Study Of the Relationship Between Covid-19 and cardiovascular disease

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Abstract. Among the deaths caused by Covid, cardiovascular diseases are the most concern. Many people, which look healthy, lost their lives because of some cardiovascular diseases, even after they thought they have already recovered from Covid. This article mainly focusses on the relationship between the Covid and cardiovascular diseases and promote the point that Covid-19 can improve the possibility of cardiovascular disease in several ways. First, Covid may direct make damage to heart and ruin the cardiovascular system. Second, Covid can damage other systems such as lung, and then damage the heart indirectly. Also, Covid lockdown can influence people's activities and change people's lifestyle. This may also contribute to the possibility of cardiovascular disease. This founding suggests that government and some health organization should prepare for the Covid-19 pandemic's potential considerable contribution to an increase in the burden of cardiovascular illnesses.

Keywords: Cardiovascular disease; Covid-19; cytokine storm; BMI; dyslipidemia.

1. Introduction

The SARS-CoV-2 virus, which causes severe acute respiratory syndrome, causes a condition known as coronavirus disease 2019, commonly known as Covid-19. Some cases show that people with underlying medical conditions suffer tougher, especially those with prevalent underlying disorders.

Numerous studies on the clinical characteristics of Covid-19 patients have produced findings that are similar to these. The prevalence of any comorbidity was 32% in an early study covering 41 patients who got Covid and sent to hospital by January 2nd, 2020. The most prevalent underlying disorders were 15% hypertension, 20% diabetes, and other CVDs (15%). Critically sick patients and those who passed away had a higher prevalence of these pre-existing illnesses [1]. Researchers in other nations have noted a rise in the occurrence of comorbidities that is similar to this one. In a study conducted in Italy on 1,591 Covid-19 patients admitted to the intensive care unit (ICU), it was discovered that 49% of patients had pre-existing hypertension, 21% of patients had cardiovascular disease, and 17% of patients had diabetes [2]. Additionally, up to 50% of 393 consecutive Covid-19 patients who were hospitalized in New York, USA, had hypertension, 36% had obesity, 25% had diabetes, and 14% had coronary artery disease [3].

Although viral pneumonia is Covid-19's most common clinical symptom, the virus can also induce heart problems such as myocardial damage, arrhythmias, ACS, and thromboembolism. Some people with Covid-19 first exhibit cardiac symptoms rather than the typical fever or cough symptoms when they first present. High mortality during Covid-19 is independently correlated with myocardial damage. Myocardial injury was discovered in the earliest cases in China, which was shown by an increase in cardiac biomarkers. According to the aforementioned study, cardiac damage was evident in 7.2% of all patients and 22% of those who needed intensive care unit (ICU) care out of 138 hospitalized COVID-19 patients in Wuhan, China. The National Health Commission of China found that among hospitalized patients without a history of CVD, cardiac arrest or elevated troponin levels occurred in roughly 12% of cases [4].

In a recent meta-analysis of 8 studies from China that included 46248 infected patients, the most prevalent co-morbidities were hypertension (17%) and diabetes mellitus (8%), followed by cardiovascular disease (CVD) (5%) [4]. It is yet unknown how these relationships work. Potential

explanations include the fact that individuals with CVD are more likely to be older, have immune systems that are functionally compromised, have increased levels of ACE2, or are predisposed to COVID-19.

2. Research Reviews

According to the United States, during the Covid-19 epidemic, 2.4 out of every 1,000 covid-19 hospitalized patients confirmed or highly suspected myocarditis. However, whether it is ordinary myocarditis or myocarditis caused by the new coronavirus, it is a relatively rare disease, and the incidence is not high, about one hundred to one thousand cases of myocarditis per million people per year. Of these confirmed patients, the incidence of myocarditis severe is extremely low. Although the overall incidence is low, once it occurs, sometimes the consequences are more serious, so it is still necessary to pay enough attention.

