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Covid 19 and Its Effects on the Cardiovascular System a Review

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ABSTRACT

The development of a novel coronavirus known as severe acute respiratory syndrome-Coronavirus-2 (SARS-CoV-2) has posed an unprecedented challenge to the global healthcare community. High infectivity, the ability to transmit even when asymptomatic, and low virulence have resulted in rapid spread. The goal of this review is to provide an overview of various CV symptoms in COVID-19 patients. The effect of pre-existing CVD and new-onset cardiac problems on clinical outcomes is also examined in these individuals. The most common clinical manifestation of COVID-19 is respiratory involvement, which can range from a mild flu-like sickness to potentially fatal acute respiratory distress syndrome or fulminant pneumonia-existing cardiovascular disease (CVD) and CV risk factors, like any other respiratory tract infection, increase vulnerability to COVID-19. Furthermore, COVID-19 can exacerbate pre-existing CVD and potentially cause new heart problems. Because our knowledge of this subject is still developing, the information in the following material is based primarily on limited early experience with COVID-19 and lessons learned from past coronavirus infections, such as SARS and Middle-East Respiratory Syndrome (MERS).

Abbreviations: CVD: Cardiovascular Disease; MERS: Middle-East Respiratory Syndrome; ACE2: Angiotensin-Converting Enzyme 2; VT: Ventricular Tachycardia; STEMI: ST-Segment Elevation Myocardial Infarction; SARS: Severe Acute Respiratory Syndrome

Introduction

A coronavirus is a type of virus that causes infections in the nose, sinuses, and upper throat. The majority of coronaviruses aren't harmful. SARS-CoV-2 was discovered as a new kind of coronavirus by the World Health Organization in early 2020, following a December 2019 epidemic in China. The disease soon spread around the globe. COVID-19 is a sickness produced by the SARS-CoV-2 virus that can cause a respiratory tract infection, according to specialists. It can affect your sinuses, nose, and throat, as well as your lower respiratory tract (windpipe and lungs). It spreads in the same way that other coronaviruses do, primarily through direct contact between people. Infections can be minor or fatal. SARS-CoV-2 is one of seven types of coronaviruses that can cause serious illnesses such as Middle East respiratory syndrome. Pneumonia, respiratory failure, cardiac issues, liver problems, septic shock, and death can all be caused by the virus. A condition known as cytokine release syndrome, or a cytokine storm may be the source of many COVID-19 problems. This happens when an infection causes your

immune system to release inflammatory proteins called cytokines into your bloodstream. They have the ability to kill tissue and harm your organs. Lung transplants have been required in some circumstances [1].

Pathological Mechanisms

SARS-CoV-2 infects human cells via attaching to angiotensin-converting enzyme 2 (ACE2), a membrane-bound aminopeptidase that is widely expressed in the heart and lungs. ACE2 is involved in the neurohumoral control of the cardiovascular system in both normal and diseased states. SARS-CoV-2 binding to ACE2 can cause ACE2 signaling pathways to be disrupted, resulting in acute cardiac and lung damage [2,3]. More severe types of COVID-19 are marked by an abrupt systemic inflammatory response and cytokine storm, which can result in organ injury and multiorgan failure. Patients with severe/critical COVID-19 have been found to have elevated amounts of proinflammatory cytokines in their blood [4,5]. Plaque rupture and coronary thrombosis. Acute myocardial infarction can be precipitated by systemic inflammation as well as increased shear stress due to increased coronary blood flow. The prothrombotic environment caused by systemic inflammation increases the risk even further. Adverse effects of various therapy-Antiviral medicines, corticosteroids, and other therapies used to treat COVID-19 can have negative effects on the cardiovascular system. Electrolyte imbalances can arise in any serious systemic illness and can lead to arrhythmias, particularly in individuals with underlying heart disease. Because SARS-CoV-2 interacts with the renin-angiotensin-aldosterone pathway, hypokalemia is a major problem in COVID-19. Hypokalemia makes you more susceptible to tachyarrhythmias [6].

