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Cardiovascular Manifestations of COVID-19: An Update

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Abstract

Higher mortality rates are linked to COVID-19 cardiovascular problems. Cardiac damage may result from a variety of factors, including combinations of cytokine storm, myocarditis, extremely high levels of physical and mental stress, ischemic injury, hyper-coagulopathy, right heart strain, and corpulmonale. Prognostic information is provided by echocardiography, c-TN, D-dimer, and NT-proBNP values. There is no particular therapy for COVID-19-related cardiac damage other from percutaneous coronary intervention for STEMI, and care is mostly supportive. It remains to be known if antiviral treatments given early in the course of the illness would stop the severe illness and cardiovascular consequences linked to COVID-19.SAIDs: Non-steroidal Anti-inflammatory Drugs; NO: Nitric Oxide; cGMP: cyclic Guanosine Monophosphate; LDL: Low-density Lipoprotein

Introduction

For billions of people worldwide, COVID-19 has been a nightmare, causing unheard-of losses in their physical, financial, and mental well-being. Despite the existence of very efficient coronavirus vaccinations, the incidence and mortality rate continue to rise [1]. This infection's multisystemic involvement in the human body is a crucial side effect. The respiratory system is not the only body system that is negatively impacted by COVID-19; other body systems and organs are also negatively impacted [2]. The SARS-CoV-2 virus has a number of potentially fatal consequences on the cardiovascular system [3]. It aggravates any underlying cardiovascular conditions and sets off additional acute events that are exacerbated by inflammation. In order to explain the risk factors, alleged causes, diagnosis, and management of cardiovascular manifestations of COVID-19, this study reviewed the literature that had been published till July 2022.

Incidence

According to two early investigations on COVID-19, 20 to 28 percent of COVID-19 patients showed cardiac damage linked to cardiac dysfunction and arrhythmias [3-4]. Cardiac damage was found to occur in 19.7 percent of 416 individuals hospitalized with confirmed COVID-19, and it was linked to an unexpectedly high risk of death during hospitalization. The severity of COVID-19 symptoms increased when cardiac damage was present, and patients with cardiac injury had a greater death rate than those without it (51.2 percent vs 4.5 percent) [3].

Risk factors

A cohort of 187 patients from a different independent research found that those who had cardiac damage were more likely to be male, older, and to have more co-morbid conditions, such as diabetes, hypertension, coronary artery disease, chronic renal disease, chronic lung disease, etc. Potentially related to severe COVID-19 infections were cardiac arrhythmias and the requirement for mechanical ventilation. Patients without underlying cardiovascular illness and with normal cardiac troponin (c-TN) levels saw a hospital mortality rate of 7.62 percent, but this rate increased to 69.44 percent for patients with underlying cardiovascular disease and elevated c-TN [4]. In a retrospective analysis studying 393 participants, patients with COVID-19 who experienced severe symptoms exhibited a greater incidence of obesity and male sex than those who did not [5].

Cardiovascular manifestations in COVID-19 patients

The prognosis for COVID-19 cardiovascular problems is often poor. As a result, COVID-19 treatment and prevention should be given top attention. Understanding the potential pathogenetic pathways causing myocardial damage might be beneficial in this regard. According to COVID-19 patients, cytokine storm,

myocarditis, extreme physical and emotional stress, ischemic injury brought on by cardiac microangiopathy or macrovascular coronary artery disease, hyper coagulopathy, right heart strain, and cor-pulmonale linked to adult respiratory distress syndrome are some of the potential causes of cardiovascular complications.

Cytokine storm

Patients with COVID-19 have also been reported to have uncontrolled inflammation, also known as a "cytokine storm," which is characterized by high levels of inflammatory markers like C-reactive protein (CRP), ferritin, and D-dimer as well as elevated levels of inflammatory cytokines and chemokines [6]. The precise pathogenetic significance of cytokine storm is yet unknown.

Myocarditis

Elevated c-TN levels have been seen in certain individuals with myocardial inflammation (myocarditis), and autopsy results demonstrate a mononuclear infiltration in the myocardium along with associated cardiomyocyte destruction. Although myocarditis has been reported in individuals with COVID-19, it is unknown whether the condition is brought on by an unchecked inflammatory response or a direct viral invasion [7].

