Commentary

Heart failure in present COVID-19 pandemic

Venkat A

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COMMENTARY

Patients with cardiovascular disease, including heart failure, are more susceptible to coronavirus disease 2019 (COVID-19), and their clinical course is more severe once infected. Increased troponin plasma levels indicate heart failure and myocardial injury in at least 10% of COVID-19 patients, with greater percentages (25 percent to 35 percent or more) when patients are seriously ill. Multiple mechanisms have been shown in patients with COVID-19 to cause myocardial injury, including those that occur with all severe infections, such as fever, tachycardia, and adrenergic stimulation, as well as those caused by an exaggerated inflammatory response, endotheliitis, and, in some cases, myocarditis.

The renin-angiotensin-aldosterone system may play a vital role. SARS-CoV-2 infects human cells by attaching to angiotensin-converting enzyme 2 (ACE2), an enzyme that converts angiotensin II into angiotensin 1-7, which has vasodilating and anti-inflammatory properties. Downregulation of ACE2 by the virus may boost angiotensin II stimulation and contribute to COVID-19's harmful hyper-inflammatory response. ACE2 may, on the other hand, be up-regulated in individuals with heart illness who are taking angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. For proper triage and management of these patients, a thorough grasp of the hemodynamic and diagnostic implications is required.

COVID-19 causes abnormal cardiac biomarkers by a variety of methods, including viral entry through the ACE2 receptors, direct cardiac damage, enhanced thrombotic activity, stress cardiomyopathy, and others. Many of the identified mechanisms and manifestations can be attributed to the cytokine storm seen in this pandemic. The two-way interaction between heart failure drugs and infection, as well as the planned COVID-19 medicines and heart failure, can lead to effective management.

Vaagdevi College of Pharmacy, Telangana, India

Correspondence: Venkat A, Vaagdevi College of Pharmacy, Telangana, India, E-mail: avenkat88@gmail.com
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