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About SARS-CoV-2 and Heart Problems in COVID Patients

Masataka Nishiga*

Department of Medical Surgical and Health Science, University of Trieste, Italy

Editorial

Millions of confirmed cases and deaths have been reported worldwide as a result of the coronavirus disease 2019 (COVID-19) pandemic, which was caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) viruses. Rapid and large-scale testing is essential for patient management and disease control; hence effective diagnostic devices are in high demand. Current SARS-CoV-2 detection technologies in clinical laboratories, as well as breakthroughs in molecular, antigen-based, and immunological point-of-care testing, as well as new sensor and biosensor device developments. It is emphasised the importance of specimen collecting time and kind, as well as issues such as illness prevalence, setting, and methods [1]. The various techniques' mechanisms of action, as well as their application breadth and known performance characteristics, are detailed. The use of diagnostic imaging technologies and biomarkers for assessing COVID-19 or monitoring the degree or implications of the disease is also discussed.

While the literature on SARS-CoV-2 is still growing, this summary highlights some of the most fascinating events from the pandemic, as well as the lessons learned. Exploring a variety of methods for detecting SARS-CoV-2 will ensure that clinicians, public health, and infection prevention and control continue to get diagnostic support during the pandemic, as well as provide guidelines for future pandemic preparedness. Angiotensin-converting enzyme 2 (ACE-2) is the entry receptor for the acute respiratory distress syndrome coronavirus-2 (SARS-CoV-2), which causes Coronavirus Disease-2019 (COVID-19) in humans (ACE2). The vascular endothelium, alveolar type 2 lung epithelial cells, renal tubular epithelial cells, testes Leydig cells, and the gastrointestinal system all include ACE-2, a type-I trans membrane metallocarboxypeptidase [2]. ACE2 mediates the interaction between host cells and the SARS-CoV-2 spike (S) protein. ACE2, on the other hand, is a homeostatic regulator of the renin angiotensin system (RAS), which is important for both the cardiovascular and immune systems. As a result, ACE2 plays an important role in the transmission of SARS-CoV-2, cardiovascular disease (CVD), and the immune system. The availability of ACE2, which is regulated by genetics, age, gender, and comorbidities, appears to be intimately associated to SARS-CoV-2 vulnerability.

Because of an uncontrolled and overwhelming immune response, COVID-19 produces severe respiratory distress syndrome (ARDS) and multiorgan failure. Despite reduced ACE2 expression on the cell surface, patients with CVDs had a higher COVID-19 mortality rate. This is most likely due to an imbalance between the ADAM metallopeptidase domain 17 (ADAM17) proteins (which is required for cleavage of the ACE-2 ectodomain, resulting in increased ACE2 shedding) and the TMPRSS2 protein (which is required for cleavage of the ACE-2 ectodomain, resulting in increased ACE2 shedding) (which is required for spike glycoprotein priming). In patients with chronic comorbidities, treatment interruption of ACE inhibitors and Angiotensin II Receptor Blockers (ARBs) appears to be justified. Researchers may now explore the effects of different COVID-19 vaccinations on ACE2 in patients receiving ACEi/ARB therapy because to the availability of COVID-19 vaccines [3].

Coronavirus disease 2019 (COVID-19) is a global pandemic with far-reaching consequences for the cardiovascular health of millions of people who survive infection. COVID-19's etiologic agent, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), can infect the heart, vascular tissues, and circulation cells via ACE2, the viral spike protein's host cell receptor. Acute heart injury is a typical COVID-19 extra pulmonary symptom that might have long-term consequences. This update discusses the clinical signs of cardiovascular involvement, proposed direct and indirect SARS-CoV-2 immune response pathways impacting the cardiovascular system, and implications for patient management after recovery from acute COVID-19 infection. Coronavirus disease 2019 (COVID-19), caused by a coronavirus strain known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has infected billions of individuals around the world [4]. The cell entrance pathway, which is initiated by the viral spike protein binding to angiotensin-converting enzyme-2, is shared by SARS-CoV-2 and SARS-CoV, the zoonotic virus that caused the 2002 severe acute respiratory syndrome outbreak. Clinical studies have also revealed a relationship between COVID-19 and cardiovascular disease. Despite the fact that COVID-19 can cause myocardial injury, arrhythmia, acute coronary syndrome, and venous thromboembolism, it appears to be linked to worse outcomes and an increased risk of death in people with pre-existing cardiovascular disease. Patients with COVID-19 and other cardiovascular diseases are becoming increasingly concerned about drug–disease interactions [5].

COVID-19's current state of knowledge, from basic mechanisms to clinical perspectives, with an emphasis on COVID-19's interaction with the cardiovascular system. By combining our knowledge of the virus's biological features with clinical results, we can gain a better understanding of the virus's possible processes, opening the way for the creation of prophylactic and therapeutic treatments. To date, seven coronaviruses (CoVs) have been documented to infect humans, with three particularly lethal strains appearing in the twenty-first century. SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), the newest member of this family, was identified near the end of 2019 in China's Hubei province. Since then, this one-of-a-kind coronavirus has spread around the world. Clinical manifestations range from asymptomatic to mild respiratory tract infections and influenza-like illness to severe disease with lung injury, multi-organ failure, and death. Despite the fact that SARS-CoV-2 is thought to replicate in the lungs, infected patients frequently report other symptoms, implying involvement of the gastrointestinal tract, heart, circulatory system, kidneys, and other organs; thus, the question of whether COVID-19 is a respiratory or systemic illness arises. The purpose of this review is to consolidate existing data on SARS-CoV-2 replication in different tissues in both patients and healthy people.

Conflict of Interest

The authors declare that there is no conflict of interest associated with this manuscript.

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***Address for Correspondence:** Masataka Nishiga, Department of Medical Surgical and Health Science, University of Trieste, Italy, E-mail: Mnishiga@gmail.com*

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