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SHORT COMMUNICATION

# Cardiovascular Complications of SARS-CoV-2 (COVID-19) in Adults

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## Abstract

Severe Acute Respiratory Syndrome-2 (COVID-19) was a devastating worldwide pandemic which affected multiple organs, resulting in significant mortality and morbidity. A hallmark of COVID-19 is the ability to mutate and infect efficiently as well as to spread widely among various age groups with special focus on the elderly and those with comorbid conditions. The advent of mRNA vaccine in a short period of time helped to mitigate hospitalization and mortality. This article will focus on the cardiovascular complications of COVID-19 and the treatment options. Also, the phenomenon of long COVID is discussed.

## Introduction

COVID-19 is a member of the Coronaviridae family and the Coronavirinae subfamily. SARS-CoV-2 is a single stranded positive sense RNA virus. Over 78 million cases have been reported in the US with a death rate of 1.2% [1]. About a third of the patients have laboratory evidence of cardiac involvement that is increased in severity due to the presence of comorbid conditions such as advanced age, hypertension, obesity, diabetes, and immune compromised conditions [2]. The mechanism of injury is related to direct viral damage and viremia. Heart involvement in combination with COVID-19 further enhances the morbidity and mortality rate [3]. Another important fact is that the COVID-19 vaccines have added additional burden on the morbidity related to cardiac involvement.

## Pathophysiology

As already mentioned, cardiac involvement from COVID-19 results from indirect injury from the virus consisting of a disturbance in the pro-inflammatory and anti-inflammatory response resulting in excessive pro-inflammatory response. This is very similar to Cytokine Release Syndrome (CRS) where T-cell involvement results in excessive cytokines in the circulation along with stress hormones such as epinephrine, resulting in subsequent cardiac injury [4] and thromboembolic complications from endothelial activation [5].

Direct injury to the heart results from viral affinity for cardiac Angiotensin-Converting Enzyme-2 Receptor (ACE-2). Comorbid conditions such as underlying hypertension and cardiac disease worsen direct injury because of the upregulation of ACE-2 receptors in the cardiac tissue [6].

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## Clinical Presentation

The heart injury caused by COVID-19 infection can vary significantly in its clinical manifestations and severity. While mild cases present only as minor fatigue and shortness of breath, more aggravated symptoms may manifest as chest tightness, chest pain, heart palpitations, and syncope. Critical illness can lead to cardiogenic shock, malignant arrhythmia, and even death.

## Dysrhythmia

Sinus tachycardia and atrial fibrillation are dysrhythmias that are commonly encountered [7]. Dysrhythmias occur in up to a third of admitted patients and in almost half of patients who are in the intensive care unit. The condition is worsened by comorbid conditions resulting in long hospitalization and the application of complex treatment modalities [8].

## Myocardial Injury

Myocarditis due to COVID-19 results from cytokine storm [9] and leads to an increase in mortality rates. Cardiac MRI (cMRI) aides in the diagnosis of myocarditis [10]

Coronary insufficiency refers to troponin values that are over 99% and need to be differentiated from myocarditis, cardiomyopathy, and acute plaque rupture [11].

Auxiliary examination of myocarditis caused by COVID-19 infection includes:

### Laboratory examination

For patients with confirmed COVID-19 infection and precordial discomfort, the (cardiac troponin I and cardiac troponin T) level (cTnI/cTnT) should be tested first. A high cTnI/cTnT ratio reflects myocardial injury caused by COVID-19 infection. In addition, elevated levels of type B Natriuretic Peptide (BNP) or N-terminal pro-B-type Natriuretic Peptide (NT-pro BNP) indicate cardiac dysfunction, which has subsidiary value in assessing the progression, efficacy, and prognosis of myocardial injury.

### ECG and holter

The most common ECG change in patients with myocarditis caused by COVID-19 infection is sinus tachycardia, frequent atrial or ventricular premature beats can be seen in patients with palpitation, and short supraventricular or ventricular tachycardia can be seen in some patients. When the conduction system is damaged, bradycardia, bundle branch block, or atrioventricular block can also occur.

### Echocardiography

When COVID-19 infection causes extensive myocardial damage, echocardiography can show complete or localized left ventricle contractile dysfunction. Partial mild myocarditis has completely normal left ventricular function.

### Cardiac Magnetic Resonance Imaging (CMR)

CMR is an important imaging tool to diagnose whether myocardial injury related to COVID-19 infection is myocarditis. It was found that two thirds of CMR patients with myocarditis showed different degrees of Late Gadolinium Enhancement (LGE), and diffuse edema was the only CMR feature of COVID-19 infection related myocarditis.

ST-elevation myocardial infarction is tackled by Percutaneous Coronary Intermediation (PCI) [12]. Fibrinolytic treatment is reserved for those with Acute Respiratory Distress Syndrome (ARDS), severe bilateral pneumonia from COVID-19, and those with poor prognosis [13].

About a quarter to a third of the patients hospitalized with COVID-19 present with compensated heart failure due to cardiogenic shock from indirect cardiac injury [14]. Treatment intervention for ARDS with mechanical ventilation worsens heart failure from increased right ventricular afterload which results from the use of a high PEEP and low tidal volume strategy despite a decrease in left ventricular afterload [15]. Stress-Induced Cardiomyopathy (SIC) (Takotsubo cardiomyopathy] from overwhelming amounts of circulating catecholamines has been described in 1-4% of COVID-19 patients [16]. Treatment consists of using β-blockers and Angiotensin Converting Enzyme Inhibitors (ACE) or Angiotensin Receptor Blockers (ARBS).

