iJRASET
International Journal For Research in Applied Science and Engineering Technology

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 8 **Issue:** XI **Month of publication:** November 2020

DOI: https://doi.org/10.22214/ijraset.2020.32118

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International Journal for Research in Applied Science & Engineering Technology (IJRASET)
ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.429
Volume 8 Issue XI Nov 2020- Available at www.ijraset.com

Coronavirus Impact on Cardiovascular System of Body - Review

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Abstract: Objectives: The coronavirus disease-2019(COVID-19) has caused the worst global pandemic, which affected nearly 214 countries affecting nearly 5.5 million people. In this review paper the effect of COVID-19 on cardiovascular system has been reviewed. We reviewed the current published research papers on the different cardiac manifestations and the correlation between virus to disease severity.
Methods: We searched the ScienceDirect, PubMed, Elsevier, Web of Science databases and Google Scholar for original research articles that are published. We selected papers that have sufficient data and information regarding their research,
Results: We analyzed multiple research papers on CVD complications due to COVID-19, and we summarized how COVID-19 has impacted people who already have cardiovascular issues, and also how COVID impacted people who do not have existing CVD issues, and also different types of cardio issues that could possibly arise due to COVID-19.

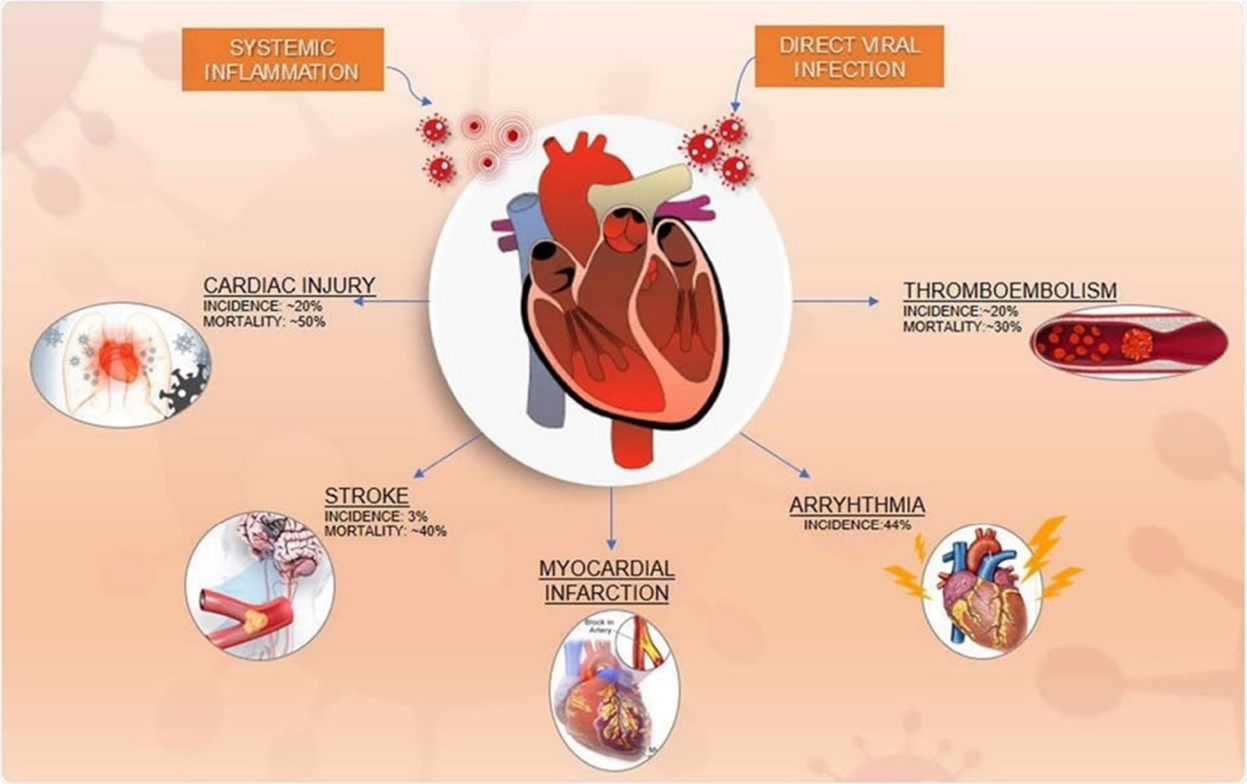
I. INTRODUCTION

The name Coronavirus is given for its spikes which look like crowns on their surface and belong to the Coronavirinae subfamily. By means of phylogenetic clustering, this virus is classified again into four types: the α, β, γ, and δ CoVs. α and β are the types of viruses that cause infection in humans [1]. Coronaviruses contain four major structural proteins: the nucleocapsid (N) protein, the membrane (M) protein, the spike (S) protein, and the envelope (E) protein [2].
In the 1960s the first human Coronavirus (HCoV) was identified, and only 2 HCoV species were recognized until 2003, which are HCoV-229E and HCoV-OC43. Currently, 7 different Coronaviruses are known to infect humans mostly causing infections that is self-resolvable, including HCoV-229E, HCoV-NL63, HCoV-OC43, and HCoV-HKU1. There are also other types of Coronaviruses one is Middle East Respiratory Syndrome coronavirus (MERS-CoV), and other is severe acute respiratory syndrome coronavirus (SARS-CoV), and the newly identified SARS-CoV-2, which can cause respiratory infections in humans [3,4].

A. SARS-CoV-2, Causing COVID-19

SARS-CoV first outbreak has started in south China in 2002, and was associated with wild animals' market in China, and after this virus SARS-CoV, a similar kind of virus was found in raccoon dogs with 99.8% nucleotide homology to human, in Himalayan palm civets [30]. In 2019, December local health hospitals of Wuhan has reported clusters of pneumonia that were linked to sea food and live-animal market. And in January 2020 a novel coronavirus initially is named as 2019-CoV and is identified as the reason for the outbreak of viral pneumonia. This viral disease was named COVID-19 [31]. SARS CoV-2 genetically similar to 82% with human SARS-Cov , and β-CoVs group that has 89% nucleotide identity [32,33]. And it is less similar to SARS-CoV [34].
