

## COVID 19 and Cardiovascular Disease: A Reality Check

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Coronavirus is highly infectious disease brought about by a new found severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). Coronavirus is a novel infection and it spread from China in a fast way and came to pandemic level and has put entire world on danger and high ready. At present shockingly, there are no particular vaccines or treatments for COVID-19 and current management incorporates isolation, travel limitations, personal care and supportive medical care. Researchers everywhere on the world are trying to find medications and immunizations for Coronavirus and critically the comprehension of basic pathobiology is required. A high infectivity and its capacity to get transmitted in any event, during asymptomatic stage and generally low virulence have brought about quick transmission of this infection past geographic areas, prompting a pandemic. As we all know that first instance of this disease, known as Coronavirus disease 2019 (Coronavirus 2019), was reported on December 8, 2019 in the Hubei area of China [1].

This disease is brought about by binding to viral surface spike protein to the human angiotensin-changing over compound 2 (ACE2) receptor after actuation of the spike protein by transmembrane protease serine 2 [2]. ACE2 is which is communicated in the lungs (primarily type II alveolar cells [3]) which has had all the earmarks of being the most significant gateway of section. ACE2 is communicated in the heart too so it prompts check the impacts of angiotensin II in conditions which lead to actuation of the renin-angiotensin framework, for example, hypertension, congestive cardiovascular breakdown, and atherosclerosis. Other than the heart and lungs, ACE 2 is likewise communicated in the intestinal epithelium, vascular endothelium, and kidneys, which provides a component for the multiorgan brokenness that is seen with SARS-CoV-2 infection [4,5]. At present this proof is connecting Coronavirus with expanded horribleness and mortality from cardiovascular illness (CVD).

This infection is caused by binding to viral surface spike protein to the human angiotensin-converting enzyme 2 (ACE2) receptor after activation of the spike protein by transmembrane protease serine 2 [2]. ACE2 is which is expressed in the lungs (principally type II alveolar cells [3]) which has appeared to be the most important portal of entry. ACE2 is expressed in the heart as well so it leads to counteract the effects of angiotensin II in conditions which lead to activation of the renin-angiotensin system, such as hypertension, congestive heart failure, and atherosclerosis. Besides the heart and lungs, ACE 2 is also expressed in the intestinal epithelium, vascular endothelium, and kidneys, which provides a mechanism for the multiorgan dysfunction that is seen with SARS-CoV-2 infection [4,5]. At present this evidence is linking COVID-19 with increased morbidity and mortality from cardiovascular disease (CVD).

Clinical manifestation	Incidence
Cardiovascular abnormality (increased cardiac troponin I)	8 - 12%
Heart failure	52% in those who died and 12% in those who recovered
Acute cardiac injury	59% in those who died and 1% in those who recovered
Arrhythmia	16.7%
Acute cardiac injury	7.2%

**Table 1:** Cardiovascular complications in COVID-19 [6-8].

The common mechanisms which are postulated to be responsible for CV complications in COVID-19 are [7,9-12]:

1. **Direct myocardial injury:** SARS-CoV-2 binds to ACE2 which resulted in alteration of ACE2 signaling pathways and finally it leads to acute myocardial and lung injury.
2. **Inflammatory response:** More extreme types of Coronavirus are described by intense fundamental incendiary reaction and cytokine storm, which can bring about injury to numerous organs prompting multiorgan dysfunction. Studies have indicated high circulatory degrees of proinflammatory cytokines in patients with severe/critical COVID-19.
3. **Myocardial demand-supply ratio:** High cardiometabolic request related with the fundamental disease combined with hypoxia brought about by intense respiratory sickness can weaken myocardial oxygen demand -supply relationship and lead to intense myocardial injury.
4. **Plaque rupture and thrombosis:** Systemic inflammation and increased shear stress because of increased coronary blood flow can lead to plaque rupture which will lead to acute myocardial infarction. Prothrombotic milieu created by systemic inflammation further is itself a high risk as well.
5. **Adverse effects of treatment:** Different antiviral medications, corticosteroids and different treatments pointed toward treating Coronavirus can adversely affect the CV framework.
6. **Electrolyte imbalances:** Electrolyte imbalances can happen in any basic ailment and lead to arrhythmias, especially in patients with basic cardiovascular disease. There is specific concern about hypokalemia in Coronavirus, because of cooperation of SARS-CoV-2 with renin-angiotensin-aldosterone network. Hypokalemia may lead to different tachyarrhythmias.

Albeit respiratory sickness is the predominant clinical appearance of Coronavirus, the sheer weight of the ailment infers that countless patients with Coronavirus would give previous history of CVD or develop cardiovascular disease over the span of the disease. The current comprehension about the interplay between CVD and Coronavirus is not fully understood at present. The diagnostic and therapeutic difficulties presented by the concurrence of these two illnesses should be sufficiently considered.

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