a variable range of clinical severity and is a significant diagnostic challenge in the COVID-19 era. Patients with COVID-19 can present with chest pain, dyspnea, dysrhythmia, and acute left ventricular dysfunction. In patients with myocarditis and myocardial injury, serum troponin values will be abnormal. The Electrocardiogram (ECG) can demonstrate a range of findings, in some cases mimicking Acute Coronary Syndrome (ACS).

The ECG abnormalities result from myocardial inflammation and include non-specific ST segment-T wave abnormalities, T wave inversion, and PR segment and ST segment deviations (depression and elevation). Echocardiography and consultation with cardiology, if either are available, is encouraged, as differentiating myocarditis and ACS is difficult. Echocardiographic evaluation is more likely to demonstrate a focal wall motion abnormality with active, significant ACS while severe forms of COVID-19-related myocarditis will show either no wall motion defects or global wall motion dysfunction. ECG and echocardiographic abnormalities in the setting of COVID-19 are markers of illness severity and are correlated with worse outcomes. Moreover, troponin elevations in patients with COVID-19 infection have been directly associated with an increased risk of adverse outcome in those patients with severe infection, including mortality. The ability of SARS-CoV-2 to get transmitted during the asymptomatic phase and its high infectivity has led to the rapid transmission of COVID-19 beyond geographic regions, leading to a pandemic. There is concern that COVID-19 is cardio tropic, and it interacts with the cardiovascular system on multiple levels. Individuals with established CVD are more susceptible to severe COVID-19.

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