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Cardiac manifestations and COVID-19- A review

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Abstract: The World Health Organization (WHO) classified the most recent coronavirus disease outbreak of 2019 (COVID-19) a pandemic on March 11, 2020. The cause of COVID-19, Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), largely affects the respiratory system, with viral pneumonia as a complication most common manifestation. Moreover, SARS-CoV-2 has a number of cardiovascular symptoms that raise COVID-19's morbidity and fatality rates. Individuals are more likely to develop COVID-19 and have a worse prognosis if they have underlying cardiovascular illnesses and traditional cardiovascular risk factors. Endothelial dysfunction, widespread microangiopathy with thrombosis, and elevated angiotensin II levels are potential pathways of cardiovascular damage. Acute coronary syndrome, myocarditis, heart failure, cardiac arrhythmias, and sudden death can all be caused by myocardial hyperinflammation. The early stages of COVID-19 show a high level of cardiac troponins and natriuretic peptides, which indicates an acute myocardial damage. Given the intricate relationship between COVID-19 and cardiovascular symptoms, comprehensive knowledge for the proper management of these patients. Treatment is symptomatic until a particular antiviral medication for COVID-19 becomes available. Information about COVID-19's cardiovascular risk factors and symptoms is provided in this review.

Keywords: Covid-19; Thrombotic events; Arrhythmias; Heart failure.

1. Introduction

On March 11, 2020, the World Health Organization proclaimed coronavirus disease 2019 (COVID-19) to be a pandemic the onset of extremely intense. The COVID-19-causing respiratory syndrome coronavirus (SARS-CoV-2) infection was first discovered on December 12, 2019 in Wuhan, China. Despite the fact that SARS-CoV-2 can infect persons of any age, older individuals with underlying cardiovascular conditions and those with typical cardiovascular risk factors with significant morbidity and mortality rates include male sex, diabetes, obesity, and hypertension [1,2]. Moreover, individuals may experience a variety of cardiovascular indications before to or following the clearance of upper respiratory infection symptoms. Acute coronary syndrome (ACS), myocarditis, cardiac arrhythmias, and out-of-hospital cardiac arrest have all been identified as fatal outcomes in individuals with COVID-1911 in numerous case studies [3–5]. The objective of this review was to present up-to-date knowledge on COVID-19 symptoms.

2. COVID-19 and Cardiovascular Risk factors

Retrospective investigations and numerous case series have validated the link between COVID-19 mortality and cardiovascular risk factors. Firstly large studies from China, later joined by those from Italy and the USA, confirmed the link between COVID-19 mortality and cardiovascular risk factors. The most prevalent cardiovascular disease in a recent series of 5700 COVID-19 patients in New York City, USA, was hypertension, which affected 56.6% of patients. Coronary artery disease (CAD), which affected 11.1% of patients, congestive heart failure, which affected 6.9% of patients, obesity, which affected 41.7% of patients, and diabetes, which affected 33.8% of patients [6].

A larger initial investigation from China examined the demographics of 44,672 confirmed COVID-19 patients and found that 4.2 percent were female. Cardiovascular disease affected percent of patients, hypertension affected 12.8% and diabetes affected 5.3%. This study has the drawbacks of not being age-adjusted and of missing data on comorbid illnesses in 53% of cases. Cardiovascular disease was present

in 15% and 14.5% of individuals in two further small single-center trials on COVID-19, respectively. In the research of 144 COVID-19 patients from India, diabetes mellitus was found in 11.1% of cases, hypertension in 2.1%, and coronary artery disease (CAD) in 0.7% of cases [7].