In many ways like clinically, epidemiologically, and in laboratory findings, it was figured that this SAS-CoV can cause infection similar to SARS-CoV infection in 2003 [35]. The main sources of transmission for COVID-19 is person to person contact, and through droplets of saliva, with incubation period(mean) of 5.2 days, and 95th percentile of distribution at 12.5 days [35,36]. SARS-CoV 2 infection primarily happens in adults, and only fewer cases were reported in children who are 15 years or younger [36, 37,38] .As per World Health Organization by March 3,2020 that global mortality rate was about 3.4%.The mortality rate in China has been nearly 3.8% [39]. When a different method of mortality estimation is used to calculate mortality rate by using the formula number of deaths by number of cases diagnosed 14 days prior yields a global mortality of 5.7% [40]. The overall attack rates for other modes of transmission for the virus in patients with COVID-19 is 10.5% for household members and 0.45% for close contacts [41].
Common symptoms of COVID-19 are fever, shortness of breath and cough. Some symptoms are less common which includes dyspnea, headache, malaise, sore throat, anorexia. Symptoms sometimes can appear in 2 days or might take up to 14 days after being exposed to virus [42].

B. Cardiovascular Complications in Patients with COVID-19



C. COVID-19 - Prevalence of Cardiovascular Disease

Studies from the past proved that, Cardiovascular Disease was common comorbidity in patients with MERS and SARS. In a case study that was done on 144 patients with SARS examined the prevalence of DM, cancer and CVD at a rate of 11%,6% and 8% respectively [43]. In another 12 study analysis done on patients infected with MERS, which includes 637 patients was found that HT and DM are found in 50% of the cases, and CVD in approximately 30% of the patients [44].The higher percentage of CVD due MERS may be due higher age group compared to SARS which is 50 years versus 39.9 years [45].In a study done by [46] with 41 patients infected with COVID-19 15% of the patients had CVD problems which is 6 people out of 41. In another study done by the author [7] with cohort size of 138. 20 patients were observed to have CVD issues due to coronavirus. In a study with cohort size of 150 done by [47] 13 people out of 150 were facing CVD problems which is about 8.7%.

D. Cardiac Arrhythmia

The risk of occurrence of ventricular arrhythmia has been reported as high due to high influenza activity [5]. In a study that was conducted on 121 patients of SARS by [6], it was found that tachycardia was the most common issue in 72% of the total patients ,and other complications like hypotension and bradycardia were reported as 50% and 15% respectively. Most of these patients do not have any symptoms of the complications. In the 138 COVID-19 patients that were hospitalized, cardiac arrhythmia was reported in 16.7% of the total patients, and it was more common in ICU patients than non -ICU patients which is 44.4% versus 6.9% [7]. In another study it was reported that patients with myocardial injury has higher incidence of ventricular tachycardia [8].