### Acute Coronary Syndrome (ACS)

The term ACS is used when there is hint or proof of myocardial injury. It encompasses Unstable Angina (UA), ST-Segment Elevation Myocardial Infarction (STEMI), and Non-Segment Elevation Myocardial Infarction (NSTEMI). It may occur because of direct cardiac injury by COVID-19 or from the breakdown of ACE-2. Clinical presentation includes left sided

chest pain, and elevated cardiac enzymes including cardiac troponin above 99 percentiles. Thrombotic plaque rupture heralds the onset of ACS. ACS is a medical emergency and needs well-timed diagnosis and treatment. UA is treated with nitrates for pain relief as well as aspirin and the anti-platelet agent, clopidogrel, if there is no risk of bleeding and invasive intervention is not contemplated. Percutaneous Invasive Therapy (PCI) depends on the clinical situation. For STEMI and NSTEMI medical therapy or PCI is considered depending on the patient's level of illness [17].

### Clot formation

There are multiple reasons for clot formation following COVID-19 infection. These reasons include immobility leading to stasis, hypercoagulable state, hypoxemia, platelet, endothelial activation, and dysfunction, overactive proinflammatory state, immune disharmony as well as comorbid conditions. As many as one third of the patients admitted into the ICU suffer from thromboembolism resulting in deep vein thrombosis, pulmonary embolism, and stroke [18]. Laboratory findings of elevated fibrin degradation products and D-dimer portend a poor outcome and must raise suspicion for clot formation [19]. Low molecular weight heparin (Lovinox) is used as a prophylaxis against clot related complications.

Complications related to medications used against COVID-19:

### Antimalarial drugs and macrolide antibiotic

Chloroquine, hydroxychloroquine, and Azithromycin induce a prolonged QT-interval, especially in those with renal and cardiac dysfunction, and lead to arrythmias such as ventricular tachycardia including torsade de pointes and cardiac arrest [20]. These drugs are ineffective against COVID-19.

### Antiviral

Remdesivir is an RNA-polymerase inhibitor that shortens hospital stay and may be useful in outpatient therapy in arresting progression of the disease. Toxicity to cardiac, liver, and renal systems is possible [21].

The combination of Nrimatrlvir and Ritonavir is a protease inhibitor used as an outpatient therapy in those with mild symptoms. It reduced hospitalization and death by 89%. This drug combination interacts with many other drugs and therefore needs careful application in the treatment of COVID-19 [22].

Molnuparivir interferes with the viral genome as a cytidine nucleoside and helps in early viral clearance [23].

## Immunomodulatory Therapy

### Steroid therapy

Dexamethasone is a glucocorticoid which has immunosuppressive and anti- inflammatory properties. It is shown to reduce mortality. It has multiple side effects including inducing arrhythmia, worsening heart failure, and glucose intolerance [24].

### Anti-IL-6 therapy

Tocilizumab is an anti-IL-6 drug that antagonizes the proinflammatory effect of IL-6. It has been shown to decrease the need for ventilator use as well as decrease mortality from COVID-19. It causes prolonged QT interval and requires caution in its use [25].

### Janus kinase inhibitor

The JAK-1 and JAK-2 inhibitor, Baricitinib, decreases IL-6 generation and, in combination with Remdesivir, hastens recovery in hospitalized patients. It can cause thromboembolism [26].

### Fulminant myocarditis

Fulminant myocarditis requires positive muscle strength support or temporary mechanical circulation support such as Intraarterial Balloon Pump (IABP), Extracorporeal Membrane Oxygenation (ECMO), and Impella which is a percutaneous ventricular assist device.

## COVID-19 Vaccine

These vaccines based on mRNA technology are very effective against COVID-19. They however cause transient pericarditis and myocarditis in adolescents and young adults [27]. It is hypothesized that cardiac complications may be related to an effective antibody response, an inflammatory response resulting from the interaction of a protein in the heart and the viral protein, or a previous infection precipitating activation of immune cells by cytokines. An elevation in ESR, C-reactive protein is noted. EKG and an ECHO shows evidence of heart involvement.

## Long COVID

Long COVID is a debilitating chronic condition occurring following COVID-19 infection and is a multisystem condition seen in about 10% of those who are vaccinated, 10-30% of outpatients, and 50-70% of in-patients. It typically afflicts those who are between 35-50 years of age. The affected systems include neurologic, cardiovascular, pulmonary, muscle, immune, endocrine, kidneys, GI tract including spleen and liver, and the reproductive system. Also characteristic is chronic fatigue. It is postulated that long COVID may result from the disturbance of the immune system via the activation of the Epstein-Barr Virus (EBV) and the Human Herpes Virus-6 (HHV-6), immune priming and autoimmunity from molecular mimicry, a disturbance in the gut microbiota, endothelial dysfunction leading to microvascular thrombosis, and signaling abnormality in the brain stem and Vagus nerve. At present, diagnostic tests and treatment options are in the developmental stage [28].

## Conclusion

Covid-19 causes serious cardiovascular complications in adult subjects and comorbid conditions are added risk factors. In addition, some of the drugs used to treat COVID-19 have cardiac effects and careful consideration is necessary when using them. Vaccines used against COVID-19 also have cardiac toxicity albeit the toxicity is not considered to be very serious. Long COVID affects multiple organs, and, at present, definitive therapy is unavailable. Long-term follow-up is needed.

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