



2019–Novel Coronavirus-Related Acute Cardiac Injury Cannot Be Ignored

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Abstract

Purpose of the Review Coronavirus disease 2019 (COVID-19), a new infectious disease caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has reached a pandemic status. Although SARSCoV-2 causes primarily respiratory problems, concurrent cardiac injury cannot be ignored since it may be an independent predictor for adverse outcomes. To resolve these issues, we aim to summarize the prevalence and its underlying mechanisms of acute cardiac injury in the setting of SARS-CoV-2 infection.

Recent Findings The main clinical manifestation of SARS-CoV-2 infection is pneumonia, cardiovascular complications have also been identified in the earliest reported cases from Wuhan, the epicenter of the outbreak. Given the SARS-CoV-2 likely uses the angiotensin-converting enzyme-2 (ACE2) receptors as its host receptor, ACE2-related signaling pathways may play a key role in mediating myocardial injury.

Summary SARS-CoV-2 infection related acute cardiac injury cannot be ignored, and its underlying mechanisms remain speculated. We would suggest that health professionals investigate cardiac function as part of the routine care.

Keywords COVID-19 · SARS-CoV-2 · 2019-nCoV · Cardiac injury · Angiotensin-converting enzyme-2

The emergence of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) epidemic is a major global public health concern. Following the initial outbreak as first reported in Wuhan, Hubei Province of the People's Republic of China [1], person-to-person transmission has already occurred in Mainland China [2–4], and in other parts of the world [5]. While SARS-CoV2 is primarily a respiratory infection, cardiac injury induced by the virus cannot be neglected. A recent study of 41 patients reported a 12% (5/41) incidence of virus-related acute cardiac injury and 33% (13/40) of elevated serum cardiac biomarker levels [6]. Another study reported a similar rate of 15% of the patients showing elevated cardiac biomarkers [7]. Wang and colleagues found that 7%

(10/138) of patients with 2019-nCoV infections developed acute cardiac injury, of whom eight were admitted to the intensive care unit (ICU), accounting for 22% of the total number of ICU patients [8]. The same study also reported 17% (23/138) of the patients developed cardiac arrhythmias. Together, data from these studies illustrate that the 2019-nCoV infection results in varying degrees of myocardial damage, which may be explained by adverse outcomes observed.

The prognosis of those with 2019-nCoV is determined by different factors. Approximately half of those infected had multiple comorbidities, mainly cardiovascular and cerebrovascular diseases [8]. Huang et al. pointed out that at baseline, the ICU care group have a higher frequency of underlying diseases, such as cardiovascular disease (23% vs. 11%) compared with those who did not need ICU care [6]. Similarly, Wang et al. reported higher frequency of hypertension (58% vs. 22%) and cardiovascular disease (26% vs. 11%) [8]. Mechanistically, similar to the severe acute respiratory syndrome (SARS)-CoV [9], the 2019-nCoV likely uses the angiotensin-converting enzyme-2 (ACE2) receptors as its host receptor. Given that ACE2 receptors are widely expressed in the cardiovascular system, theoretically these sites are potential targets for 2019-nCoV infection. Indeed, ACE2-related signaling pathways may play a key role in mediating myocardial injury. Cytokine storms, which are

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associated with an imbalance between types 1 and 2 helper T cells, may explain the severe phenotype of the disease and mediate myocardial damage. Prognosis may be improved by therapies that reduce the inflammatory response. Recent work has found that transplantation of ACE2⁻ mesenchymal stem cells by the intravenous route led to improvement in symptoms and pulmonary function of 2019-nCoV patients [10]. As noted by the accompanying editorial [11], the mechanisms likely involve reduced overactivation of the immune system, as reflected by lower tumor necrosis factor- α and C-reactive protein levels, increased peripheral lymphocytes and CD14⁺CD11c⁺CD11b^{mid} regulatory dendritic cells, and disappearance of the cytokine-secreting immune cells CXCR3⁺CD4⁺ T cells, CXCR3⁺CD8⁺ T cells, and CXCR3⁺ natural killer cells [10].

In conclusion, the clinical course of patients harboring the 2019-nCoV infection remains to be fully elucidated. We would suggest that health professionals investigate cardiac function as part of the routine care, and the data may shed light on whether myocardial damage is an independent predictor of adverse outcomes.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflicts of interest.

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