Ischemic harm

ST-segment elevation myocardial infarction (STEMI) may be the initial clinical sign of COVID-19 in some people. Patients with elevated c-TN levels might not exhibit epicardial coronary artery blockage at angiography, nevertheless.

Hypercoagulopathy

A hypercoagulable condition is related to COVID-19. The pathophysiology is not well understood. The following symptoms may be seen, increased fibrinogen and D-dimer; prothrombin time and activated partial thromboplastin time prolongation; moderate thrombocytosis or thrombocytopenia. Even with thromboprophylaxis, individuals with COVID-19 commonly experience major adverse cardiovascular events and symptomatic thromboembolism, especially those in the intensive care unit (ICU) [8].

Thrombocytopenia

Studies have shown that thrombocytopenia is linked to a higher risk of COVID-19 death in individuals with severe illness [9]. It is not yet known how thrombocytopenia should be taken into account when deciding whether to recommend anticoagulant medication.

Psychological and physical strain

There have also been cases of typical stress cardiomyopathy reported, which indicates that both physical and mental stress may play a role in certain cases of cardiovascular consequences of COVID-19 [10].

Right heart strain

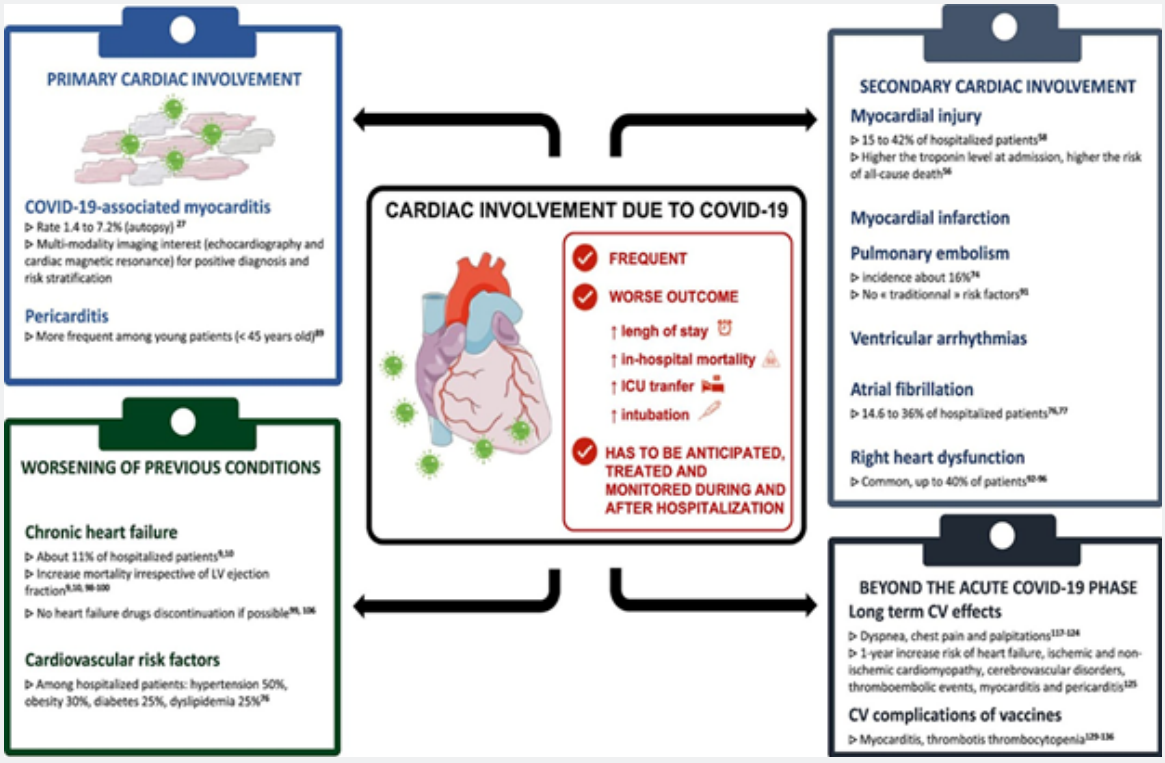

Figure 1: Cardiovascular manifestations of COVID-19 [13].

