Opinion
Volume 11:10, 2022

Journal of Hypertension: Open Access

ISSN: 2167-1095 Open Access

Hypertension Issue in Covid 19 Patients

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Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) viruses triggered the coronavirus disease 2019 (COVID-19) pandemic, which resulted in millions of confirmed cases and deaths worldwide. For patient management and disease control, rapid and extensive testing is necessary; As a result, there is a high demand for efficient diagnostic tools. New sensor and biosensor device developments, advancements in molecular, antigen-based, and immunological point-of-care testing, and current SARS-CoV-2 detection technologies in clinical laboratories are also noteworthy. Time and type of specimen collection, as well as issues like illness prevalence, setting, and methods, are emphasized [1]. The various techniques' mechanisms of operation, application range, and well-known performance characteristics are all described in detail. It is also discussed how diagnostic imaging technologies and biomarkers can be used to evaluate COVID-19 or track the disease's severity or consequences.

Description

This summary highlights some of the most fascinating events from the pandemic as well as the lessons learned, despite the fact that the body of research on SARS-CoV-2 is still expanding. During the pandemic, clinicians, public health, and infection prevention and control will continue to receive diagnostic support, and guidelines for future pandemic preparedness will be provided by investigating a variety of methods for detecting SARS-CoV-2. The entry receptor for the acute respiratory distress syndrome coronavirus-2 (SARS-CoV-2), which is responsible for human Coronavirus Disease-2019 (COVID-19), is angiotensin-converting enzyme 2 (ACE2). ACE-2, a type-I trans membrane metallocarboxypeptidase, is found in the vascular endothelium, alveolar type 2 lung epithelial cells, renal tubular epithelial cells, testes Leydig cells, and the gastrointestinal system [2]. The SARS-CoV-2 spike (S) protein and host cells interact through ACE2. On the other hand, ACE2 is a homeostatic regulator of the renin angiotensin system (RAS), which is crucial for the immune and cardiovascular systems. SARS-CoV-2 transmission, cardiovascular disease (CVD), and the immune system are all influenced by ACE2. SARS-CoV-2 susceptibility appears to be intimately linked to the availability of ACE2, which is influenced by genetics, age, gender, and comorbidities.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].

The global pandemic of coronavirus disease 2019 (COVID-19) has far-reaching effects on the cardiovascular health of millions of people who survive infection. Through ACE2, the host cell receptor of the viral spike protein, the etiologic agent of COVID-19, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) can infect the heart, vascular tissues, and circulation cells. A typical COVID-19 extra pulmonary symptom with the potential for long-term consequences is acute heart injury. The clinical signs of cardiovascular involvement, the proposed direct and indirect SARS-CoV-2 immune response pathways that impact the cardiovascular system, and the implications for patient management following acute COVID-19 infection are discussed in this update. The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) strain of coronavirus that causes coronavirus disease 2019 (COVID-19) has infected billions of people worldwide [4]. SARS-CoV-2 and SARS-CoV, the zoonotic virus that caused the 2002 outbreak of severe acute respiratory syndrome, share the cell entrance pathway, which is initiated by the viral spike protein binding to angiotensin-converting enzyme 2. COVID-19 has also been linked to cardiovascular disease, according to clinical studies. Even though COVID-19 has the potential to cause myocardial injury, arrhythmia, acute coronary syndrome, and venous thromboembolism, it appears to be associated with worse outcomes and a higher risk of death in people who already have cardiovascular disease. Drug–disease interactions are raising concerns among COVID-19 and other cardiovascular disease patients [5].

Conclusion

The current state of knowledge regarding COVID-19, from its fundamental mechanisms to clinical perspectives, with a focus on how it interacts with the cardiovascular system. We can better understand the virus's possible processes by combining our understanding of its biological characteristics with clinical findings, paving the way for the development of therapeutic and preventative treatments. Seven coronaviruses (CoVs) have been shown to infect humans to date, with three particularly lethal strains emerging in the 21st century. The most recent member of this family, SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), was discovered in China's Hubei province near the end of 2019. This one-of-a-kind coronavirus has since spread worldwide. Asymptomatic respiratory tract infections, influenza-like illness, and severe disease with lung injury, multi-organ failure, and death are all examples of clinical manifestations. In spite of the fact that it is believed that SARS-CoV-2 reproduces in the lungs, patients who are infected frequently report other symptoms, suggesting that the gastrointestinal tract, heart, circulatory system, kidneys, and other organs are involved; As a result, the issue of whether COVID-19 is a systemic or respiratory illness arises. This review aims to consolidate existing data on SARS-CoV-2 replication in various tissues of healthy and sick individuals.

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Received: 01 October, 2022, Manuscript No. jhoa-23-85983; **Editor assigned:** 04 October, 2022, Pre QC No. P-85983; **Reviewed:** 18 October, 2022, QC No. Q-85983; **Revised:** 24 October, 2022, Manuscript No. R-85983; **Published:** 01 November, 2022, DOI: 10.37421/2167-1095.2022.11.370

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How to cite this article: Nishiga, Masataka. "Hypertension Issue in Covid 19 Patients." J Hypertens 11 (2022): 370.