From asymptomatic myocardial damage to severe cardiovascular manifestations, COVID-19 has a wide range of symptoms to an external cardiac arrest. The cumulative incidence of out-of-hospital cardiac arrest has dramatically increased in Italy during the COVID-19 pandemic cardiovascular signs and symptoms of both direct viral damage and an immune response to the virus, as seen in myocarditis are potential causes of SARS-CoV-1. In a few cases of SARS-CoV-2 and SARS-CoV-1, direct virus invasion of the heart has been observed during postmortem research. The diagnostic sample of COVID-19 patients showed microvascular thrombosis and inflammation. Hyperinflammation and endothelial dysfunction are brought on by the release of cytokines such interleukin-1 (IL-1), tumour necrosis factor-alpha (TNF-), and IL-6 by localized macrophage activation. Patients with COVID-19 had lung tissue samples that demonstrated severe thrombotic microvascular damage syndrome caused by complement, together with persistent activation of the alternate complement pathway. In individuals with severe COVID-19, more than 70% meet the requirements for disseminated intravascular coagulation. It is believed that endothelial dysfunction and the development of thrombi in the coronary microcirculation are what cause myocardial damage in patients with severe COVID-19 [8–10].

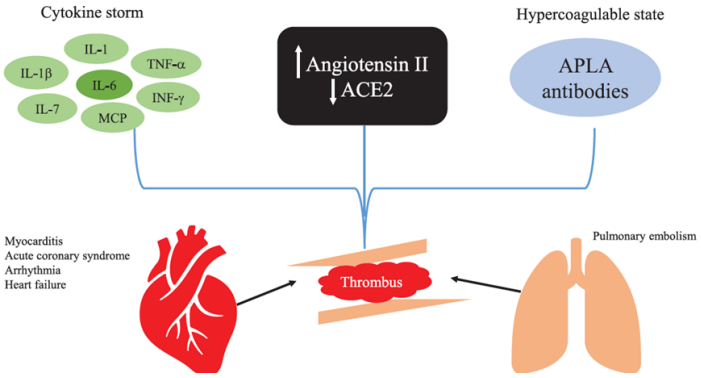

Figure 1. Mechanism of Cardiovascular Manifestation in COVID-19

100 recovered COVID-19 patients from a recent study who underwent cardiac magnetic resonance imaging (MRI) indicated continuing inflammation in 60 (60%) individuals and cardiac involvement in 78 (78%) patients. Of the 39 COVID-19 positive individuals, 24 (61.5%) had SARS-CoV-2 in their myocardium, according to another autopsy study. In 16 of 39 (41%) instances, a viral load of more than 1000 copies/g RNA was found. Although increased cytokine levels were linked to higher viral RNA levels, an invasion of inflammatory cells was not linked to this [11].

3. Cardiovascular Manifestation in COVID-19

3.1. Myocardial Infarction:

A ST-segment elevation myocardial infarction (STEMI) mimic is COVID-19. Plaque may be the cause of ACS rupture, coronary artery spasm, supply-demand imbalance, or cytokine-induced endothelial dysfunction. Up to 27.8% of COVID-19 patients in the initial study from Wuhan, China, showed increased troponin levels, which suggested cardiac injury during the index hospitalization for COVID. Nine (50%) of the 18 patients with COVID-19 who had STEMI in a recent case study by Bangalore et al underwent coronary angiography, and five of these nine patients underwent percutaneous coronary angiography [12].

The findings demonstrated a significant frequency of non-obstructive CAD, variability in presentation, and a poor prognosis even after 13 individuals (72%) died in hospitals, necessitating revascularization. Worldwide reports of delayed ACS presentation have been made, and it is believed that some people may have passed away from ACS without seeking medical care. Most of these fatalities can be attributable to severe arrhythmias and mechanical issues that result in cardiogenic shock [13].

3.2. Myocarditis:

Myocardial damage is indicated by a rise in troponin and NT-proBNP (N-terminal pro-brain natriuretic peptide) levels resulting from the SARS-CoV-27. With COVID-19, a few cases of fulminant myocarditis have also been documented. The cardiac imaging of COVID-19 patients has little available data. According to anecdotal reports, echocardiography showed significant left ventricular systolic dysfunction. A significant amount of biventricular myocardial interstitial oedema and diffuse late gadolinium enhancement involving the entire biventricular wall were seen on cardiac MRI, both of which were suggestive of a significant amount of myocardial injury [14].