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Right heart failure also appears to be a primary kind of secondary cardiac involvement in the COVID-19 scenario as a direct result of both pulmonary vascular disease (e.g., pulmonary embolism, endothelial dysfunction) and parenchymal lung involvement. This represents a dysfunctional relationship between the right heart and the pulmonary vascular tree, which can be exacerbated if RV dysfunction, which is a complication of left heart disease and/or pulmonary hypertension, existed prior to the infection. Numerous echocardiographic investigations have shown that COVID-19 patients frequently have right cardiac dysfunction. According to Szekely et al. [11], RV dilatation and dysfunction were observed in 39% of people with COVID-19, which is consistent with findings from previous research. Additionally, RV dysfunction and dilatation have been recognized as independent prognostic markers in COVID-19 patients. Medical professionals must be aware that RV dysfunction and/or dilatation can also be beneficial to identify patients at high risk (i.e., of mortality and/or transfer to an intensive care unit), in addition to the often utilized clinical and/or biological prognostic factors [12]. Figure 1 demonstrates several cardiovascular manifestations in patients following COVID-19.

Effects of SARS-CoV-2 infection over time

It is well established that viral infections have the potential to produce long-term cardiovascular consequences. A poorer cardiovascular prognosis following infection and altered lipid metabolism were suggested by prior research on the severe acute respiratory syndrome coronavirus 1 [12]. Similar to this, cardiovascular disease mortality was linked to visits to the emergency room for influenza-like illness [12]. A constellation of enduring symptoms, including cardiovascular symptoms, may linger beyond the acute phase of COVID-19. Chest pain is frequently reported long term after the acute phase of COVID-19; its prevalence is roughly 20% at 60 days after a moderate-to-severe form of the disease, and 5% at 6 months [12]. In contrast, dyspnea is the most common persistent symptom of a probable multifactorial etiology. This common symptom parallels the several CMR anomalies seen during the acute COVID-19 phase, as was previously noted. Future research will be needed since the causes and processes of recurrent chest discomfort are still not fully understood. Palpitations also occurred often, with prevalence rates of 14% at 60 days and 9% at 6 months [12]. Parallel to this, following a mild type of COVID-19, thorough screening revealed electrocardiographic alterations and arrhythmias in around one-third of patients [12]. Last but not least, COVID-19 infection may have long-term effects, including: (1) potential long-term sequelae of acute cardiovascular complications, like pulmonary embolism, acute MI, or myocarditis; (2) disruption of routine follow-up of chronic cardiac conditions caused by the pandemic; and (3) persistence of the aforementioned symptoms, the mechanisms, prognosis, and psychological impact of which are not well known.

The long-term cardiovascular effects of COVD-19 were recently published by Xie et al. [13]. There was a significant 12-month increase in ischemic and non-ischemic heart disease, heart failure, thromboembolic events, cerebrovascular disorders, pericarditis, and myocarditis among the 153,760 COVID-19 patients studied (non-hospitalized, hospitalized, and admitted to intensive care unit), and this risk was consistent among non-hospitalized patients and patients who were not hospitalized. These findings underline the significance of close cardiovascular follow-up, at least for the first year after a COVID-19, as well as the need for physicians to communicate preventative messages to the general public more broadly.

Conclusion

For health systems, health professionals, and patients, it has been a very difficult and trying time since the start of the SARS-CoV-2 outbreak and the subsequent unmatched health catastrophe. Numerous published clinical studies and data indicate that COVID-19 disease can have an impact on the cardiovascular system. In this pandemic condition, decreased physical activity may potentially have a negative impact on the management of cardiovascular risk factors. It is obvious that people with COVID-19 who already have cardiovascular disorders typically have increased mortality and worse prognoses. COVID-19 is directly associated to cardiovascular disorders such myocarditis, arrhythmias, pericardial effusion, pulmonary hypertension, and heart failure. These diseases may have direct cell damage from ACE2 through stress, hypoxia, inflammatory factors, and drug usage as plausible pathways. Patients with pre-existing cardiovascular diseases should get special consideration, and it is important to ensure that the cardiovascular system is adequately protected during the treatment of COVID-19 patients. To safeguard patients with pre-existing cardiovascular conditions and prevent COVID-19-induced cardiovascular complications before and after immunization, a thorough management guideline should be created for clinicians, healthcare professionals working in hospital settings, and non-hospital caregivers. By doing so, the mortality rates of COVID-19 patients can be decreased.

Conflict of Interest

The author has no conflict of interest to declare.

Ethics Statement

Not applicable.

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