



The enigma of cardiovascular diseases during the COVID-19 pandemic

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Letter to Editor

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Dear Editor

The tragic story called 'COVID-19 History' started in Wuhan, China, in late February 2019. The newly identified severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) or coronavirus disease 2019 (COVID-19) is the protagonist of this story, which so far has resulted in more than 3.2 million deaths around the world. The virus has made a great influence on different aspects of human life, and clinical efforts for disease control and treatment are taking longer than expected. Several factors including limited knowledge on the virus pathogenicity and inadequate health care services have contributed to virus spread, and impeded containment efforts. Acute respiratory failure is the most common clinical manifestation of severe COVID-19; nonetheless, it can damage many other organs including kidney and heart.¹

The broad tissue tropism of SARS-CoV-2 is associated with the presence of cell surface receptors which determine the accessibility of the virus to the host tissues and organs. Angiotensin-converting enzyme 2 (ACE2) is the main entry receptor for SARS-CoV-2, and their extensive distribution in many cell types and tissues including the lungs, heart and blood vessels could explain the multi-organ dysfunction observed in infected patients. ACE2 interact with coronavirus spike protein which is responsible for virus attachment and infection of host cell. A powerful expression of ACE2 in the heart and coronary arteries is thought to be one of the major factors of cardiac vulnerability to SARS-CoV-2 infection.

The cardiotropism of SARS-CoV-2 has been demonstrated in several studies both in patients with and without cardiovascular diseases. For instance, Pietsch et al. showed the presence of SARS-CoV-2 genomes in endomyocardial biopsies (EMB) of infected patients with symptoms of heart failure.² Moreover, viral-related particles were detected in interstitial cytopathic macrophages in an

EMB from a patient with COVID-19 who developed cardiogenic shock.³

Viral tropism seems to influence SARS-CoV-2 disease progression and cardiac organ damage. This opinion is supported by evidence such as a higher risk of diverse cardiovascular diseases in infected patients and elevated levels of cardiac injury biomarkers including troponins.⁴

Unfortunately, elderly patients with COVID-19 have a higher mortality rate, and the risk of death increases sharply with the presence of comorbidities that occur naturally in older adults. A number of studies also indicate that hypertension, diabetes, and ischemic heart disease are among the top three COVID-19 comorbidities, especially in the elderly.⁵

Although the cause of cardiovascular complications is unknown, it is widely thought to be due to direct myocardial damage which associates with abundant expression of ACE2 in the heart and coronary vessels.

The presence of these receptors is reduced through several mechanisms including the accelerated rate of receptor internalization or extensive viral lysis of ACE2-expressing cells. Downregulation of ACE2 levels, and ACE/ACE2 ratio alteration is accompanied by different pathological conditions, especially those of the cardiovascular system.

In addition to direct SARS-CoV-2 infection of the heart, virus may damage the heart indirectly. These indirect effects have been attributed to a number of mechanisms, such as abnormal immune responses to infection or reduced supply of oxygen to cardiovascular tissue due to pneumonia.⁵

Altogether, the cardiovascular system can be affected by SARS-CoV-2 even among people who have never displayed heart symptoms.

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Cardiovascular abnormalities are usually associated with significantly worse outcome or prognosis of COVID-19. Therefore, particular attention should be given to identification and treatment of cardiovascular deficits in infected patients with COVID-19.

Conflict of Interests

Authors have no conflict of interests.

Authors' Contribution

ZJ: Approval of the final version of the manuscript, Elaboration and writing of the manuscript, Critical review of the literature, Critical review of the manuscript

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