Review Article

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Role of Ace Inhibitors in Covid-19 Disease Progression with Underlying Cardiovascular Diseases and Hypertension

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Abstract

Background: Coronaviruses are a family of viruses that can cause illnesses such as the common cold, severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS). In 2019, a new coronavirus was identified as the cause of a disease outbreak that originated in China. The virus is now known as the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The disease it causes is called coronavirus disease 2019 (COVID-19). In March 2020, the World Health Organization (WHO) declared the COVID-19 outbreak a pandemic.[1]

Rationale: With the current changes in the lifestyle it has been observed that the Non communicable diseases have increased in the past decade. India has an estimated 77 million people with diabetes, which makes it the second most affected in the world and the numbers are expected to increase according to International Diabetic Federation. National Family Health Survey says that approximately 207 million people (men 112 million, women 95 million) are suffering from hypertension in our country and the numbers are projected to grow. According to a study conducted by Lancet in 2016 around 23.8 million prevalent cases of ischemic heart disease and 6.5 million cases of stroke were estimated in India. It has been observed lately that patients with Covid-19 along with an underlying disease (Hypertension, CVDs, diabetes) might have an increased risk of death. A sizeable majority of the Indian population is afflicted with the diseases, therefore it is crucial to understand the proposed etiopathogenesis of these comorbidities Understanding the damage caused by SARS-CoV-2 and their underlying mechanisms is of the great importance, so that treatment of these patients can be done effectively and the mortality can be reduced. In this article we will be discussing how Covid-19 infection affects patients with pre-existing hypertension, cardiac conditions, and diabetes.

Method: Thorough review of articles from Google Scholar and PubMed till July 15, 2020 was done with keywords "SARS-CoV-2", "COVID-19", "nCoV 2", "pathophysiology","cardiovascular disease", "hypertension" "myocardium", "symptoms", "diagnosis", "prognosis", "management".

Conclusions: Suggestions are made on the possible pathophysiological mechanisms of CVD in patients suffering from COVID-19, and its management. No firm conclusions can be drawn based on limited evidence that we possess. Though further research between the possible relationship of COVID-19 and cardiovascular damage is warranted.

Keywords: "SARS-Cov2"; "Hypertension"; "Diagnosis"; "Treatment"; "Cardiovascular diseases"; "Hypertension".

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Introduction

At the beginning of 2020 the World Health Organisation declared COVID-19 as a global pandemic. It was first seen in China and in the

months after that spread rapidly to engulf all countries around the globe. COVID-19 is caused by severe acute respiratory syndrome coronavirus 2, which enters the cells through the angiotensin-converting enzyme 2 receptor. Among patients with COVID-19, there is a high prevalence of cardiovascular disease, many patients experience myocardial injury from the infection. COVID-19 is primarily a respiratory illness but cardiovascular involvement can occur through several mechanisms. This article shall elucidate the difference in suspected pathophysiology of covid-19 infection among patients with underlying conditions as compared to the other patients with no underlying disease.

Covid-19 and its Effects on Hypertension: Hypertension has emerged as one of the most pertinent comorbidities for Covid-19. Severe acute respiratory syndrome coronavirus (SARS-CoV) and (SARS-CoV-2) bind to their target cells through angiotensin-converting enzyme 2 (ACE2), which is expressed by epithelial cells of the lung, intestine, kidney, and blood vessels. There is a propensity for ACE-2 to be strongly expressed in the bronchial epithelium.