3.3. Cardiac Failure:

Individuals with COVID-19 may experience heart failure-related symptoms include tiredness, palpitations, and dyspnea. According to a preliminary Chinese study adult COVID-19 inpatients exhibited dyspnea when they first arrived, or roughly 18.7% of them. According to numerous research, the prevalence of acute heart failure ranges from 4.1% to 23%, complicating the clinical course of COVID-19. A tachyarrhythmia, myocarditis, or myocardial ischaemia may be the cause of the acute heart failure. The signs of acute respiratory distress syndrome and acute heart failure are difficult to distinguish from one another. Rising cardiac troponin levels and NT-proBNP levels in COVID-19 should raise concerns about myocardial damage. Due to their old age and the presence of other comorbidities, patients with chronic heart failure are more likely to develop COVID-19. Heart failure precipitations could become more frequent in the epidemic of COVID-19. Individuals with persistent heart failure should continue to take their medications as prescribed [15].

3.4. Arrhythmia:

In a preliminary analysis of COVID-19 from China, the incidence of malignant arrhythmia was 5.9% international cross-sectional survey. Some COVID-19 patients have experienced arrhythmias ranging from mild to possibly life-threatening. The most frequent tachyarrhythmia was atrial fibrillation, followed by atrial flutter, and the most frequent bradyarrhythmia was severe sinus bradycardia. Arrhythmias may result from myocarditis, cytokine storm, or Torsades de Pointes-induced hyperinflammation (TdP). The COVID-19 pandemic's high troponin levels were linked to an increase in malignant arrhythmias and mortality. The drug hydroxychloroquine (HCQ). A safe medication, it can cause TdP to precipitate by lengthening the QT interval. Additionally, in patients who are already prone to arrhythmias, the combination of HCQ with azithromycin raises the risk of TdP by lengthening QT interval. In adjusted Coxproportional Hazards Models, the mortality was lower for people getting both drugs than it hazard ratio (HR), 1.35, HCQ alone (HR, 1.08), or azithromycin alone (HR, 0.56), were not statistically different for patients receiving HCQ + azithromycin. The implications of QTc prolongation in outpatients or hospitalized patients, who are not in critical care units (ICUs), where continuous monitoring is not accessible, are unknown because these investigations were conducted in ICU settings with continuous QTc monitoring [16,17].

3.5. Pulmonary Embolism:

Current research has focused on PE as a hidden cause of death in COVID-19 pneumonia. The total number in two separate Italian case series [18], the incidence of PE ranged from 20.6% to 23%. The pulmonary artery segmental arteries frequently contained the PE. PE is the third most frequent cause of death in patients with COVID-19, after acute respiratory distress syndrome and myocarditis. The hyperinflammatory condition in COVID-19 and PE are conceivably related. Moreover, the existence of antiphospholipid the presence of antibodies in some COVID-19 pneumonia cases suggests that COVID-19 may be in a hypercoagulable condition. The COVID-19 pneumonia makes it challenging to distinguish the PE symptoms and signs, which delays the diagnosis of PE. Hypoxia, hypotension, and unexplained sinus tachycardia that worsen suddenly should raise the suspicion of PE. Following a thorough assessment of the symptoms and a strong clinical suspicion, echocardiography and CT pulmonary angiography could be performed. D-dimers are often elevated as a result of the cytokine storm in COVID-19 and will not aid in the diagnosis of PE [19,20].

4. Management of arrhythmias with COVID-19

Arrhythmias in COVID-19 patients should be managed in a safe manner by limiting exposure and being aware of drug-drug interactions. In confirmed COVID-19 cases, there aren't enough clinical trials to provide recommendations for managing arrhythmia. Any bradyarrhythmias or tachyarrhythmias in a COVID-19 patient should be treated in a manner similar to that given to patients who have an arrhythmia brought on by an infection or a temporary metabolic imbalance.

