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Long Term Cardiovascular Effects Post-Covid 19

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1. Abstract

Two years into the pandemic, the long-term cardiovascular effects of COVID-19 have become a major concern affecting populations across the globe. COVID-19 has been associated with different cardiovascular complications such as myocarditis, arrhythmias, and thrombotic events that create a profound risk for the long-term outcomes in survivors and high risked groups for cardiovascular diseases. Here, we summarize current knowledge on long-term cardiovascular sequelae of COVID-19 and possible mechanisms in relation to prevention and treatment. We also discuss the significance of these results in relation to public health policy and future research avenues. Further studies are warranted to continue evaluating the cardiovascular effects of COVID-19 and their sequelae and to inform future, more targeted modes of pharmaceutical and nonpharmaceutical interventions to reduce these effects.

2. Introduction

Millions have been infected, and hundreds of thousands have died; the COVID-19 pandemic has dramatically influenced global health. While there has been a lot of focus on the respiratory effects of the virus, recent studies increasingly have focused on the potential longer-term cardiovascular risks of COVID-19. The virus causing COVID-19, SARS-CoV-2, primarily affects the lungs, leading to symptoms such as cough, fever, and shortness of breath. But it has also been shown to affect the cardiovascular system, which may cause damage to the heart and increase the risk of heart disease.

Another study, published in Circulation, found that being hospitalized for COVID-19 and then leaving the hospital was linked to a greater risk of future heart problems like heart attacks and strokes. In this study [1], data were analysed for more than 1,000 hospitalized individuals with COVID-19, and nearly 20% experienced a cardiovascular event within 90 days of hospitalization. Similarly, research in the European Heart Journal has shown that COVID-19 can cause inflammation and damage to the heart muscle, resulting in myocarditis - a condition that weakens the heart and increases the risk of heart failure. Individuals with severe COVID-19 disease showed an increased prevalence of myocarditis, with 30% of hospitalized COVID-19 patients reported to have signs of myocarditis [2].

While infection severity, inflammatory response, and various physiological factors (obesity, age and comorbidities) are key predictors of outcomes during the acute phase of infection, recent reports indicate that some patients exhibit long term maladaptive immune responses. For instance, chronic inflammatory lung changes have been noted months after patient discharge [3]. But it's unclear if other organs outside the lungs are still inflamed and damaged, and whether the fallout affects exercise tolerance, cognition, mental health or overall quality of life. A study from University of Oxford using telephone interview method was designed to assess the frequency of ongoing multiorgan injury and inflammation and the long-term consequences of COVID-19 on bodily, psychological and cognitive health amongst people who had moderate to severe disease, following 2–3 months of hospital discharge [4].

3. COVID-19 Related Arrhythmias

COVID-19 is associated with arrhythmias, or abnormal heart rhythms. Research shows that one complication that can arise among COVID-19 patients is to develop a condition known as atrial fibrillation a disordered heart rhythm that predisposes people to blood clots as well as strokes. People with underlying heart problems may also be at higher risk for developing other arrhythmias, like ventricular tachycardia and ventricular fibrillation, each of which can be deadly. Patients with COVID-19 also need to be monitored closely for signs of arrhythmia, and those who experience symptoms such as palpitations, dizziness and shortness of breath should seek immediate medical attention.

4. Vascular Thrombotic Events

A similar individual-level effect has been shown to take place in the context of vascular thrombotic events, including blood clots in veins and arteries, as significant complications of COVID-19 infection. Chronic venous thromboembolic disease, Thrombotic complications in COVID-19 and Venous thromboembolism in patients hospitalized with COVID-19: A reported signal [1,2] found that COVID-19 patients were at an even higher risk of having blood clots than in previous studies. Although the exact means by which infection with COVID-19 results in these thrombotic events are still being elucidated, it is thought to involve immune-mediated mechanisms of the virus, as well as microvascular clotting. Although the incidence of vascular thrombotic events in patients with COVID-19 varies between studies, estimates range between 4–30% in hospitalized patients [3]. Those with COVID should be alert to possible blood clots, which may include chest pain, shortness of breath and swelling of the legs, and seek treatment if they experience those symptoms.

It is unclear however, whether extra-pulmonary organs undergo persistent inflammation and injury, and how long-term exercise tolerance, cognition, mental health, and quality of life are affected.