Effects of Covid on the Cardiovascular System

Although myocardial infection with COVID-19 is thought to be responsible for 7% of COVID-19-related deaths, its actual prevalence is unknown. The symptoms can range from modest chest pain, weakness, and dyspnea to more severe left and right ventricular failure, arrhythmias, cardiogenic shock, and sudden cardiac death due to fulminant myocarditis. While there is no current evidence to support direct COVID-19 viral myocarditis, MERS-CoV and SARS-CoV viral RNAs, which are closely related to SARS-CoV-2, have been found in infected organism cardiac tissue. COVID-19 myocarditis is thought to be caused by a combination of direct cell injury and cytotoxicity mediated by T lymphocytes, which can be exacerbated by cytokine storms. COVID-19-induced myocarditis can be mistaken for coronary artery disease. Due to damage, the ST segment and cardiac enzymes are elevated [7]. The most commonly diagnosed arrhythmia in COVID-19 disease is symptomatic/asymptomatic tachycardia. There's also been a report of bradycardia. Myocarditis, myocardial ischemia, and generally unwell people with shock and hypoxia can all cause arrhythmias. Because of the COVID-19 disorder, various forms of arrhythmias have been identified. In COVID-19 patients, a few instruments may cause or aggravate arrhythmias.

Unsettling electrolyte influence (most commonly hypokalemia), antagonistic effects of treatments (e.g., chloroquine, Azithromycin, and Hydroxychloroquine) that prolong the QT interval [measured from the beginning of the QRS complex to the end of the T wave with the expected progression of polymorphic ventricular tachycardia (VT) and fever, which may reveal instances of heart channelopathies, such as brugada disorder and long QT condition [8,9]. There is a possibility that the data on ST-segment elevation myocardial infarction (STEMI) caused by intracoronary plaque rupture or obstruction is inaccurate. In high-risk patients, plaque collapse and coronary thrombosis can lead to acute coronary events due to inflammation and increased shear stress. Tam et al. reported a significant decrease in the frequency of STEMI patients seeking clinical attention at their facility. They attributed this to patients' reluctance to visit an emergency clinic during the COVID-19 incident, as well as delays in diagnosing patients with STEMI after medical clinic visits due to preventive measures such as a complete travel and contact history, symptomatology, and a chest X-ray. As a result, the transfer of patients to the catheterization center is postponed. Additional precautionary measures used in the catheterization laboratory, such as the time required to wear protective clothes, can lengthen the procedure even further [10]. There are insufficient data on the incidence of left ventricular systolic dysfunction, abrupt left ventricular collapse, and cardiogenic shock. In one study, cardiac illness was noted by 52 percent of dying patients and 12 percent of discharged patients. 29 Many critically ill individuals are at risk of developing infections. Reversible sepsis-related cardiomyopathy with left ventricular dilation and decreased systolic function that heals in 7-10 days.

Infections with COVID-19 can lead to decompensation of underlying heart failure and mixed shock syndrome. In such cases, intrusive hemodynamic monitoring, if practicable, can aid in the control of the cardiac component of shock [11]. Long-term cardiovascular consequences in COVID-19-infected patients are too early to predict. However, the anticipated outcomes will be similar to those seen in SARS-CoV virus-induced Severe Acute Respiratory Syndrome (SARS). According to the results of a 12-year study of SARS patients, 40% had cardiovascular issues, 60% had impaired glucose metabolism, and 68 percent had abnormal lipid metabolism [12]. COVID-19 infection is associated with a prothrombotic state that causes venous and arterial thrombosis as well as an increased D-dimer. The reported incidence of cerebrovascular disease in

COVID-19 patients ranged from 2.3 percent to 22 percent. 34 Increased generation of antiphospholipid antibodies has been suggested as a possible cause of ischemic stroke. The link between COVID-19 and stroke was demonstrated by a 2.5-fold increase in illness incidence [13]. A hypercoagulable condition with significant COVID-19 illness was discovered in the laboratory and autopsy reports. Platelets and coagulation factors are implicated in the control of the host immune response, displaying proinflammatory functions apart from their immunological response. Hemostatic effects and a high D-dimer level are linked to poor results [14].

Conclusion

Although respiratory illness is the most common clinical manifestation of COVID-19, the disease's high prevalence means that many patients with COVID-19 will have pre-existing CVD or may acquire new-onset cardiac dysfunction during their illness. Given this, our present understanding of the relationship between CVD and COVID-19 is woefully inadequate. Future COVID-19 research should therefore focus on describing the incidence, causes, clinical presentation, and outcomes of various CV symptoms in these patients. The diagnostic and therapeutic issues provided by these two disorders coexisting must also be thoroughly investigated.

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