4.1. Bradyarrhythmias:

Patients with COVID-19 may experience bradycardia, including sinus or AV block, as a result of (hydroxy)chloroquine side effects, azithromycin, lopinavir/rotinavir, and. If the conduction system is affected by myocarditis, AV blockages may also be observed. Due to temporarily elevated vagal tone, intubated patients may have transitory bradycardia during tracheal secretion suctioning or when the patient is proning. Prior to temporary pacemaker implantation, isoprenaline and atropine usage can be considered in individuals with chronic bradycardia implantation [21]. Due to the transient nature of bradyarrhythmias, the nature of critical illness, the possibility of bacterial superinfection, and the risk of device infection, temporary pacemaker implantation is thought to be a suitable alternative before implanting a permanent device. However, once the patient has recovered from COVID-19 infection, the need for a permanent pacemaker should be re-evaluated [22].

4.2. Atrial tachyarrhythmias:

Secondary causes of atrial tachyarrhythmias such hypoxia, metabolic and electrolyte imbalances, and proarrhythmic conditions must be recognized and treated pharmacological interaction or cardiac ischemia. Although more research is required, intravenous adenosine can be utilized for acute termination in patients with supraventricular tachycardia. Those with refractory cases may want to undergo electrical cardioversion, but stable, asymptomatic people should wait. If beta-blockers (BBs) are contraindicated, one should have a low threshold for starting maintenance therapy with BBs or calcium-channel blockers (CCBs); however, to prevent bradycardia and QT prolongation, BB and CCB drug interactions with antiviral drugs should be assessed prior to starting these medications [23,24].

Consider stopping antiarrhythmic medications (AADs) in individuals with recurrent atrial fibrillation and flutter who are hemodynamically stable, especially To begin rate-controlling meds with BBs or CCBs unless contraindicated, with or without digoxin, and given serious drug-drug interactions with antiviral medications, sotalol, flecainide, and probably amiodarone and propafenone. Synchronized cardioversion or AAD drugs like amiodarone can be used as a rhythm control approach, but they should be closely monitored and used with caution is advised for individuals receiving fingolimod as part of a therapy regimen that also includes amiodarone. When using fingolimod, structurally normal hearts should choose propafenone or flecainide. TTE (transthoracic echocardiography) is only advised in patients with hemodynamic instability [24].

4.3. Ventricular arrhythmias:

Secondary reasons include hypoxia, metabolic or electrolyte imbalances, and drug-induced proarrhythmic effects must be recognized and treated. In those with amiodarone or intravenous lidocaine is the first line of treatment for ventricular tachycardia storms that are unrelated to the causes previously mentioned, especially if an underlying myocardial infarction is suspected? Other treatments include sympathetic blockade with esmolol, sedation, and potential intubation. Replace electrolytes to a target potassium level >4.5 in individuals with polymorphic ventricular tachycardia or ventricular fibrillation with a prolonged QTc interval [25].

Start an intravenous magnesium and isoprenaline infusion, and stop taking QT-extension medications. In case of recurring TdP or bradycardia, temporary pacemaker installation at the bedside utilizing a floatation guided approach can be explored to overdrive end ventricular tachycardia. Also, one should determine whether additional therapy, such as extracorporeal membrane oxygenation, is necessary. If the malignant ventricular arrhythmia has just developed and is unrelated to QT prolongation, patients should consider TTE. After COVID-19 recovery, assess the necessity of catheter ablation and ICD for secondary prevention. If myocarditis-related transient cardiomyopathy is detected, think about using wearable defibrillators [25].

5. Conclusion

While being primarily a respiratory illness, COVID-19 has grown so far that many patients are experiencing cardiovascular involvement. According to COVID-19, people with pre-existing cardiovascular diseases and risk factors are not the only ones who face an increased risk of morbidity and mortality. Cardiovascular problems such myocardial infarction, myocarditis, heart failure, arrhythmias, or PE may directly present. The evidence-based management of diverse cardiovascular complications, the care of chronic patients with cardiovascular problems who are not COVID-19 patients and the ethical difficulties in triaging these patients are a few of the research gaps. The search for SARS-CoV-2 vaccines and antiviral medications is currently underway on a global scale. The urgent requirement is to manage these patients according to the best evidence-based recommendations and to be aware of the daily new data that is emerging.

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