[16] A retrospective study of 187 patients has highlighted the links of CVD and myocardial injury with fatal outcomes in COVID-19. 5.9% of the patients, which means 11 patients are reported ventricular tachycardia. Patients who have higher levels of cardiac troponin (Tn) levels had high incidence of arrhythmias than the ones with normal Tn levels, and the percentages are 11.5% versus 5.2%.

[17] Analyzed 85 fatal cases in COVID 19 patients and concluded that 60% of the patients have some form of arrhythmia and the cause of death in 10% of the patients was malignant arrhythmia. As the infection develops the risk of arrhythmia is likely to increase. A study by [18] reveals that cardiac arrhythmias was the reason for hospital mortality. A study made by [19] reported that a Caribbean male who was admitted with tachycardia and atrial flutter with 2-to-1 atrioventricular block, this has transitioned into atrial fibrillation with rapid ventricular response.

E. Myocardial Injury

In patients infected with COVID-19 especially in extreme cases, high amount of cardiac biomarkers have been noticed. In recent COVID cases it was reported that, even though patients do not have any respiratory symptoms or issues, cardiac involvement as a complication has been noted [9 ,10]. Myocardial injury, described as an increase in high-sensitivity cardiac troponin I (hs-cTnI) levels (>28 pg/mL).These high levels were found in five of the 41 patients (which is 12%) with COVID-19 in Wuhan [11]. Even in this study the incidence of Myocardial injury was higher in ICU patients.

[12] reported that in 416 hospitalized patients due to COVID 19, 57 of them died, and 10.6% of the patients had coronary heart disease, around 5.3% had cerebrovascular disease, and 4.1% had coronary heart disease. In 20% of the patients hs-TNI greater than the 99% percentile which is the upper limit, and these patients with elevated hs-TNI were of older age, and had more comorbidities, and had higher levels of leukocytes, N-terminal pro-brain natriuretic peptides [12].

A small study which includes 41 Patients infected by COVID 19 reported that patients who are critical in condition they had an increase of troponin I levels and also low density of epicardial adipose tissue that is detected in CT scan indicating cardiac inflammation [13]. From a study report that was conducted by authors in China had reported that nonsurvivors had a late increase of hs-cTnl. Inflammatory markers, including IL-6, D-dimer, and lactate dehydrogenase, also showed the same trend, which provides a possible mechanism for acute myocardial injury due to myocarditis or stress-cardiomyopathy [14,15].

F. Heart Failure and Cardiogenic Shock

Heart failure due to pneumonia is very common in patients admitted in hospitals. But several recent studies identified that COVID-19 has caused heart failure in multiple patients [20,21,22]. One of the studies on heart failure due to COVID-19 which involves 191 patients , and it was found that 44 of these patients had heart failure and the mortality rate is 64%, more than half of the patients who died among 191 was due to heart failure [14].

[23] reported that SARS-CoV-2 localization in myocardium led to cardiogenic shock. A study on patients from Spain reported that 3 patients out of 4 who developed cardiogenic shock died, leading to a mortality rate of 75%.and another fact to note here is that three of the patients who died does not have any comorbidities or cardiovascular risk factors. [24] a study from USA and Germany also specified that cardiogenic shock is a major complication of COVID-19.[25, 26] these studies also reported that 8% of the patients developed cardiogenic shock or failure because of COVID-19 manifestation.

In a study made by [27] 68 fatal cases of coronavirus admitted patients, 58% of them died due to respiratory failure, and 33% died from both heart and respiratory system failure, and 7% died only from heart failure. Further study concluded that, in the latter stages of the disease heart failure played a significant role. A recent report by [28] described the three cases of acute systolic heart failure due to COVID -19 infection. This study suggested that left ventricular dysfunction could have been caused by myocarditis stress-cardiomyopathy, and hypoxemia on underlying cardiovascular disease. However due to low availability of data regarding this, it is unclear if the acute heart failure is due to exacerbation of pre-existing cardiac dysfunction or new-onset cardiomyopathy, in COVID patients.