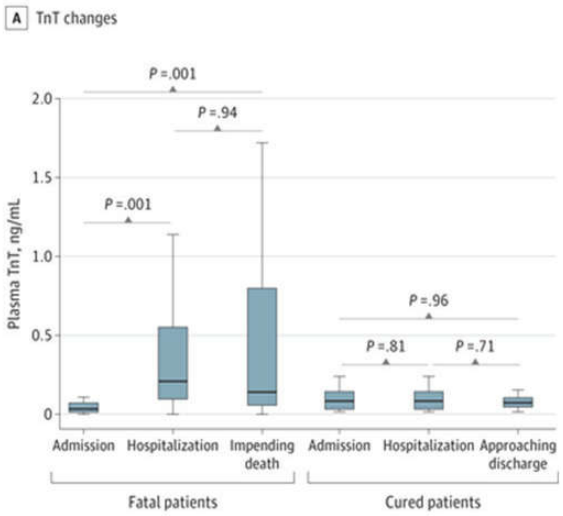
Tissue-specific and circulating components of the RAAS make up a complex intersecting network of regulatory and counter regulatory peptides. ACE2 is a key enzyme that cleaves angiotensin II to angiotensin-(1–7), thereby diminishing its effects on vasoconstriction and sodium retention in turn bringing down the blood pressure. After endocytosis of the viral complex surface ACE-II is further downregulated resulting in unopposed Angiotensin–II accumulation. Increased levels of Angiotensin–II in blood leads to acute lung injury, adverse myocardial remodeling, vasoconstriction, vascular permeability Human pathogenic coronaviruses (severe acute respiratory syndrome coronavirus (SARS-CoV) and SARS-CoV-2) attach themselves to the intended cells that they are targeting through angiotensin-converting enzyme 2 (ACE2), which is expressed by epithelial cells of the lung, intestine, and blood vessels.[2]

After nCoV2 binds to the Angiotensin Converting Enzymne 2 , the amount of ACE2 is depleted and the ACE2/Ang (1-7)/Mas receptor pathway is disturbed leading to adverse events because the catalysis of Angiotensin 2 does not take place (Physiologically ACE 2 is responsible for the cleaving Angiotensin 2 into Angiotensin 1,7 and bringing down the blood pressure) . The delicate balance of the Renin Angiotensin - Aldosterone system is disrupted, and this leads to the aggravation of pneumonia. It is thus hypothesized that Angiotensin Converting Enzyme Inhibitors and Angiotensin 2 Receptor blockers can be used in patients with COVID-19 pneumonia to manage hypertension and bring down the pulmonary inflammation

Covid-19 and its Effects on Cardiac Patients: The number of cases of Covid 19 is growing daily and the cardiovascular changes seen in these patients is quite alarming .According to the data presented by National Health Commission of China about 11% of the patients who died because of Covid 19 showed considerable damage of cardiac tissue. These patients did not have any prior history of cardiovascular ailments .This shows that the incidence of CVD is high in patients suffering from Covid 19. It can be attributed to the systemic inflammation caused by the virus followed by a cytokine storm.[5]

It was observed that patients with Cardiovascular diseases had a higher level of TnT (Troponin T) levels. Troponin T is a part of the troponin complex which are proteins vital for the contraction of skeletal and heart muscles. Troponin T binds to tropomyosin and help position it on actin. Together with the rest of the troponin complex it modulates the contraction of striated muscles. The cardiac subtype of troponin T is released in the bloodstream in case of any damage to the cardiac muscle.[6]

Patients with higher TnT levels were more prone to complications while hospitalized including Acute Respiratory Distress Syndrome (ARDS) and malignant arrhythmias. The patients suffering from cardiovascular diseases are at a greater risk and more often than not the virus targets the myocardium. The mechanisms of disease progression may have some relation to the destabilized plaques and hypoxia observed in such cases. Patients with pre existing cardiovascular disease but normal Troponin T levels had a better prognosis. The patients suffering from cardiovascular disease should be carefully monitored and their myocardial biomarkers recorded periodically for better mangement. Patients with underlying cardiovascular disease are more prone to develop critical outcomes therefore there is a need to triage these patients in order to provide them with preferential treatment and more aggressive management strategies.



Source: JAMA Cardiol (TnT- troponin T)

Dynamic Changes of TnT during Hospitalization. The horizontal lines represent the median value in each group.

Conclusion

Underlying Cardio vascular disease and/or development of acute cardiac injury are associated with significantly worse outcome in these patients. Information about other cardiovascular manifestations is very limited at present.CVD patients with higher levels of troponin T were more susceptible to adverse outcomes after being infected with SARS CoV2. The exact mechanism of disease progression is not very clear as of now. The available clinical data from the pandemic till date is insufficient to reach a final conclusion. Although no therapy is currently established for SARS-CoV-2 patients, the field is moving rapidly with potential approaches being considered.

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