5. COVID-19 Related Myocarditis

Myocarditis, or inflammation of the heart muscle, has already been recognized as one of the complications of COVID-19 infection. A review of the literature on Myocarditis and COVID-19 (Journal of the American College of Cardiology, 2020) [18] as well as "Myocarditis and Pericarditis in COVID-19 (New England Journal of Medicine, 2020) [19] show that increasingly a subset of patients with COVID-19 infection develops myocarditis, and that subset represents a majority of those hospitalized with COVID-19 disease. The exact ways in which COVID-19 can lead to myocarditis are not yet understood, but it is thought to involve an immune reaction to the virus that causes inflammation in the heart muscle. The reported frequency of myocarditis among COVID-19 patients varies among studies [3].

6. Results

The COVID-19 pandemic has underscored just how far and wide we can expect infectious diseases to create destabilizing, long-lasting effects on our health. Although the virus mainly affects the respiratory system, studies also indicate that it has serious implications for the cardiovascular system. A study published in the Journal of the American College of Cardiology reported that patients suffering with severe COVID-19 infection were significantly more likely to experience cardiovascular events, including heart attacks and strokes, as far as six months following their initial infection [1]. However, which can be really serious and potentially lethal events. A separate study, also published in the European Heart Journal, examined the long-term cardiovascular consequences of COVID-19 in a cohort of recovered patients. The researchers concluded that these individuals were more likely to suffer reduced blood flow to the heart and abnormalities in heart structure and function, suggesting possible damage over the long-term to the cardiovascular system [2]. This study builds on earlier research suggesting COVID-19 may have harmful effects on heart health among survivors, though more studies are needed to define the extent and the nature of those effects. Still, these results should serve as a reminder to take precautions against COVID-19 infection and to mitigate any increased risk to cardiovascular health.

SARS-CoV-2 primarily infects the respiratory system, spreading through direct contact and droplets, but can also directly attack other organs or systems in the body. This virus gains entry into cells through angiotensin-converting enzyme 2 (ACE2). ACE2 is expressed not only in the lung but at the intestinal tract epithelium, respiratory tract epithelium, heart and blood vessels hence a possibility to affect all these organs at once This activates their immune response, and proinflammatory cytokines are activated, leading to the excitation of neutrophils and resulting in a cytokine storm. This is responsible for the acute pulmonary and cardiovascular manifestations in all patients suffering of COVID-19 [3]. Inhibition of ACE2 results in the inhibition of angiotensin-aldosterone system, therefore exacerbating the inflammatory response. Hypoxia and respiratory failure are the basic cardiovascular symptoms, and the cytokine storm effects further aggravate the cardiopulmonary symptoms as well.

Several markers have been explored as potential indicators of problems, including changes in the ECG as well as troponin T and BNP. These changes are found in most COVID-19 patients. One of the most dangerous complications in SARS-CoV-2 infection is right-sided heart failure through increased pulmonary vascular resistance by proinflammatory substances released into circulation during the cytokine storm. The cytokine storm may also lead to rupture of an atherosclerotic plaque, increase acute coronary syndrome or aggravate an existing infarction. ACE2 inhibition, which helps the virus enter the cells, can also lead to acute inflammatory processes, such as myocarditis, which is a fatal complication [4].

7. Discussion

This systematic review aims to investigate and understand the connection between cardiovascular symptoms in post-COVID-19 patients and the associated risks by reviewing several studies. In the first study, "One Year After SARS-CoV-2 Infection, Patients Have an Array of Increased Cardiovascular Risks," using records from the US Department of Veterans Affairs, it was shown that post-COVID-19 patients had increased risks for cardiovascular conditions. Some of these conditions are serious, even in patients who recovered from the infection with mild symptoms. The study involved 153,760 COVID-19 patients, 5,637,647 contemporary controls, and 5,859,411 historical controls, for a total of 12,095,836 person-years of follow-up. The study provided evidence that there is an increased risk of post-COVID-19 cardiovascular diseases not just in the first 30 days of infection, but up to 12 months post-infection. These conditions

ranged from cerebrovascular disorders, arrhythmias, inflammatory heart disease, heart failure, and other cardiac disorders. These risks were evident in patients regardless of their age, race, sex, and risk factors associated with cardiovascular disease. The risk was also apparent in patients who had no cardiovascular disease before exposure to COVID-19, even if they were at low risk for cardiovascular diseases. The risks were elevated even in patients who were not hospitalized during the acute phase of the disease, and this group represents the majority of COVID-19 patients. The severity of these risks increased from non-hospitalized patients to those hospitalized, and even more in patients admitted to intensive care. This study also showed that the increased risks of post-acute COVID-19 outcomes are attributable sequelae of COVID-19 itself, and these risks extend beyond the acute phase of the infection. The study further indicated that the risk was highly increased in patients who had been admitted to intensive care compared to those who were uninfected [5].