II. CONCLUSIONS

SARS-CoV 2 virus which is the reason for COVID-19, has resulted in mortality and morbidity worldwide and has become major health threat. CVD is observed as common condition among the patients admitted due to COVID 19 and is associated with severe risk and mortality. myocardiopathy and Myocardial injury are present in considerable number of patients having already existing problems of CVD or heart failure seem to be susceptible to myocardial injury. Despite the concerns and efficiency of RAAS inhibitors on ACE2 and the way drugs effect he severity of COVID 19, it is recommended to continue using RAAS inhibitors, based on the available evidence.

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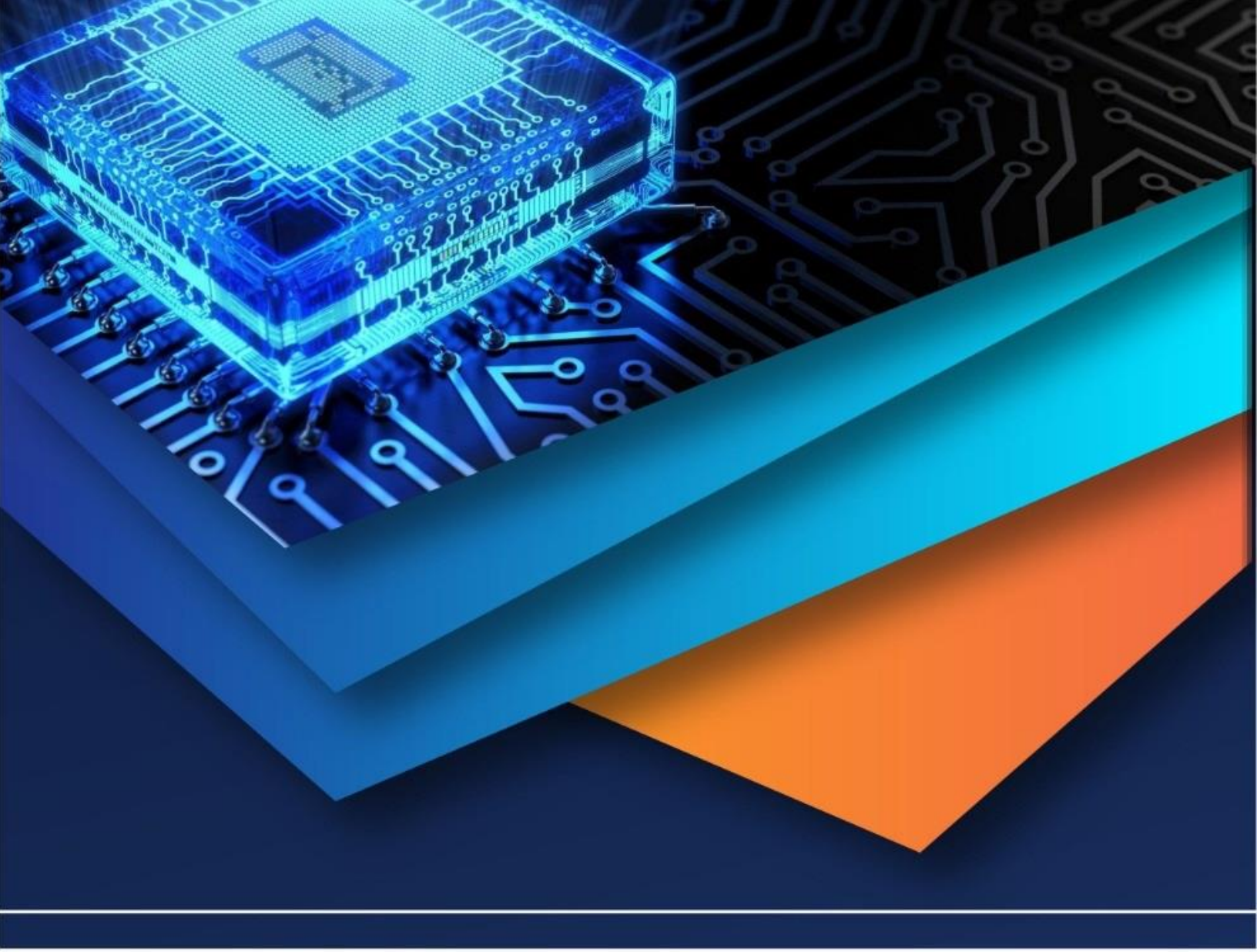

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