PERSPECTIVE

Pharmacological treatments for heart damage brought on by COVID-19

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ABSTRACT

The 2019 Coronavirus Illness (COVID-19), a current global pandemic that started in March 2020, is caused by SARS-CoV-2. About 935,000 people have died as a result of COVID-19 so far, across more than 200 nations. Injuries brought on by COVID-19 primarily impact the respiratory system, but they can also damage other organs, such as the cardiovascular system. The Angiotensin 2 Conversion Enzyme allows SARS-CoV-2 to enter host cells (ACE-2). ACE-2 is expressed in various organs, including the cardiovascular system, in addition to the lungs. A significant number of individuals with severe COVID-19 have cardiac abnormalities, such as myocardial fibrosis, edema, and pericarditis, according to several studies. Myocardial fibrotic lesions are brought on by the pathological remodeling of the extracellular matrix brought on by viral infection.

INTRODUCTION

A The SARS-CoV-2, the coronavirus disease-causing virus, global pandemic was announced by the World Health Organization (WHO) on March 11, 2020. (COVID-19). According to information published by the Johns Hopkins University (JHU) Coronavirus Resource Center, there have been 935,000 fatalities and 29.6 million cases of infection worldwide too far. One of the biggest health crises in human history, the COVID-19 pandemic is having major and unanticipated effects on both medicine and society. Seven vaccines have thus far entered phase 3 trials, offering a significant amount of hope for the eventual defeat of SARS-CoV-2. Since there are currently no viable vaccinations, it is crucial to comprehend the biology and clinical characteristics of COVID-19 infection and utilize appropriate therapeutic techniques to minimize serious sequelae. Typically, SARS-CoV-2 infection (COVID-19) is asymptomatic or only manifests as mild flu-like symptoms including fever, coughing, and exhaustion. Less frequently seen gastrointestinal symptoms such diarrhea and vomiting may also be present. However, in a tiny percentage of infected people, particularly in elderly patients or patients with pre-existing conditions, the infection can induce respiratory distress syndrome, which results in catastrophic lung harm and can even cause death. The Angiotensin 2 Conversion Enzyme allows SARS-CoV-2 to enter host cells (ACE-2). ACE-2 is expressed in various organs, including the cardiovascular system, in addition to the lungs. A significant number of individuals with severe COVID-19 have cardiac abnormalities, such as myocardial fibrosis, edema, and pericarditis, according to several studies. Patients who are elderly or who already have diseases may be more likely to experience a severe clinical consequence COVID-19. Particularly, it appears that the outcome and severity of the COVID-19 are strongly influenced by the physiological state of the Cardiovascular System (CV). Acute coronary syndrome, cardiac arrhythmia, severe myocardial lesions, and venous thromboembolism can all be brought on by COVID-19 alone. According to epidemiological data, people with conditions including diabetes, heart disease, and Chronic Respiratory Disorders had a higher COVID-19 death rate (e.g. COPD, asthma) Compared to pulmonary diseases, certain studies indicate that the presence of CV pathologies has a greater impact on the severity of COVID-19. Cardiovascular illnesses have already been linked to and seen in the SARS and MERS epidemics. According to epidemiological data,

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COVID-19 and heart illness are related. It appears that COVID19 and the Cardiovascular System (CV) interact in a complicated manner. The fast release of pro-inflammatory mediators known as a "cytokine storm" in severe COVID-19 instances leads to a rapid escalation of systemic inflammation and multi-organ lesions, especially in the heart and lungs. Direct cardiac injuries can result from the sudden and excessive release of pro-inflammatory mediators in particular. Heart lesions such myocarditis, cardiac fibrosis, edema, and pericarditis are brought on by a state of systemic inflammation. the patients with COVID-19 have a bad prognosis. An aberrant inflammatory or immunological response that may be triggered by the infection may serve as an indirect cause of cardiac harm brought on by COVID-19, as opposed to a direct cause mediated by virus penetration into the organ. SARS-CoV-2 infects host cells, mainly type 2 pneumocytes, macrophages, endothelial cells, and cardiac myocytes, through the transmembrane protein ACE-2, resulting in inflammation and organ damage. In particular, virus penetration into endothelial cells can lead to inflammatory/immune hyperreactivity, which can destabilize atherosclerotic plaques and result in acute coronary syndromes, as well as microvascular and macrovascular dysfunctions. In addition, patients with severe COVID-19 have been shown to have a rise in D-Dimer and a procoagulative impact. According to epidemiological statistics, people with COVID-19 had elevated serum concentrations of Lactate Dehydrogenase (LDH) and Creatine Kinase (CK). Similar damage and malfunction of the cardiac electrophysiology can result from viral invasion of cardiac cells. Additionally, severe heart lesions and fulminating myocarditis can develop as a result of systemic inflammation and high amounts of cytokines in the blood. An important clinical and molecular study on cardiac involvement during COVID-19 recently advocated a relationship between (hyper)inflammation and myocardial damage and showed that inflammatory/immune dysregulation caused by SARS-CoV-2-induced and high cytokine concentrations may be responsible for myocardial lesions and a worse prognosis. Analyzing autopsy cases is a fascinating study once more. By proving that SARS-CoV-2 receptors are expressed within cardiac cells and that cytokine-induced organ dysfunction contributes to the illness process, COVID-19 demonstrated that the SARS-CoV-2 genome was present in myocardial tissue. Last but not least, recent data indicate that COVID-19 patients without a history of cardiovascoular disease had pericardial effusion. These studies demonstrate that SARS-CoV-2 can cause direct or indirect cardiac damage that can have short-term, acute effects that cause cardiac dysfunction or long-term effects. The development of cardiac fibrotic tissue is a dangerous disease brought on by the uncontrolled inflammatory/immune state typical of the more advanced phases of SARS-CoV-2 infection. The cytokine storm and the abrupt and high release of proinflammatory mediators, such as Tumor Necrosis Factor Alpha (TNF-alpha) and Interleukin-1-beta (IL-1), are likely to blame for the development of cardiac fibrotic tissue. The pathological remodeling of the Extracellular Matrix (ECM) that results in structural defects and compromised heart function is known as cardiac fibrosis. Heart tissue function is hampered by excessive and persistent ECM deposition. In order to reduce mortality but also serious complications following the post-positive SARS-CoV-2 infection, in the convalescence period, it is crucial that the COVID-19 patient receive multidisciplinary evaluation and treatment, including assessment of the CV system conditions and the most appropriate therapy.