Patients with cardiovascular problems are especially susceptible to severe infections during the start of the pandemic [5]. There have been reports of a substantial percentage of Covid-19 individuals having cardiovascular disease already. Myocarditis, arrhythmias, and myocardial infarction are only a few of the cardiovascular symptoms of Covid-19 that are described in early case reports [6]. It has also been proposed that Covid-19 might aggravate heart failure by causing or accelerating myocardial damage, myocarditis, and cytokine storm-related hyperinflammatory syndrome [7]. The influenza virus is a well-known cause of cardiovascular problems and, like SARS-CoV-2, has a major impact on the inflammatory and coagulation pathways. Host genetics, such as host fragility, are likely to account for a considerable percentage of susceptibility in addition to the underlying hyperinflammatory condition, which both viruses' cardiovascular repercussions are most usually associated with. SARS-CoV-2 is more fatal than early respiratory illnesses, and it triggers a higher inflammatory response that could result in more cardiovascular problems, according to recent studies [8]. Numerous epidemiological studies have hypothesized a connection between cardiovascular mortality and influenza. Peak influenza seasons are associated with an increase in the mortality rate from acute myocardial infarction (MI) and ischemic cardiovascular disease, according to time-series study [5]. There is presently no time series analysis for Covid-19. However, it has been discovered that Covid-19 patients have greater rates of cardiovascular death, comparable to those observed with influenza. There are currently no studies examining the long-term cardiovascular effects of SARS-CoV-2 infection months after the acute illness has subsided. When analyzing the myocardial injury in Covid-19 patients, one study discovered that 78% of patients still exhibited cardiac involvement, which is uncharacteristic of people who are recovering from influenza virus and is more typically associated with the acute viremic phase [9].

3. Cardiovascular Risk Factors

Cardiovascular risk factors

Preliminary study indicates that Covid-19 damages the myocardium in two different ways. One study found that values of the median hs-cTnI were 2.5 pg/mL in survivor group and 8.8 pg/mL in non-survivor group 4 days following the start of symptoms. During follow-up, the survivors' median hs-cTnI (2.5-4.4) did not change appreciably. However, among non-survivors, it increased rapidly [9]. Notably, it took 18.5 days in general from the beginning of symptoms to death. Because hs-cTnI rises together with other inflammatory markers, it's probable that the rise in this market is due to a cytokine storm or subsequent hemophagocytic lymph histiocytosis rather of a single myocardial injury [10]. Another report from China documented a man who is 63 with severe respiratory symptoms, fulminant myocarditis, an enlarged left ventricle and reduced left ventricular function. This man had no prior history of cardiac disease [11]. Investigations are still being done to determine how the heart is actually involved in Covid-19. Direct cardiac engagement mediated by ACE2 is one such pathway. An ACE2-dependent heart infection was also brought on by SARS-Covid lung

infection, according to a mousem [12]. Given that multiple studies have shown that SARS-CoV-2 and SARS-CoV share many biological characteristics, it is possible to comprehend the diseases that Covid-19 involves by using our understanding of the pathophysiological mechanisms driving SARS. A possible target for the prevention and treatment of Covid-19, the interaction between the S protein and ACE2 is believed to have a significant role in the onset of the disease, notably in the cardiovascular symptoms of the condition. Two further hypotheses for how Covid-19 affects the heart include a cytokine storm, which is resulting from an uneven response from different T helper cell subtypes, and increased intracellular calcium brought on by hypoxia, which results in cardiac myocyte mortality [13]. Numerous cytokines are released by the body in response to coronavirus infection, helping cells communicate and fight off invaders. Some persons experience an intensification of this typical protective event, perhaps because of a genetic difference, making them more susceptible to a cytokine storm. The immune system's response to inflammation during a cytokine storm overwhelms the body, causing damage to healthy tissue as well as vital organs including the kidneys, liver, and heart.

Another common explanation that how Covid-19 damage the heart is about oxygen. As the virus inflames the body, fluid fills the lungs' air sacs, less oxygen may be able to reach the circulation. The heart must increase blood pumping effort throughout the body, which can be troublesome for those who already have cardiac problems. A shortage of oxygen can induce cell death and tissue damage in other organs, including the heart, and overworked hearts may stop beating altogether. But this is not unique to Covid-19, other viruses that can damage the lung could also cause this problem.