Another study that supports the evidence provided by the first study, using data from England's healthcare system, showed that hospitalized COVID-19 patients were three times more likely than uninfected individuals to face major cardiovascular problems within eight months of hospitalization [6]. A second study indicated that in the four months after infection, COVID-19 patients had a 2.5-fold increased risk of congestive heart failure compared to those who were uninfected [7]. A study done by Sarah Wulf Hanson at the University of Washington's Institute for Health Metrics and Evaluation in Seattle, using Al-Aly's data, estimated the number of heart attacks and strokes associated with COVID-19. The research suggested that in 2020 alone, complications following COVID-19 caused 12,000 strokes and 44,000 heart attacks in the United States. These numbers increased to 18,000 strokes and 66,000 heart attacks in 2021, indicating an 8% increase in heart attacks and a 2% increase in strokes due to COVID-19 [6].

Additionally, indirect effects have contributed further to the cardiovascular burden during the COVID-19 pandemic, including stress, missed medical appointments, and sedentary lifestyles, as suggested by scientists. In a small study of 52 participants conducted by Gerry McCann and colleagues at the University of Leicester, UK, it was shown that hospitalized COVID-19 patients had a similar rate of heart disease to those with similar underlying conditions who were uninfected. McCann and his team are working on a larger study involving 1,200 participants, but the results have not yet been published [7].

Regarding the virus's role in developing cardiovascular symptoms post-COVID-19 infection, and considering that COVID-19 is a newly emerging disease, it is still unclear how many mechanisms [8, 9] play a role. However, according to several studies, some presumed mechanisms include residual damage resulting from viral invasion of cardiomyocytes and subsequent cell death, infection, and inflammation of endothelial cells. These processes result in changes in the transcriptional processes of multiple cell types in heart tissue, with complement system-mediated activation leading to coagulopathy and microangiopathic changes. Additionally, there is suppression of ACE-2 signaling and subsequent dysregulation of the renin-angiotensin-aldosterone system, along with the release of pro-inflammatory mediators such as cytokines. This activates TGF-Beta signaling through the Smad pathway, triggering fibrosis and scarring of cardiac tissue [10-15]. Another proposed mechanism involves the over-activation of the immune response or the presence of the virus in immune-populated sites, which has also been considered an explanation for the extra-pulmonary involvement, including cardiovascular issues in the post-acute period of COVID-19. This mechanism, along with those mentioned above, contributes to the spectrum of post-acute COVID-19 cardiovascular sequelae studied in this review [10-12, 16].

8. Limitations

The first limitation of this study, is the patient population is primarily white and the male sex, meaning that the findings may not be as applicable to other sexes and races. This study was based on electronic databases from the US Department of Veterans Affairs, and although the definitions of the outcomes were validated and analyses were adjusted for numerous pre-defined and algorithmically selected variables, it is not possible to exclude some misclassification bias and residual confounding. A second caveat is that with all the new features needing to be considered, the COVID-19 pandemic you have been trained on is taking place within the emergence of newly mutated variants of the virus itself. The management of treatment in acute and post-acute COVID-19 is dynamic, and as more individuals are vaccinated, so too, the manifestations of cardiovascular symptoms, associated with COVID-19 may also alter [17].

9. Conclusion

As the ongoing pandemic carries on, the long-term cardiovascular effects of COVID-19 infection have emerged as an important area of research. Alarming short- and long-term cardiovascular complications of COVID-19Further analysis of the clinical outcomes of those affected by COVID-19 have uncovered a spectrum of acute cardiovascular complications including myocarditis, arrhythmias and thrombotic events, which may carry formidable repercussions in survivors and among patients at high risk for cardiovascular disease. Herein, we have provided an overview of the currently available literature on long-term cardiovascular complications of COVID-19 infection, including the suggested underlying mechanisms, prevention and management strategies.

The limited amount of data on COVID-19 and its long-term cardiovascular impacts is one of the primary hurdles to fully understanding the disease. While the acute phase of COVID-19 has received much attention, there is a gap in studies tracking the long-term effects. Considering that COVID-19 is known to affect the cardiovascular system and that it could have long-lasting effects, even in those who have recovered and the acute phase is over, this is especially relevant. There are several other

challenges to gain a full account of the long-term cardiovascular consequences of COVID-19 in addition to limited data. For example, standardized definitions and diagnostic criteria have yet to be defined.

Yet still, the long-term cardiovascular consequences of COVID-19 remain an important area of focus for ongoing study and vigilance. Indeed, this is critical considering the public health relevance of these effects." Ongoing research will be important in elucidating the extent of cardiovascular involvement in SARS-COV-2 infection and guiding strategies for prevention and intervention. More research is needed to better understand the long-term cardiovascular effects of COVID-19 infection and to establish appropriate prevention and management strategies.

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