Acute coronary syndrome, as known as ACS, can be triggered by Covid-19. Early research from China found that a tiny percentage of Covid-19 patients admitted to hospitals complaining of chest discomfort, but the symptoms of the pain were not reported. Five of the six myocardial infarct patients in a study from New York State that included 18 individuals with Covid-19 and an elevated ST-segment—a sign of possible acute myocardial infarction—needed percutaneous coronary intervention. These findings imply that Covid can still result in ACS even in the absence of significant systemic inflammation [13]. However, it is still unclear how frequently ACS occurs in Covid-19 individuals. Due to overcrowded medical facilities in various locations during the Covid-19 pandemic, early studies may have undercounted the frequency of sudden cardiac death among Covid-19 patients. The underlying mechanism by which Covid-19 induces ACS may involve the formation of microthrombi brought on by systemic inflammation or cytokine storms, coronary artery spasm, or plaque rupture.

Dyslipidemia would be another point that should be paid attention to as it can also cause cardiovascular disease. According to a different study, individuals who make it through the first month of Covid-19 infection had a higher likelihood of developing incident dyslipidemia and using incident lipid-lowering drugs [14]. The potential mechanisms behind the rise in dyslipidemia are unknown. According to experimental data, the inflammatory and immune systems response to the earliest infection may change how hepatic lipoproteins are metabolized, possibly causing briefly low levels to occur during the acute phase and an imaginary over compensatory rebound to occur during the post-acute phase. SARS-CoV-2 infection is linked to an increased incidence of incident dyslipidemia in individuals who have no history of the condition. These results lend credence to the general conclusion that cardiometabolic disease is both a risk factor for and a consequence of SARS-CoV-2, indicating a potential reciprocal link. The study also observed that people with Covid-19 had a higher risk of an increase in BMI compared to both the contemporary and historical controls. But other behavioral modifications, like less exercise due to the lockdown or dietary modifications due to stress, also contribute to the rise in BMI. Future studies should concentrate on the extent to which incident dyslipidemia may be mediated by the rise in BMI following SARS-CoV-2 infection, or whether dyslipidemia itself may be mediated by the illness itself.

Apart from those direct connections, lockdown tactics may have caused people to become less active, which may have made it harder to control cardiovascular risk factors. Besides, some people

are “trapped” in their houses and become stressed, which may lead to their mental health problems, causing irregular sleep and diet. That might also contribute to cardiovascular diseases.

The post-acute cardiovascular symptoms of Covid-19 are a significant issue in addition to the cardiovascular problems of acute Covid-19. Using national healthcare information from the US Department of Veterans Affairs, a study created a cohort of 153,760 patients with Covid-19. It also created two sets of control cohorts, which included 5,637,647 (current controls) and 5,859,411 (historical controls) individuals [11]. The study shows that after the initial 30 days following infection, people with Covid-19 are more prone to experience different forms of cardiovascular problems. The study makes a new point that the integration of the genome of SARS-CoV-2 into the DNA of infected human cells has also been proposed as a potential method for maintaining the immune-inflammatory-procoagulant cascade's activation. After that, this integration can appear as chimeric transcripts that combine viral and cellular sequences [14].

4. Conclusion

The risk and burden of incident cardiovascular disease over a 12-month period are significant and include numerous types of cardiovascular disease. The risks and expenses of cardiovascular disease were evident even in those who did not require hospitalization because to acute COVID-19. The virus may not the attack heart directly, but it can cause a cytokine storm, leading to myocardial damage. The virus also damages the lung, causing less oxygen to enter the circulation and make damage to cardiovascular system. The virus and the lockdown caused by the virus may cause an increase in BMI and the possibility of dyslipidemia, which would also increase the possibility of cardiovascular disease.

In other words, preventing SARS-CoV-2 infection in the first place is the best strategy to prevent Long Covid with all of its numerous consequences, including the risk of catastrophic cardiovascular sequelae. These findings highlight the need for ongoing improvement of primary prevention strategies for SARS-CoV-2 infections. Second, Governments and healthcare systems worldwide should be ready to handle the Covid-19 pandemic's potential considerable contribution to an increase in the burden of cardiovascular illnesses. Because these illnesses are chronic, their effects on patients and healthcare systems are likely to be long-lasting, and they may also have significant effects on economic productivity and life expectancy.

The limitations are obvious. Most of the research used data from the first two years of the pandemic and did not include some new virus variants. Also, in the early days of the epidemic, due to factors such as medical shortages, there was little systematic recording and research on the connection between Covid-19 and cardiovascular disease. Future studies may focus on the comparison between Covid-19 and other diseases, to determine whether the percentage of cardiovascular disease is much higher than other viruses as other viruses such as SARS and MERS also have reports of cardiovascular disease.

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